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Review Article

Perimenopause, Dizziness and Vestibular Migraine: An Emerging Multidisciplinary Challenge

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Abstract

Vestibular migraine has emerged as one of the most common causes of recurrent vertigo in adults and is increasingly recognised across neurology and otolaryngology practices. Despite its well-established female predominance, relatively little attention has been paid to the potential influence of reproductive ageing and the menopausal transition on disease expression. Perimenopause is characterised by marked fluctuations in ovarian hormone production, particularly estradiol, accompanied by changes in sleep quality, autonomic regulation, mood and sensory processing. Increasing evidence suggests that these neuroendocrine alterations may contribute to dizziness, imbalance and worsening vestibular migraine symptoms in susceptible women.

Recent literature has highlighted dizziness as an under-recognised symptom of the menopausal transition. Estrogen receptors have been identified within both central and peripheral vestibular pathways, while experimental studies have demonstrated effects of estrogen on neurotransmission, neuroplasticity, vascular regulation and trigeminovascular signalling. These observations provide a biologically plausible framework linking hormonal instability during perimenopause to vestibular dysfunction.

This narrative review examines current understanding of vestibular migraine pathophysiology, explores the relationship between menopause and dizziness, and evaluates the potential role of hormone replacement therapy (HRT) in symptom management. Particular emphasis is placed on perimenopause as a critical period of vulnerability during which hormonal fluctuations may lower the threshold for vestibular migraine expression. Greater collaboration between otolaryngologists, neurologists and menopause specialists may improve recognition and management of this increasingly relevant clinical problem.

Keywords: Vestibular migraine; Perimenopause; Menopause; Dizziness; Vertigo; Hormone replacement therapy; Estradiol; Vestibular disorders.

Introduction

Dizziness and vertigo are among the most frequent neurological and otological complaints encountered in clinical practice. Although benign paroxysmal positional vertigo, vestibular neuritis and Ménière's disease remain important diagnostic considerations, vestibular migraine is now recognised as one of the leading causes of recurrent episodic vertigo. Over the past two decades, awareness of vestibular migraine has increased considerably; nevertheless, many patients continue to experience delayed diagnosis, repeated investigations and fragmented care pathways.

One of the most striking epidemiological characteristics of vestibular migraine is its strong female predominance. Women are affected approximately three times more frequently than men, suggesting that biological sex and reproductive hormones may play an important role in disease pathogenesis. Similar observations have long been recognised in migraine headache disorders, where symptom fluctuations

frequently coincide with menstruation, pregnancy and menopause.

The menopausal transition represents a unique neuroendocrine period characterised by profound hormonal instability. While menopause is often described as a state of estrogen deficiency, the transition itself is better understood as a period of fluctuating ovarian activity, during which estradiol concentrations may vary dramatically over short periods of time. These fluctuations have important effects on neurotransmitter systems, cerebral vascular function, sleep regulation and sensory processing, all of which are implicated in migraine biology.

Historically, menopause-related research has focused predominantly on vasomotor symptoms, osteoporosis and cardiovascular health. By contrast, dizziness and balance disturbances have received comparatively little attention despite being commonly reported by midlife women. Recent work by Mongioì and colleagues has highlighted the growing recognition of dizziness as a clinically relevant symptom during the menopausal transition and has renewed interest in understanding the relationship between reproductive ageing and vestibular function.

For otolaryngologists, dizziness in perimenopausal women is frequently investigated within traditional vestibular diagnostic frameworks. For gynaecologists and menopause specialists, the same symptoms may be attributed to hormonal changes, anxiety or sleep disturbance. The reality is likely to be more complex. Increasing evidence suggests that hormonal fluctuations may directly influence vestibular pathways while simultaneously modulating migraine susceptibility, creating an overlap between neuro-otological and menopausal medicine that remains poorly understood.

