**REVIEW ARTICLE**

# Diagnostic challenges in vestibular migraine: multisensory triggers, neurological-epileptic overlap and the role of functional MRI in oto-neurological practice – A narrative review

Thuduvage VS[1], Ediri Arachchi WM[2], Wanniarachchi AA[3]

[1]Department of Clinical Sciences, Faculty of Medicine, General Sir John Kotelawala Defence University (KDU), Sri Lanka

[2]Faculty of Allied Health Sciences, General Sir John Kotelawala Defence University, Sri Lanka

[3]Department of Otorhinolaryngology, University Hospital, Kotelawala Defence University, Sri Lanka

**Article Information**

**Corresponding Author**
Dr. Vasanthika Thuduvage

Email: vasanthikat@kdu.ac.lk

https://orcid.org/0000-0002-5730-5409

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## Abstract

Vestibular migraine (VM) poses diagnostic challenges in oto-neurological practice due to its heterogeneous presentation, multisensory triggers, and clinical overlap with epilepsy and other vestibular disorders.[1] Current diagnostic criteria, primarily based on subjective symptomatology, are limited in capturing atypical and non-headache-dominant cases.[2] Emerging evidence highlights functional MRI (fMRI) as a promising tool for revealing altered connectivity in sensory-processing brain regions, offering potential biomarkers for improved diagnosis.[3] This review aims to elucidate multisensory triggers, explore the neurological-epileptic overlap, and assess the diagnostic potential of fMRI in VM. The discussion emphasizes controversies in classification, gaps in current knowledge, and future directions toward establishing imaging-informed diagnostic frameworks.

**Keywords:** Vestibular migraine, functional MRI, multisensory triggers, neurological-epileptic overlap, diagnostic challenges

## Introduction and objectives

Vestibular migraine (VM) is increasingly recognized as one of the most common causes of recurrent vertigo, affecting an estimated 1-3% of the general population and up to 10% of patients presenting to specialist neuro-otology clinics.[4,5] Despite this relatively high prevalence, VM continues to be underdiagnosed. Its diagnostic complexity stems from its heterogeneous presentation, frequent absence of headache, and clinical overlap with both peripheral vestibular disorders (e.g., Ménière's disease, benign paroxysmal

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positional vertigo) and central neurological syndromes (e.g., epilepsy, transient ischemic attacks).[1, 6]

The clinical burden of VM is considerable, often resulting in prolonged episodes of vertigo, imbalance, photophobia, phonophobia, nausea, and cognitive disturbances. These symptoms not only impair quality of life but also lead to repeated consultations across specialties, including ENT, neurology, and emergency care.[7] In ENT practice, particularly, VM is frequently overlooked because initial evaluation often focuses on structural ear pathologies, sinus disorders, or non-specific labyrinthine dysfunction.[8] Epidemiological surveys suggest that a significant proportion of patients with unexplained dizziness in ENT clinics actually fulfill criteria for VM once systematic evaluation is performed.

The complexity of VM is compounded by its multimodal triggers, which include visual motion, loud sounds, tactile or postural changes, and even stress or hormonal fluctuations.[9,10] These features suggest a fundamental dysfunction of cortical and thalamocortical sensory processing networks, complicating their differentiation from epilepsy and other vertigo syndromes.

This narrative review aims to (1) elucidate the multisensory triggers and cortical mechanisms of VM, (2) analyze its clinical and pathophysiological overlap with epilepsy, (3) identify diagnostic pitfalls within ENT practice, and (4) evaluate the role of functional MRI in advancing diagnostic accuracy.

## Methods

A literature search was conducted in PubMed, Scopus, and Web of Science up to March 2025 using the keywords 'vestibular migraine,' 'functional MRI,' 'multisensory triggers,' and 'epileptic overlap.' The study included 19 original research articles, reviews, and case reports published in English. Reference lists of selected papers were also screened to identify additional relevant publications. This approach ensured a broad representation of clinical, neuroimaging, and pathophysiological evidence relevant to VM.

## Results

### Multisensory triggers and cortical dysregulation

VM is distinguished by sensitivity to diverse sensory stimuli, including visual motion, flashing lights, loud or rhythmic auditory inputs, and somatosensory changes such as neck movements or positional shifts. These triggers provoke vertiginous episodes often accompanied by migraine features, consistent with a disorder of central sensory amplification.[9, 10]

Neurophysiological evidence supports the concept of disrupted thalamocortical relay pathways.[11,12] The thalamus, acting as a sensory hub, exhibits aberrant activation patterns during VM attacks, reflecting impaired inhibitory control. This disruption resembles cortical spreading depression (CSD) phenomena observed in migraine with aura,[12] extending into vestibular and somatosensory regions. The posterior insula, a central hub for vestibular integration and interoception, demonstrates altered connectivity in fMRI studies,[3,10] further implicating VM as a disorder of multisensory integration rather than purely a vestibular origin.

