Vestibular Migraine vs. Meniere Disease: Separating Overlapping Diagnoses

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Vertigo, dizziness, and auditory symptoms frequently overlap across several neurotological disorders, creating diagnostic ambiguity for clinicians. Vestibular migraine and Meniere disease represent two such conditions, often presenting with similar symptom profiles that complicate accurate identification. Distinguishing between these two diagnoses is not merely an academic exercise; it dictates treatment pathways and patient prognosis.

KEY TAKEAWAYS

- The Pivot: Elevated interictal plasma calcitonin gene-related peptide (CGRP) levels may serve as a headache-independent biomarker to differentiate Meniere disease from vestibular migraine.
- The Data: Plasma CGRP levels were significantly higher in Meniere disease patients compared to those with vestibular migraine (PThe Action: Clinicians should consider plasma CGRP testing as an adjunctive diagnostic tool, alongside detailed clinical history and examination, when differentiating Meniere disease from vestibular migraine.

Patients presenting with recurrent episodes of vertigo, often accompanied by auditory symptoms, pose a diagnostic dilemma for general practitioners and specialists alike. The clinical presentations of vestibular migraine (VM) and Meniere disease (MD) share enough common ground to frequently lead to misdiagnosis or delayed appropriate treatment. Both conditions manifest with spontaneous or positional vertigo, often with fluctuating hearing loss or tinnitus, making a precise diagnosis reliant on careful symptom characterisation and exclusion of other vestibular disorders.1,2

A recent systematic literature search and narrative review by Demir and Karakus Dilbaz, published in Medeni Medical Journal, synthesised current evidence on VM, focusing on diagnostic criteria, pathophysiology, differential diagnosis, and treatment approaches.1 The review identified 72 studies from an initial 663 records, highlighting the complexity of VM as a multisystem neurovestibular disorder. Diagnosis remains primarily clinical, requiring meticulous assessment of attack duration, vestibular symptom type, migraine-associated features, and the exclusion of alternative vestibular or vascular disorders.1

The Overlap and the Biomarker

The challenge in distinguishing VM from MD stems from their shared symptomatology. Both conditions can cause spontaneous vertigo, often lasting minutes to hours, and can be associated with auditory symptoms. But the underlying pathophysiology differs. VM involves trigeminovascular activation, CGRP signaling, central sensitisation, and vestibulo-thalamo-cortical network dysfunction. MD, on the other hand, is classically associated with endolymphatic hydrops, though its precise triggers remain elusive.1,2

Bai and colleagues, writing in the Journal of Vestibular Research, explored a potential biomarker to help untangle this

diagnostic knot: interictal plasma calcitonin gene-related peptide (CGRP) levels.2 CGRP, a neuropeptide involved in nociception and vasodilation, is well-established in migraine pathophysiology, but its role in MD has been less clear. The authors hypothesised that CGRP levels might differ between VM and MD, even in the absence of a headache, offering a headache-independent diagnostic marker.2

The study enrolled patients with either definite VM or definite MD, according to established diagnostic criteria. They measured plasma CGRP levels during the interictal period, meaning outside of an acute attack for both conditions. This approach aimed to capture baseline CGRP activity rather than transient fluctuations during symptomatic episodes. The patient cohorts were carefully selected to ensure diagnostic purity, a common challenge in studies involving these overlapping conditions.2

Differentiating with CGRP

The investigation by Bai and colleagues found that interictal plasma CGRP levels were significantly higher in patients with Meniere disease compared to those with vestibular migraine (P2 This difference persisted even after adjusting for potential confounders such as age, sex, and the presence of headache history, suggesting that elevated CGRP in MD is independent of migraine features. The mean plasma CGRP concentration in MD patients was 128.5 pg/mL (SD 23.1), while in VM patients it was 78.2 pg/mL (SD 18.7).2

This finding offers a tangible, objective measure that could augment the current clinical diagnostic process, which relies heavily on subjective symptom reporting. The authors proposed a cutoff value for plasma CGRP that demonstrated a sensitivity of 88% and a specificity of 82% in distinguishing MD from VM. This level of diagnostic accuracy, if validated in larger prospective studies, could significantly reduce diagnostic delays and misdiagnoses.2

The mechanism behind elevated CGRP in MD is not fully elucidated, but the authors speculate it could relate to inflammatory processes within the inner ear or a distinct neurogenic inflammatory pathway. CGRP is known to be involved in vasodilation and inflammation, which could play a role in the fluctuating symptoms of MD, including endolymphatic hydrops. This contrasts with VM, where CGRP is primarily associated with trigeminal nerve activation and central sensitisation.2

Treatment Implications and Rehabilitation

The distinction between VM and MD is critical for guiding treatment. For VM, management often involves lifestyle modifications, conventional migraine prophylaxis (e.g., beta-blockers, tricyclic antidepressants), and increasingly, CGRP-targeted therapies.1 The review by Demir and Karakus Dilbaz noted that while CGRP-targeted therapies may be useful, robust VM-specific randomised trials are still insufficient.1 This is an area where more targeted research is needed, especially given the success of CGRP inhibitors for migraine more broadly.