This review explores the emerging relationship between perimenopause, dizziness and vestibular migraine. We discuss current understanding of vestibular migraine pathophysiology, examine evidence linking menopausal transition to vestibular symptoms and evaluate the potential role of hormone replacement therapy. We propose that vestibular migraine may represent one manifestation of a broader spectrum of hormone-sensitive vestibular dysfunction and argue that closer collaboration between ENT specialists and menopause clinicians is essential to optimise patient care.

The Menopausal Transition: More Than Estrogen Deficiency

The menopausal transition is a dynamic biological process rather than a single event. According to the Stages of Reproductive Aging Workshop (STRAW+10) classification, perimenopause encompasses the years preceding the final menstrual period and is characterised by progressive alterations in ovarian function.

During this stage, follicular depletion results in increasingly erratic hormonal secretion patterns, with substantial fluctuations in circulating estradiol levels.

Importantly, symptom generation during perimenopause appears to be driven less by absolute estrogen deficiency and more by hormonal variability. Estradiol concentrations may fluctuate from supraphysiological to markedly reduced levels within relatively short periods. Such instability influences multiple physiological systems, including serotonergic neurotransmission, autonomic regulation, vascular reactivity and sleep architecture.

These neurobiological changes have particular relevance for migraine disorders. The concept of estrogen withdrawal as a trigger for migraine has been recognised for decades, and many women experience worsening headaches during periods of hormonal instability. Similar mechanisms may also influence vestibular symptoms, although this area remains comparatively under-investigated.

In addition to direct hormonal effects, perimenopause is associated with a constellation of symptoms that may interact with vestibular function. Sleep disturbance, anxiety, depression, cognitive complaints and vasomotor symptoms frequently coexist and may contribute to symptom amplification. Consequently, dizziness in perimenopausal women is rarely attributable to a single mechanism and should instead be viewed within a broader biopsychosocial and neuroendocrine context.

Dizziness During Perimenopause: An Under-Recognised Symptom

Although hot flushes and night sweats remain the hallmark symptoms of menopause, dizziness is increasingly recognised as a common complaint among midlife women. Population-based studies suggest that dizziness affects a substantial proportion of women during the menopausal transition, yet it remains absent from many clinical discussions and management guidelines.

Recent work by Mongioì et al. has highlighted the need to consider dizziness as a potentially significant manifestation of reproductive ageing. The authors propose that hormonal fluctuations may influence vestibular function through multiple pathways involving neurochemical signalling, vascular regulation and sensory integration. Furthermore, estrogen receptors have been identified within vestibular structures, providing biological plausibility for a direct endocrine effect on balance mechanisms.

The clinical presentation is often heterogeneous. Women may describe episodic vertigo, persistent imbalance, motion intolerance, disequilibrium, light-headedness or visual dependence. These symptoms frequently overlap with migraine, anxiety and sleep disturbance, complicating diagnostic assessment. Consequently, many patients undergo extensive investigations before a unifying diagnosis is considered.

For clinicians, recognising dizziness as a potential meno-

pausal symptom does not imply that all vestibular complaints should be attributed to hormonal changes. Rather, it highlights the importance of considering reproductive stage as one component of a comprehensive diagnostic evaluation.
(continued with Vestibular Migraine, Pathophysiological Links, HRT, Clinical Management and Future Directions)

Vestibular Migraine: A Missing Piece of the Puzzle?