Clinical observations consistently highlight sensory hypersensitivity with reported intolerance to environments with excessive visual motion (e.g., supermarkets),[9] heightened responses to everyday auditory stimuli, and vertigo provoked by minor head or neck movements. These findings align with patient-reported outcomes and underscore VM's position within the spectrum of sensory processing disorders.

### Neurological-epileptic overlap

VM and focal epilepsies – particularly those of temporal and parietal origin – share striking similarities in clinical manifestation.[14] Both present with paroxysmal, sensory-driven episodes, often triggered by visual or auditory inputs. This overlap diagnosis, particularly problematic when otological signs are absent.

Electrophysiological studies have demonstrated cortical hyperexcitability in VM patients, resembling epileptiform patterns seen in partial epilepsy. Reports of *migralepsy,* where a seizure follows a migraine aura,[15] further blur diagnostic boundaries. In paediatric and adolescent populations, these overlapping features can delay appropriate intervention if clinicians fail to consider both conditions.

From an ENT perspective, patients presenting with recurrent vertigo without hearing loss or tinnitus are often mistakenly excluded from migraine or epilepsy pathways.[5, 8] Yet, the involvement of temporoparietal and insular regions in both VM and epilepsy reinforces the necessity of functional evaluation. While vestibular epilepsy (VE) is typically associated with epileptiform EEG discharges and postictal features, VM lacks these hallmarks, though occasional responsiveness to antiepileptic drugs adds to the diagnostic dilemma.[16, 17]

**Diagnostic criteria and ENT pitfalls**

The ICHD-3/Bárány criteria for VM require a minimum of five vestibular episodes in a patient with a migraine history and at least one migraine-associated feature.[2] While useful, these criteria are limited in sensitivity and specificity. Many patients present with vertigo as the predominant or sole symptom, without concurrent headache, leading to misclassification.

In ENT clinics, this diagnostic gap is particularly problematic.[13] Standard vestibular tests – including caloric testing, vestibular evoked myogenic potentials (VEMPs), and video head impulse testing (vHIT) – often yield normal or inconclusive results.[5, 8] Consequently, VM is frequently misattributed to conditions such as sinusitis, eustachian tube dysfunction, or "non-specific labyrinthine disease".[8] Such misdiagnoses not only delay correct treatment but can subject patients to unnecessary antibiotics, sinus surgery, or prolonged vestibular suppressant therapy.

The absence of objective biomarkers in current diagnostic criteria underscores the need for complementary neuro-functional tools.

## Imaging: Conventional MRI vs Functional MRI

Conventional MRI plays an essential role in excluding structural lesions in patients presenting with unexplained vertigo, such as tumors, demyelinating lesions, or infarcts.[18] However, in VM, MRI findings are almost invariably normal, highlighting the functional rather than structural nature of the disorder.

In contrast, functional MRI (fMRI) has provided valuable insights into altered connectivity patterns in VM. Resting-state fMRI demonstrates disrupted coupling within the default mode network, while task-based paradigms show hyperactivation in vestibular-responsive regions such as the insula, cerebellum, and temporoparietal junction.[3] Importantly, fMRI studies have also revealed enhanced connectivity between visual and vestibular cortices, consistent with clinical hypersensitivity to visual motion.[10]

Despite these advances, translation into clinical practice remains limited. Barriers include the high cost of fMRI, limited availability outside academic centers, and the lack of standardized diagnostic paradigms. Nevertheless, fMRI remains a promising candidate for establishing neuro-functional biomarkers that could reduce misdiagnosis in ENT settings.

**Comparative insights: vestibular migraine (VM) vs vestibular epilepsy (VE)**

Clinical, electrophysiological, and imaging comparisons reveal both overlaps and distinctions. VM is characterized by prolonged episodes (minutes to hours), often with migraine-associated features. VE presents as brief vertiginous seizures with EEG epileptiform activity.[19] While fMRI in VM reveals altered connectivity, VE diagnosis hinges on EEG confirmation despite often normal structural MRI.

These differences underscore the need for functional imaging approaches and are summarized in a comparative Table 1.

## Discussion

Vestibular migraine (VM) is increasingly understood as a disorder of multisensory integration and cortical excitability rather than a purely vestibular condition. The overlap with epilepsy complicates clinical recognition, particularly in ENT practice, where access to EEG and advanced imaging may be limited. Current diagnostic practice often relies on subjective clinical history and criteria that fail to capture atypical cases, resulting in VM being underrecognized. In many ENT clinics, patients presenting with recurrent vertigo but no overt otological pathology risk misdiagnosis unless VM is specifically considered.

