# Do CGRP Monoclonal Antibodies Offer Relief for Vestibular Migraine Vertigo?

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Vestibular migraine presents a significant diagnostic and therapeutic challenge, often leaving patients with debilitating vertigo attacks that severely impact quality of life. While calcitonin gene-related peptide (CGRP) monoclonal antibodies have transformed the prophylactic treatment of episodic and chronic migraine, their role in specifically addressing the vestibular symptoms of migraine has been a subject of ongoing clinical interest. The question remains whether these targeted therapies, effective for headache, can extend their benefit to the complex sensory disturbances of vestibular migraine.

**KEY TAKEAWAYS**

- The Pivot: CGRP monoclonal antibodies, while effective for headache-dominant migraine, are being investigated for their impact on the vertigo component of vestibular migraine.
- The Data: Clinical experience suggests a reduction in vertigo attack frequency and severity for some patients, but dedicated, large-scale trials are still needed to quantify this effect precisely.
- The Action: Clinicians should consider CGRP monoclonal antibodies for vestibular migraine patients with frequent, severe vertigo attacks, particularly when conventional therapies have failed, while acknowledging the current evidence base.

Vestibular migraine, a common cause of recurrent spontaneous vertigo, affects a substantial portion of the population, often overlapping with classic migraine symptoms but sometimes presenting primarily with vestibular complaints. The condition is characterised by episodes of vertigo, dizziness, and imbalance, which can last from minutes to days, often accompanied by migraine-associated symptoms such as headache, photophobia, phonophobia, and aura. Diagnosing vestibular migraine can be complex, as its symptoms mimic other vestibular disorders, including Meniere's disease and benign paroxysmal positional vertigo (BPPV). The diagnostic criteria require a history of at least five episodes of vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours, in a patient with a current or past history of migraine, and at least half of the vestibular episodes must be associated with migraine features. This clinical entity represents a significant unmet need, as existing prophylactic treatments, largely borrowed from headache migraine management, often yield inconsistent results for the vestibular component.

Current management strategies for vestibular migraine typically involve lifestyle modifications, dietary changes, and pharmacological prophylaxis using medications such as beta-blockers, tricyclic antidepressants, and antiepileptic drugs. These treatments aim to reduce the frequency and severity of both headache and vertigo attacks. But many patients experience inadequate relief or intolerable side effects, highlighting the need for more targeted and effective therapies.

The pathophysiology of vestibular migraine is thought to involve a complex relationship between cortical spreading

depression, central vestibular pathways, and neuroinflammation, with CGRP playing a potential role in modulating these processes. CGRP, a neuropeptide widely distributed in the central and peripheral nervous systems, is implicated in nociceptive transmission and vasodilation, both key elements in migraine pathogenesis. Its role in vestibular function, while less understood, is an area of active investigation.

## The CGRP Pathway and Vestibular Function

CGRP monoclonal antibodies (mAbs) target either the CGRP ligand itself or its receptor, thereby preventing CGRP from binding and activating its pathway. This mechanism has proven highly effective in reducing migraine attack frequency and severity in patients with episodic and chronic migraine. The four CGRP mAbs currently available for migraine prophylaxis in Europe are erenumab, fremanezumab, galcanezumab, and eptinezumab. Each of these agents has demonstrated robust efficacy in large-scale clinical trials for headache-dominant migraine, leading to their widespread adoption. But the question of whether this efficacy extends to the specific vestibular symptoms of migraine is more complex. The vestibular system, comprising the inner ear and its central connections, is intimately linked with brain regions involved in migraine processing. Activation of trigeminal pathways, a hallmark of migraine, can influence vestibular nuclei, potentially contributing to vertigo. CGRP is present in these trigeminal ganglia and in central vestibular structures, suggesting a plausible biological basis for its involvement in vestibular migraine.

The precise mechanism by which CGRP mAbs might influence vestibular symptoms is still being elucidated. It is hypothesised that by modulating CGRP activity, these antibodies could reduce neuroinflammation, stabilise neuronal excitability in vestibular pathways, or indirectly impact cortical spreading depression, which is believed to underlie both migraine aura and potentially some vestibular symptoms. The effect might not be a direct suppression of vestibular input but rather a normalisation of central processing that becomes dysregulated during a vestibular migraine attack. Understanding the complex relationship of neurological pathways in chronic migraine could offer further insights into this mechanism.

## Clinical Experience and Emerging Data

While dedicated, large-scale randomised controlled trials specifically powered to assess the effect of CGRP mAbs on vertigo attacks in vestibular migraine are still limited, accumulating clinical experience and smaller observational studies offer some insights. Many clinicians have observed that patients receiving CGRP mAbs for headache migraine who also suffer from vestibular migraine report a reduction in both headache and vertigo symptoms. This anecdotal evidence, while not definitive, suggests a broader therapeutic effect beyond headache alone. Some retrospective analyses of patient registries and case series have indicated that CGRP mAbs can lead to a decrease in the frequency, duration, and intensity of vertigo attacks in individuals with vestibular migraine. These observations, though not from prospective, placebo-controlled trials, provide a basis for further investigation.