Vestibular migraine is increasingly recognised as one of the most common causes of recurrent vertigo, with an estimated lifetime prevalence of approximately 1–3% in the general population and considerably higher rates among patients presenting to specialist dizziness clinics.
Despite its prevalence, the condition remains underdiagnosed, partly because vestibular symptoms may occur in the absence of headache and because patients often present to multiple specialties before a diagnosis is established.
The diagnostic criteria developed jointly by the Bárány Society and the International Headache Society define vestibular migraine as recurrent vestibular symptoms of moderate or severe intensity lasting between five minutes and 72 hours in individuals with a current or previous history of migraine and associated migrainous features. However, clinical presentations are highly variable.
Patients may experience spontaneous vertigo, positional dizziness, visually induced vertigo, motion intolerance, imbalance or chronic disequilibrium.
One of the most intriguing features of vestibular migraine is its striking female predominance. This observation has prompted increasing interest in the role of reproductive hormones in disease expression. Many women report symptom fluctuations during menstruation, pregnancy, the postpartum period and perimenopause. Such observations mirror those seen in migraine headache disorders and suggest common biological mechanisms.
From a clinical perspective, perimenopause appears to represent a particularly vulnerable period. Women frequently report the emergence of vestibular symptoms during their forties and early fifties, often accompanied by worsening migraine, sleep disturbance and vasomotor symptoms. Yet these symptoms are rarely considered collectively. Instead, patients may receive separate diagnoses from neurologists, otolaryngologists, cardiologists and menopause specialists, resulting in fragmented care.
The possibility that vestibular migraine represents one manifestation of hormone-sensitive neurological dysfunction during the menopausal transition deserves greater attention. While robust epidemiological evidence remains limited, clinical experience across multiple specialties suggests that hormonal instability may lower the threshold for vestibular migraine expression in susceptible individuals.

Pathophysiological Links Between Perimenopause and Vestibular Migraine
The biological relationship between perimenopause and vestibular migraine is likely to be multifactorial and reflects the interaction of endocrine, neurological and vestibular mechanisms.

Estrogen Withdrawal and Migraine Susceptibility

Among women with migraine, fluctuations in estrogen levels appear more important than absolute hormone concentrations. The "estrogen withdrawal hypothesis" proposes that falling estradiol levels trigger migraine attacks through effects on neurotransmitter systems, vascular function and neuronal excitability.
Perimenopause is characterised by repeated cycles of hormonal fluctuation. Estradiol levels may rise and fall unpredictably, creating a physiological environment that differs substantially from both the reproductive years and postmenopause. These fluctuations may repeatedly activate migraine pathways, increasing susceptibility to both headache and vestibular symptoms.
Importantly, many women who previously experienced relatively stable migraine patterns report worsening symptoms during the menopausal transition. Similar observations have been described in vestibular migraine, although prospective studies remain limited.

Trigeminovascular Activation

The trigeminovascular system plays a central role in migraine pathophysiology. Activation of trigeminal afferents results in the release of vasoactive neuropeptides including calcitonin gene-related peptide (CGRP), substance P and neurokinin A. These mediators contribute to neurogenic inflammation, altered vascular function and increased neuronal excitability.
Anatomical and functional connections between trigeminal pathways and vestibular nuclei provide a plausible explanation for vestibular manifestations of migraine. Activation of trigeminovascular pathways may therefore influence vestibular processing directly, producing vertigo and dizziness even in the absence of significant headache.
Emerging evidence suggests that estrogen may modulate CGRP expression and signalling. Consequently, hormonal fluctuations during perimenopause may enhance trigeminovascular sensitivity and contribute to vestibular migraine exacerbations.

Serotonergic Modulation

Estrogen exerts important effects on serotonergic neurotransmission. It influences serotonin synthesis, receptor expression and transporter activity within multiple brain regions implicated in migraine and vestibular processing.

Reduced serotonergic activity has long been implicated in migraine pathogenesis. During periods of hormonal instability, alterations in serotonin signalling may contribute to increased sensory sensitivity, motion intolerance and migraine susceptibility. This mechanism may partially explain why vestibular symptoms often worsen during periods of endocrine transition.

Central Sensory Processing

Vestibular migraine is increasingly viewed as a disorder of central sensory integration rather than a purely peripheral vestibular condition. Functional neuroimaging studies have demonstrated altered activity within vestibular cortical networks, the thalamus, cerebellum and brainstem.