Controversies remain regarding the diagnostic boundaries between VM and VE.[4,6] Some VM patients respond positively to antiepileptic drugs, while normal interictal EEG recordings in VE can mimic VM, creating diagnostic ambiguity.[17] This clinical overlap underscores the need for objective diagnostic tools. fMRI has revealed altered connectivity in key sensory integration regions such as the insula, thalamus, and temporoparietal cortex, highlighting its potential as a biomarker.[3,18] However, fMRI remains underutilized in clinical workflows due to issues of cost, limited availability, and the absence of standardized diagnostic protocols.

Significant gaps persist in the current understanding and management of VM. No objective biomarkers exist in diagnostic criteria, and large-scale validation studies of fMRI findings are minimal.[18] Additionally, standardized imaging protocols that could reliably differentiate VM from related disorders such as VE or Meniere's disease remain undeveloped. In routine ENT practice, this gap often leads to inconclusive vestibular testing or misattribution of vertigo to non-specific labyrinthine or sinus conditions.[8]

**Table 1. Comparative clinical, electrophysiological, and neuroimaging features distinguishing vestibular migraine from vestibular epilepsy**

| Feature | Vestibular Migraine (VM) | Vestibular Epilepsy (VE) |
|---|---|---|
| Symptom duration | Episodes last 5min to 72h, often accompanied by headache, photophobia, phonophobia or aura | Seconds to minutes, typically brief paroxysms of vertigo sometimes preceding full seizure |
| Triggers | Multisensory triggers including visual stimuli, motion, stress, hormonal changes; migraine context often present [4,9,10] | Vestibular and auditory stimulation may cause vertigo[14,16] |
| EEG findings | Interictal EEG is usually normal; no epileptiform activity has been observed[16,17] | About 25% of sleep EEGs show interictal epileptic discharges, which are frequently posterior temporo-parietal. Localised EEG abnormalities have been observed [14,19] |
| Neuroimaging (fMRI/EEG) | No structural lesions are visible, but MRI reveals changed connectivity in the nociceptive, vestibular, and visual cortices [3,18] | fMRI-EEG correlation may show altered parietal-temporal activation[14, 19] |
| Key diagnostic markers | Diagnosis based on ICHD-3/Bárány criteria: history of migraine with ≥5 vestibular episodes, ≥50% accompanied by migraine features. Supportive markers include visual-motion hypersensitivity and altered thalamocortical connectivity on fMRI, which serves as a research-level adjunct[2,3,4,9,10] | Diagnosis centered on clinical-electro-radiological criteria. And relies on recurrent isolated vestibular seizures confirmed by ictal or interictal epileptiform EEG activity, typically localized to posterior temporo-parietal or insular regions, with normal structural MRI findings; fMRI may show transient cortical hyperactivation consistent with seizure onset zones [14, 16, 19] |
| Treatment response | Migraine prophylaxis; partial response to antiepileptics in certain times[4,6]. | Antiepileptic drugs; poor response to migraine therapy[14, 19] |

Future research should focus on establishing standardized fMRI paradigms and longitudinal studies to monitor treatment response and disease progression. Integrating neurofunctional imaging with clinical assessment may enhance diagnostic accuracy and reduce misclassification. Multidisciplinary collaboration between neurology, otolaryngology, and radiology is critical to develop precision diagnostic frameworks. Ultimately, embedding advanced imaging into oto-neurological workflows has the potential to transform the clinical management of patients with recurrent unexplained vertigo.[13]

## Conclusions

Vestibular migraine is an underrecognized but disabling disorder with multisensory triggers and significant overlap with epilepsy. Reliance on subjective diagnostic criteria perpetuates misclassification.[4] Functional MRI demonstrates distinct connectivity changes, suggesting its potential as a biomarker for more accurate diagnosis. Future progress requires standardized imaging protocols, large-scale validation studies, and interdisciplinary collaboration to refine diagnostic frameworks and integrate neurofunctional tools into clinical oto-neurology.

## Authors' Contributions

Conceptualized the review framework, coordinated the overall manuscript preparation, and supervised data synthesis and manuscript revision. Led the formulation of the diagnostic comparison model between vestibular migraine and vestibular epilepsy, and finalized the submitted version: TVS.

Jointly contributed to conceptual design, literature selection, and methodological structure. Participated in critical analysis of clinical and neuroimaging evidence and co-drafted the sections on multisensory triggers and functional MRI findings. Reviewed and revised the manuscript critically for intellectual content: EWM.

Contributed equally to methodological design, data extraction. Assisted in writing and editing the sections on diagnostic criteria, ENT implications, and comparative analysis. Organizing the diagnostic comparison model between vestibular migraine and vestibular epilepsy. Participated in comprehensive manuscript revision: WAA.

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