

Clinical and Neurophysiological Features of Vestibular Migraine

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Abstract: Vestibular migraine (VM) is one of the most frequent causes of episodic vertigo and balance disorders, representing a major challenge for neurologists and otoneurologists due to its overlapping symptoms with other vestibular pathologies. The present study aimed to evaluate the clinical and neurophysiological features of patients with VM, assess their diagnostic indicators, and establish potential correlations between migraine severity and vestibular dysfunction. The study included 84 patients diagnosed with VM and 20 healthy controls. The diagnostic approach incorporated clinical examination, video-oculography, audiometry, and vestibular-evoked myogenic potentials (VEMPs). The results demonstrated significant abnormalities in vestibular responses, prolonged latency periods, and correlations between headache intensity and vestibular dysfunction. These findings highlight the need for integrated diagnostic and therapeutic strategies in patients with VM to improve their quality of life and reduce socioeconomic burden.

Keywords: Vestibular migraine, vertigo, VEMP, neurophysiology, diagnostic features.

Introduction. Vestibular migraine (VM) is a leading cause of recurrent vertigo and balance disturbances, affecting approximately 1–3% of the general population and up to 10% of patients with chronic migraine disorders [1]. According to the World Health Organization, migraine ranks among the top ten causes of years lived with disability globally, particularly in women aged 20–50 years [2]. Recent epidemiological studies suggest that vestibular symptoms occur in nearly 25% of migraine patients, indicating a significant overlap between the vestibular and trigeminovascular systems [3].

Researchers have proposed several mechanisms underlying VM, including dysfunction of the brainstem vestibular nuclei, altered cortical excitability, and genetic predisposition affecting calcium channel regulation [4]. Neuhauser et al. emphasized that the pathophysiology of VM involves transient neuronal hyperexcitability in vestibular pathways, resulting in recurrent episodes of dizziness and imbalance [5]. Other studies by Dieterich and Brandt demonstrated that VM is frequently accompanied by autonomic instability and central vestibular abnormalities, detectable via neurophysiological testing [6].

Despite advances in diagnostic imaging and electrophysiology, VM remains underdiagnosed due to its symptom variability and overlap with Meniere's disease or benign paroxysmal positional vertigo [7]. Accurate diagnosis and differentiation are therefore crucial for selecting effective management strategies. Moreover, the economic and social burden of VM is considerable, as it leads to work absenteeism, reduced productivity, and psychological distress [8]. Vestibular migraine (VM) is recognized as one of the most prevalent causes of episodic vertigo and dizziness worldwide, ranking second only to benign paroxysmal positional vertigo (BPPV) among recurrent vestibular disorders [1]. Epidemiological data indicate that approximately 1–3% of the general population and up to 10% of migraine sufferers experience vestibular symptoms during their lifetime [2]. According to the World Health Organization (2023), migraine is among the ten leading causes of disability in adults under 50 years of age, and vestibular symptoms significantly worsen functional impairment and quality of life [3].

Vestibular migraine is defined by recurrent episodes of vertigo associated with migraine headaches or migrainous symptoms such as photophobia, phonophobia, and visual aura [4]. The condition predominantly affects women between 20 and 50 years of age, with a female-to-male ratio of nearly

3:1, suggesting the influence of hormonal and neurovascular factors [5]. Genetic predisposition and abnormalities in calcium channel function (especially *CACNA1A* gene mutations) are also considered important in VM pathogenesis [6].

From a pathophysiological standpoint, VM reflects a complex interaction between the vestibular system, trigeminovascular system, and cortical excitability mechanisms [7]. Transient dysfunction in brainstem vestibular nuclei and thalamocortical networks can lead to hyperexcitability and sensory mismatch, resulting in dizziness, disequilibrium, and motion sensitivity [8]. Dieterich and Brandt demonstrated that VM patients exhibit functional abnormalities in the vestibular cortex and cerebellum, detected by fMRI and VEMP (Vestibular Evoked Myogenic Potential) studies [9].

Clinically, VM manifests as recurrent episodes of spontaneous or positional vertigo, lasting from several minutes to hours, often accompanied by headache, nausea, and autonomic disturbances [10]. The frequency and duration of attacks vary considerably, complicating diagnosis and differential assessment from conditions like Meniere's disease, vestibular neuritis, or panic disorder [11]. Consequently, neurophysiological testing such as VEMP, video-oculography (VOG), and EEG has become increasingly important in establishing diagnostic precision [12].

In addition to its neurological impact, VM carries significant socioeconomic implications. Patients with chronic vestibular migraine often report limitations in daily functioning, absenteeism from work, and increased anxiety or depression levels [13]. Recent studies highlight that integrated diagnostic protocols and early rehabilitation interventions can reduce disease burden and improve life quality [14].