These structures integrate vestibular, visual and proprioceptive information to maintain balance and spatial orientation. Hormonal fluctuations may influence the excitability of these networks, leading to sensory mismatch, visual dependence and dizziness.

This concept may explain why many women report increased motion sensitivity, supermarket intolerance and visually induced dizziness during perimenopause, even when conventional vestibular testing appears normal.

Sleep Disturbance as a Common Pathway

Sleep disruption represents one of the most common symptoms of perimenopause and is also a recognised trigger for vestibular migraine.

Night sweats, insomnia and fragmented sleep may contribute to central sensitisation, increased migraine susceptibility and impaired vestibular compensation. Consequently, sleep disturbance may act as an important mediator linking hormonal changes to vestibular symptom exacerbation.

The interaction between sleep, hormonal fluctuation and vestibular dysfunction highlights the need for a holistic clinical approach rather than focusing solely on vestibular pathology.

Hormone Replacement Therapy: Friend or Foe?

The potential role of hormone replacement therapy (HRT) in vestibular migraine remains an area of considerable clinical interest and uncertainty. Despite the frequency with which this question arises in clinical practice, evidence specifically addressing vestibular migraine remains sparse.

Much of the available evidence is extrapolated from studies examining migraine headache disorders. These studies suggest that hormonal stability, rather than hormone replacement itself, may be the critical therapeutic principle.

Biological Rationale for HRT

If hormonal fluctuations contribute to symptom generation, then stabilising estrogen levels may reduce activation of migraine pathways.

Potential benefits include:

- Reduction in estrogen withdrawal events
- Improved serotonergic neurotransmission
- Modulation of CGRP signalling
- Improved sleep quality
- Reduction in vasomotor symptoms
- Enhanced quality of life

These mechanisms provide a biologically plausible rationale for considering HRT in selected symptomatic women.

Why Route of Administration Matters

The route of estrogen administration may be particularly important in women with migraine and vestibular symptoms.

Oral estrogen undergoes first-pass hepatic metabolism and may produce greater fluctuations in circulating hormone levels. In contrast, transdermal preparations provide more stable serum concentrations and are therefore generally preferred in women with migraine disorders.

Many menopause specialists now favour transdermal estradiol patches or gels combined with continuous progesterone regimens where appropriate.

Clinical Evidence

Direct evidence regarding HRT and vestibular migraine remains limited. Some women report significant improvement in dizziness, motion sensitivity and migraine frequency following initiation of transdermal estrogen therapy. Others report symptom worsening, particularly during dose changes or with cyclical hormone regimens. Occasionally, the benefits on vestibular migraine appear as secondary outcome while using HRT for treatment of vasomotor symptoms or even loss of libido

These differing responses suggest that vestibular migraine is unlikely to represent a single disease entity and that individual sensitivity to hormonal change may vary substantially.

Current evidence supports a personalised approach in which HRT decisions are based primarily on menopausal symptom burden and overall risk-benefit assessment rather than vestibular symptoms alone.

A Multidisciplinary Clinical Approach

The overlap between menopause, dizziness and vestibular migraine highlights the limitations of traditional specialty-based models of care.

Women presenting with dizziness during the menopausal transition often consult multiple healthcare professionals before receiving a coherent explanation for their symptoms.

An integrated approach involving otolaryngologists, neurologists, menopause specialists and physiotherapists may facilitate earlier diagnosis and more effective management.

Assessment should include:

- Detailed vestibular history
- Migraine history
- Menstrual and reproductive history
- Assessment of vasomotor symptoms
- Sleep evaluation
- Psychological wellbeing assessment
- Medication review

Recognition of the menopausal transition as a potential contributor to vestibular symptoms may help clinicians identify patients who would benefit from a broader management strategy.