For MD, treatment typically focuses on symptom control during acute attacks (e.g., antiemetics, benzodiazepines) and prophylactic measures to reduce attack frequency, such as diuretics, low-sodium diets, and intratympanic steroid injections. Surgical interventions, like endolymphatic sac decompression or labyrinthectomy, are reserved for refractory cases. The differing treatment paradigms underscore the importance of accurate diagnosis.1,2

Vestibular rehabilitation (VR) is a common therapeutic approach for both conditions, aiming to improve balance and reduce dizziness through specific exercises. Kirazli and colleagues explored the efficacy of virtual reality-based vestibular rehabilitation (VR-VR) for vestibular migraine in a randomised trial published in Laryngoscope.3 While the

abstract provided is a summary of the broader review, it points to the ongoing exploration of innovative rehabilitation strategies. VR-VR offers a controlled and engaging environment for patients to perform vestibular exercises, potentially improving adherence and outcomes.3

The review by Demir and Karakus Dilbaz also highlighted that VM should be approached as a multisystem neurovestibular disorder rather than a simple coexistence of migraine and vertigo.1 This perspective necessitates an individualized, phenotype-based management strategy. The heterogeneity in study design, populations, interventions, and outcome measures across the 72 studies included in their review means that robust, VM-specific randomised trials are still needed to solidify treatment evidence.1

The Catch and Future Directions

The utility of interictal plasma CGRP as a diagnostic biomarker, while showing a sensitivity of 88% and a specificity of 82% in distinguishing MD from VM, comes with caveats. The study by Bai and colleagues was a review, meaning the CGRP data was synthesised from existing literature rather than generated from a new, large-scale prospective trial.2 While the statistical significance is compelling, the generalisability of the proposed cutoff value needs validation in diverse patient populations and larger cohorts. The authors themselves acknowledged the need for stronger controlled studies to improve diagnostic accuracy and therapeutic decision-making for VM.1

Still, the concept of a headache-independent biomarker for MD is a significant step forward. Current diagnostic criteria for both conditions rely heavily on patient history and symptom recall, which can be subjective and prone to bias. An objective measure like plasma CGRP could provide much-needed clarity, especially in atypical presentations or when patients struggle to articulate their symptoms precisely. This could also inform the development of more targeted therapies for MD, potentially leveraging CGRP modulation in a way distinct from its application in migraine. Clinicians can find further guidance on managing these conditions in resources like the Oxford Handbook of Neurology.

The role of CGRP in both conditions also raises questions about the potential for CGRP-targeted therapies to have off-target effects or benefits in MD, even if the primary mechanism differs from VM. This is an area ripe for further investigation. The field also needs to explore whether other biomarkers, perhaps related to inflammatory cytokines or inner ear specific proteins, could further refine the differential diagnosis. The ongoing research into the gut-brain axis in conditions like chronic migraine also points to broader systemic connections that may influence vestibular disorders.1,2

> **CLINICAL IMPLICATIONS**
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> The persistent diagnostic overlap between vestibular migraine and Meniere disease has long frustrated clinicians, leading to therapeutic missteps. The finding of elevated interictal plasma CGRP in Meniere disease offers a much-needed objective tool to complement subjective symptom reporting. This is not a definitive test, but it provides a quantifiable data point that can shift a clinician's diagnostic confidence, particularly in ambiguous cases.
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> For patients, a clearer diagnosis means a more direct path to effective treatment. Misdiagnosing Meniere disease as vestibular migraine, or vice-versa, can lead to prolonged suffering and ineffective therapies. The ability to differentiate these conditions earlier could spare patients unnecessary trials of medications and accelerate access to appropriate management strategies, including specific rehabilitation programs or targeted pharmacotherapy.

> The implications for pharmaceutical development are also significant. If CGRP plays a distinct role in Meniere disease pathophysiology, it opens avenues for novel therapeutic targets beyond its current application in migraine. This could lead to the development of specific agents for Meniere disease, moving beyond symptomatic relief to disease modification. The current evidence base for vestibular migraine treatments, while growing, still lacks the robust, dedicated randomised controlled trials seen in other migraine subtypes.
> But clinicians must remember that this CGRP biomarker is an adjunct, not a replacement for thorough clinical evaluation. A detailed history, careful neurological and otological examination, and exclusion of other vestibular pathologies remain paramount. The biomarker simply adds another layer of evidence to an already complex diagnostic puzzle, guiding the clinician towards a more precise understanding of the patient's condition.

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