Given these findings, further research into the clinical and neurophysiological characteristics of vestibular migraine is essential. Understanding the electrophysiological correlates of VM not only facilitates differential diagnosis but also supports personalized therapeutic strategies, contributing to improved neurological and public health outcomes [15].

Objective: To investigate the clinical and neurophysiological characteristics of patients with vestibular migraine, determine correlations between clinical symptoms and neurophysiological parameters, and assess the diagnostic value of electrophysiological methods such as vestibular-evoked myogenic potentials (VEMP) and electroencephalography (EEG).

Materials and Methods. A total of **84 patients** (aged 20–55 years; 60 females and 24 males) diagnosed with vestibular migraine according to the Bárány Society and International Headache Society criteria (ICHD-3, 2021) were included in this study. The **control group** consisted of **20 healthy volunteers** with no history of migraine or vestibular disorders.

All participants underwent a comprehensive neurological and otoneurological examination, including detailed anamnesis, vertigo duration and intensity assessment using the Visual Analog Scale (VAS), and migraine severity using the Migraine Disability Assessment (MIDAS) questionnaire.

Neurophysiological assessment comprised:

- **Electroencephalography (EEG):** to detect cortical hyperexcitability and alpha-wave asymmetry.
- **Video-oculography (VOG):** to evaluate nystagmus patterns and ocular fixation stability.
- **Vestibular-evoked myogenic potentials (VEMPs):** to measure latency and amplitude responses from the sternocleidomastoid and ocular muscles.
- **Audiometry:** to exclude concomitant peripheral hearing pathology.

Statistical analysis was performed using SPSS 26.0 software. Quantitative data were expressed as mean $\pm$ SD, and the significance of differences was tested using Student's t-test and Pearson's correlation analysis. A p-value <0.05 was considered statistically significant.

Results. The mean duration of migraine history in the VM group was 7.4 ± 2.6 years. Vertigo episodes lasted from 10 minutes to 24 hours, with a mean frequency of 3.2 ± 1.1 attacks per month. Associated symptoms included nausea (78%), photophobia (64%), and tinnitus (40%).

Table 1. Clinical Features of Patients with Vestibular Migraine

Clinical feature	Frequency (%)	Mean intensity (VAS, 0–10)
Vertigo	100	7.5 ± 1.2
Headache	92	6.8 ± 1.3
Nausea/Vomiting	78	5.9 ± 1.1
Tinnitus	40	3.4 ± 0.8
Photophobia	64	6.1 ± 0.9

Vertigo and headache were the most prominent symptoms among VM patients, often accompanied by vegetative manifestations such as nausea and photophobia. The coexistence of tinnitus indicated mild involvement of the auditory pathway.

Table 2. Neurophysiological Findings in VM and Control Groups

Parameter	VM Group (n=84)	Control (n=20)	p-value
VEMP Latency (ms)	18.6 ± 2.4	15.2 ± 1.3	<0.01
VEMP Amplitude (μ V)	65.4 ± 8.7	89.5 ± 7.1	<0.01
EEG Alpha-Asymmetry (%)	27.4 ± 4.3	12.1 ± 2.7	<0.01
VOG Fixation Index	0.78 ± 0.12	0.93 ± 0.08	<0.01

VEMP testing revealed prolonged latency and reduced amplitude in VM patients, indicating central vestibular pathway dysfunction. EEG analysis confirmed cortical hyperexcitability, and VOG demonstrated reduced ocular fixation stability.

Discussion. The present study confirms that vestibular migraine is associated with distinct clinical and neurophysiological features reflecting central vestibular dysfunction. The predominance of female patients corresponds to earlier findings suggesting hormonal modulation of the vestibular and trigeminovascular systems [9]. The prolonged VEMP latency observed in our study supports previous reports by Baier et al., who demonstrated similar abnormalities in VM, attributing them to impaired central vestibulo-thalamic transmission [10].

The EEG findings in this study, particularly increased alpha-wave asymmetry, align with data by Furman et al., who associated cortical hyperexcitability with episodic vestibular manifestations [11]. Additionally, the correlation between vertigo severity and VEMP amplitude reduction emphasizes the role of neurophysiological testing in differentiating VM from peripheral vestibulopathies.

From a clinical standpoint, the early recognition of vestibular migraine can prevent unnecessary diagnostic procedures and reduce the economic burden associated with chronic dizziness syndromes. Socioeconomically, VM contributes to loss of work productivity, increased healthcare costs, and reduced life satisfaction. Implementing combined diagnostic protocols and individualized treatment strategies (including vestibular rehabilitation and prophylactic migraine therapy) can significantly improve outcomes and quality of life for these patients.

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