The challenge in conducting such trials lies in the heterogeneity of vestibular migraine presentation and the difficulty in objectively quantifying vertigo. Unlike headache frequency, which is relatively straightforward to measure, vertigo is a subjective experience with varying characteristics. Developing standardised outcome measures for vestibular symptoms, including validated patient-reported outcome measures (PROMs) and objective vestibular function tests, is critical for future research. The current evidence, therefore, relies heavily on patient self-reporting and clinician assessment, which can introduce variability. Still, the consistent pattern of improvement reported by patients and clinicians is compelling

enough to warrant continued exploration. For a broader understanding of managing dizziness, clinicians might consult resources on vertigo in primary care.

## Patient Selection and Practical Considerations

In the absence of definitive trial data, patient selection for CGRP mAb therapy in vestibular migraine often mirrors that for headache-dominant migraine. Patients with frequent and severe vertigo attacks, particularly those who have failed multiple conventional prophylactic treatments, are typically considered candidates. The decision to initiate CGRP mAb therapy should involve a thorough discussion of the potential benefits and the current limitations of the evidence base for vestibular symptoms. Clinicians should set realistic expectations with patients, emphasising that while headache improvement is well-established, the impact on vertigo may vary. Monitoring for improvement in vertigo attack frequency, severity, and associated symptoms, alongside headache parameters, is essential. This requires detailed patient diaries and regular follow-up. The safety profile of CGRP mAbs has been generally favourable in migraine trials, with common adverse events including injection site reactions, constipation, and nasopharyngitis. Serious adverse events are rare. But the long-term safety data, particularly in specific subgroups, continues to evolve. Some concerns have been raised regarding potential cardiovascular effects, especially in high-risk patients, a topic that has been explored in discussions about CGRP inhibitors and cardiovascular safety.

The cost of CGRP mAbs is a significant factor in their accessibility and utilisation. Reimbursement policies vary across European countries, often requiring patients to have failed a certain number of oral prophylactic treatments before qualifying for CGRP mAb therapy. This can delay access for patients who might benefit, particularly those whose primary burden is vestibular rather than headache. The administrative hurdles can be substantial, requiring careful documentation of prior treatment failures and symptom severity. The potential for CGRP monoclonal antibodies to be linked to fracture risk is another area requiring ongoing vigilance and further research, though the evidence remains observational and requires confirmation.

## Where the Evidence Falls Short

The primary limitation in definitively answering whether CGRP mAbs reduce vertigo attacks in vestibular migraine is the lack of large, dedicated, placebo-controlled trials. Most of the current understanding is extrapolated from headache migraine studies or derived from real-world observational data and case series. These sources, while valuable for generating hypotheses, cannot establish causality or provide precise effect sizes for vertigo reduction. The heterogeneity of vestibular migraine itself also complicates research. Patients present with a wide spectrum of vestibular symptoms, from mild dizziness to severe rotational vertigo, and the duration and frequency of these attacks vary considerably. This makes it challenging to define a uniform primary endpoint for clinical trials. The subjective nature of vertigo also poses a measurement challenge, requiring robust and validated patient-reported outcomes that are not always consistently applied across studies. The absence of a clear biomarker for vestibular migraine further hinders objective assessment of treatment response. Still, the clinical need is undeniable, and the biological plausibility for CGRP involvement in vestibular migraine pathology remains strong. Future research must focus on designing trials specifically for this patient population, employing rigorous methodology and appropriate outcome measures to provide definitive answers.

**CLINICAL IMPLICATIONS**

The current market for vestibular migraine treatment is one of cautious optimism, tempered by a clear gap in robust evidence. Clinicians are left to extrapolate from headache migraine data and anecdotal experience, which, while suggestive, is not the same as a dedicated, well-powered trial. For patients suffering from debilitating vertigo, the prospect of a new, targeted therapy is compelling, but the lack of specific data means prescribing decisions often involve a degree of clinical judgment beyond the usual evidence-based threshold.
The industry has a clear imperative to invest in trials specifically designed for vestibular migraine. The current reliance on post-hoc analyses or observational data is insufficient for a condition that significantly impairs quality of life. A clear understanding of efficacy for vertigo, distinct from headache, would not only benefit patients but also streamline reimbursement processes, which currently often struggle to accommodate off-label use based on indirect evidence.
For European GPs and specialists, the CGRP monoclonal antibodies represent a valuable addition to the migraine prophylactic armamentarium. When conventional therapies fail to control both headache and vertigo in vestibular migraine, these agents offer a plausible next step. But careful patient selection and diligent monitoring of vestibular symptoms are paramount, given the current limitations in the evidence base. The Oxford Handbook of Neurology provides a useful quick reference for managing complex neurological conditions like this.
The unanswered question remains: what specific patient phenotypes within vestibular migraine respond best to CGRP inhibition? Future research needs to move beyond simply demonstrating an effect and begin to identify biomarkers or clinical characteristics that predict a favourable response, allowing for more precise and personalised treatment strategies.

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