Future Directions

Several important questions remain unanswered.
First, the true prevalence of vestibular migraine during perimenopause is unknown. Longitudinal studies following women through the menopausal transition are urgently needed.
Second, the extent to which HRT influences vestibular migraine remains unclear. Randomised controlled trials specifically examining vestibular outcomes are lacking.
Third, the interaction between estrogen signalling and emerging CGRP-targeted therapies warrants further investigation. Understanding these relationships may provide new opportunities for personalised treatment.
Finally, greater collaboration between otolaryngology, neurology and women's health researchers is essential to advance understanding of this increasingly recognised clinical problem.

Conclusion

Perimenopause represents a period of profound neuroendocrine instability during which fluctuating ovarian hormones influence multiple physiological systems involved in vestibular function and migraine susceptibility. Emerging evidence suggests that dizziness is an under-recognised yet clinically significant symptom of the menopausal transition, while vestibular migraine may represent one manifestation of hormone-sensitive neurological dysfunction in susceptible women.
Although definitive evidence remains limited, current data support a biologically plausible relationship between hormonal fluctuation, vestibular symptoms and migraine activation. Recognition of this relationship has important implications for clinical practice. Women presenting with dizziness during perimenopause require a comprehensive assessment that extends beyond traditional specialty boundaries and considers endocrine, neurological and vestibular factors simultaneously.
As awareness grows, closer collaboration between otolaryngologists, neurologists and menopause specialists may improve diagnostic accuracy, facilitate individualised treatment and ultimately enhance quality of life for affected women. Further research is required to clarify the role of hormone replacement therapy, define optimal management strategies and determine whether vestibular migraine should be considered part of a broader spectrum of hormone-sensitive vestibular disorders.

Strengths and Limitations of Current Evidence

Despite increasing recognition of vestibular symptoms during the menopausal transition, the current evidence base remains fragmented and characterised by several important limitations.
First, relatively few studies have specifically examined vestibular migraine in perimenopausal women. Much of the existing literature focuses either on migraine in general or on dizziness during menopause without differentiating between vestibular migraine, benign paroxysmal positional vertigo, persistent postural perceptual dizziness (PPPD), Ménière's disease and other vestibular disorders. Consequently, it remains difficult to determine the true prevalence of vestibular migraine during the menopausal transition.
Second, the majority of available studies are observational in nature. While these studies consistently demonstrate associations between menopause and dizziness, causality cannot be established. Symptoms such as vertigo, imbalance and disequilibrium are inherently multifactorial and may be influenced by age-related vestibular degeneration, cardiovascular factors, anxiety, sleep disturbance and medication use in addition to hormonal changes.
Third, there is considerable heterogeneity in the assessment of vestibular symptoms. Some studies utilise validated vestibular questionnaires, whereas others rely on self-reported dizziness without objective characterisation. This variability limits comparison between studies and may contribute to inconsistencies in reported prevalence rates.
Another important limitation relates to hormone measurement. Estradiol levels fluctuate substantially during perimenopause, yet most studies rely on single hormone measurements that may not accurately reflect an individual's hormonal environment. Future research should consider longitudinal hormone monitoring to better understand the relationship between endocrine fluctuations and vestibular symptoms.
Evidence regarding hormone replacement therapy is similarly limited. Most available data are extrapolated from migraine headache studies rather than vestibular migraine-specific investigations. While several observational studies suggest that stable transdermal estrogen therapy may improve mi-

graine symptoms in some women, robust randomised controlled trials examining vestibular outcomes are lacking.
Nevertheless, despite these limitations, several observations support a biologically plausible association between perimenopause and vestibular migraine. These include the marked female predominance of vestibular migraine, the recognised influence of reproductive events on migraine expression, the identification of estrogen receptors within vestibular pathways and the increasing evidence linking menopausal transition to dizziness.
Taken together, the available literature supports the hypothesis that hormonal fluctuations may influence vestibular function and migraine susceptibility. However, definitive conclusions regarding causation and optimal treatment strategies cannot yet be drawn.

