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The Clinical and Neurophysiological Features of Vestibular Migraine

Akhrorova Shakhlo Botirovna, Kamalova Feruza Umarovna
Bukhara State Medical Institute

Abstract: Vestibular migraine (VM) is one of the most common causes of recurrent vertigo, representing a significant clinical and social problem due to its chronic course and impact on quality of life. This study aimed to investigate the clinical and neurophysiological characteristics of VM using modern diagnostic methods, including vestibular evoked myogenic potentials (VEMP) and electroencephalography (EEG). A total of 80 patients with VM and 30 healthy controls were examined. The results revealed distinct vestibular dysfunction patterns, abnormal EEG activity in temporal–parietal regions, and correlations between dizziness severity and migraine frequency. The findings highlight the importance of comprehensive neurophysiological assessment for improving diagnostic accuracy and treatment strategies in VM.

Keywords: vestibular migraine, dizziness, VEMP, EEG, neurophysiology, vestibular dysfunction

Introduction. Vestibular migraine (VM) is a neurological disorder characterized by episodic vertigo associated with migraine features such as headache, photophobia, and phonophobia. According to recent epidemiological data, VM affects approximately 1–2.7% of the general population, accounting for up to 10% of all cases of vertigo [1]. It predominantly affects women between 20 and 50 years of age and is frequently underdiagnosed due to overlapping symptoms with other vestibular pathologies [2].

Several studies have emphasized that vestibular migraine represents a complex interplay between migraine mechanisms and vestibular system dysfunction [3]. According to Lempert and Neuhauser [4], VM results from abnormal excitability of the vestibular nuclei due to cortical spreading depression and impaired brainstem modulation. Furman et al. [5] demonstrated abnormal vestibulo-ocular reflex responses and delayed vestibular evoked potentials in VM patients. Neuroimaging studies revealed reduced gray matter volume in the parieto-insular vestibular cortex, suggesting a central origin of the disorder [6].

The socio-economic burden of VM is considerable, as patients experience recurrent dizziness, imbalance, and cognitive difficulties that interfere with daily activities and occupational functioning [7]. Early diagnosis and pathophysiological understanding are crucial for targeted therapy. However, despite significant progress in migraine research, VM remains poorly understood and frequently misdiagnosed.

Purpose of the Study: To evaluate the clinical and neurophysiological features of vestibular migraine through clinical assessment, vestibular function testing, and electrophysiological methods to enhance diagnostic precision and optimize treatment strategies.

Materials and Methods. The study was conducted at the Neurology Department of the Central Asian Medical University between 2023 and 2025. It included 80 patients diagnosed with vestibular migraine (58 females and 22 males, aged 20–55 years) and a control group of 30 healthy volunteers matched for age and sex. All participants provided informed consent.

Inclusion criteria: recurrent vertigo episodes associated with migraine features according to the ICHD-3 criteria.

Exclusion criteria: Ménière’s disease, benign paroxysmal positional vertigo, central nervous system lesions, and hearing loss.

Clinical evaluation included detailed neurological and otoneurological examination, the Dizziness Handicap Inventory (DHI), and the Migraine Disability Assessment (MIDAS).

Neurophysiological testing comprised:

- EEG recording (19-channel system, 10–20 international montage) to assess cortical excitability and paroxysmal activity.
- VEMP testing (air-conducted stimuli, 500 Hz tone bursts) for evaluating saccular and inferior vestibular nerve function.
- Video head impulse test (vHIT) for detecting semicircular canal dysfunction.
- Audiometry and caloric testing were performed to exclude peripheral causes.

Statistical analysis was conducted using SPSS 26.0. Quantitative data were expressed as mean ± SD. The Student's t-test and Pearson correlation were applied, with $p < 0.05$ considered statistically significant.

Results. Among the 80 VM patients, 72.5% reported recurrent vertigo lasting 5 minutes to several hours, while 65% experienced simultaneous migraine headaches. Photophobia and phonophobia were reported by 68% and 55% of patients, respectively. DHI scores indicated moderate to severe functional impairment in 64% of participants.

Table 1. Clinical manifestations of vestibular migraine

Symptom	Frequency (%)	Mean duration of episodes (min)
Vertigo	100	35 ± 12
Headache	65	42 ± 10
Nausea	52	—
Photophobia	68	—
Phonophobia	55	—

Note: Vertigo and headache were the most common manifestations, often accompanied by sensory hypersensitivity.

Neurophysiological findings revealed significant abnormalities in VEMP and EEG results. The P13 and N23 latencies in cVEMP were prolonged in 58% of VM patients, and inter-amplitude reduction was observed in 46%. EEG analysis demonstrated paroxysmal theta and beta activity in temporal and occipital regions in 40% of patients, suggesting cortical hyperexcitability.

Table 2. Neurophysiological parameters in VM and control groups

Parameter	Control group (n=30)	VM group (n=80)	p-value
P13 latency (ms)	13.8 ± 0.9	16.5 ± 1.2	<0.01
N23 latency (ms)	23.4 ± 1.1	27.1 ± 1.6	<0.01
Amplitude (μV)	82.5 ± 6.4	56.2 ± 8.3	<0.05
EEG paroxysmal discharges (%)	10	40	<0.01

Note: VM patients exhibited significant prolongation of VEMP latencies and increased paroxysmal EEG activity compared to healthy controls.

A moderate correlation ($r = 0.61$; $p < 0.01$) was found between DHI scores and VEMP latency, indicating that vestibular dysfunction severity parallels clinical dizziness handicap.

Discussion. The results confirm that vestibular migraine is not limited to peripheral vestibular impairment but involves significant central neurophysiological alterations. The prolongation of VEMP latencies and reduction in amplitude indicate dysfunction in the vestibulo-collic pathways and possible impaired brainstem processing [8]. Increased EEG paroxysmal activity corresponds to cortical hyperexcitability, consistent with previous studies by Furman et al. and Beh et al. [5,9].

Our findings support the hypothesis that VM involves abnormal integration between cortical, subcortical, and vestibular centers. This explains the coexistence of vertigo and migraine symptoms and justifies the use of multimodal neurophysiological assessments in diagnostic practice.

From a clinical perspective, identifying electrophysiological markers helps differentiate VM from other vestibular disorders, reducing diagnostic errors.

From an economic and social viewpoint, early diagnosis and adequate management reduce recurrent hospital visits, improve work productivity, and enhance patients' quality of life.

Conclusion. Vestibular migraine represents a multifactorial disorder involving both cortical and brainstem mechanisms of vestibular regulation. The combined use of clinical scales, VEMP, and EEG provides an effective diagnostic framework for early recognition. The identification of prolonged vestibular evoked potential latencies and increased cortical excitability may serve as reliable markers of VM. Comprehensive diagnostic strategies are essential for optimizing treatment, preventing chronicity, and minimizing socio-economic burden associated with vestibular migraine.

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