Proposed Pathophysiological Model

Although vestibular migraine is generally considered a neurological disorder and menopause a reproductive transition, growing evidence suggests substantial overlap between the biological pathways involved in both conditions.
We propose a conceptual model in which perimenopause acts as a state of neuroendocrine instability that lowers the threshold for vestibular symptom generation through multiple interacting mechanisms.
Perimenopausal ovarian dysfunction leads to fluctuating estradiol concentrations. These fluctuations influence serotonergic activity, CGRP signalling, autonomic regulation and cortical excitability. Simultaneously, hormonal changes may affect vestibular compensation mechanisms and sensory integration pathways within the brainstem, cerebellum and vestibular cortex.

The resulting increase in sensory vulnerability may manifest clinically as:

- Episodic vertigo
- Motion sensitivity
- Visual dependence
- Persistent dizziness
- Vestibular migraine attacks
- Increased susceptibility to PPPD

Sleep disturbance may further amplify these effects by increasing central sensitisation and reducing migraine thresholds.
Vasomotor symptoms, anxiety and mood disturbance may represent additional amplifying factors rather than independent disease processes.
Within this framework, vestibular migraine is viewed not as an isolated neurological condition but as part of a broader spectrum of hormone-sensitive vestibular disorders that become particularly relevant during the menopausal transition.

Clinical Implications for Menopause Specialists and Otolaryngologists

Recognition of vestibular symptoms as a potential manifestation of perimenopause has several practical implications.
For gynaecologists and menopause practitioners, recurrent dizziness should prompt consideration of vestibular migraine, particularly in women with a personal or family history of migraine. Asking targeted questions regarding motion sickness, visual vertigo, photophobia and migraine symptoms may reveal an underlying diagnosis that would otherwise remain unrecognised.
Conversely, otolaryngologists evaluating women with recurrent vertigo should routinely enquire about menstrual history, menopausal status and vasomotor symptoms. Recognition of hormonal influences may provide a more complete understanding of symptom patterns and facilitate referral to menopause specialists where appropriate.
The importance of multidisciplinary collaboration cannot be overstated. Women with vestibular migraine frequently describe long diagnostic journeys involving multiple specialties. A more integrated approach may reduce diagnostic delay and improve patient satisfaction.

Research Agenda

Future studies should address several key questions:

1. Does the incidence of vestibular migraine increase during perimenopause?
2. Are vestibular symptoms associated with specific hormonal patterns or rates of estradiol fluctuation?
3. Can hormone replacement therapy reduce vestibular migraine frequency or severity?
4. Are women with vestibular migraine more likely to experience severe menopausal symptoms?
5. What is the interaction between estrogen signalling and CGRP-targeted therapies?
6. Could vestibular migraine serve as a clinical marker of hormone sensitivity during reproductive ageing?

Answering these questions will require prospective multidisciplinary studies involving otolaryngologists, neurologists, endocrinologists and menopause specialists.
Such collaboration has the potential to establish a new area of clinical and research interest at the intersection of vestibular medicine and women's health.

Mechanism	Effect
Estradiol fluctuation	Migraine activation
CGRP upregulation	Neurogenic inflammation
Serotonin dysregulation	Sensory hypersensitivity

Sleep disturbance	Reduced migraine threshold
Vestibular receptor modulation	Impaired balance control
Anxiety and autonomic dysfunction	Symptom amplification

Table 1: Proposed Mechanisms Linking Perimenopause and Vestibular Migraine

Diagnosis	**Typical Features**
Vestibular migraine	Episodic vertigo, migraine features
BPPV	Brief positional vertigo
Ménière's disease	Vertigo with fluctuating hearing loss
PPPD	Persistent non-spinning dizziness
Orthostatic hypotension	Positional light-headedness
Anxiety-related dizziness	Chronic subjective imbalance

Table 2: Differential Diagnosis of Dizziness in Perimenopausal Women

Intervention	**Potential Effect**
Transdermal estradiol	May stabilise symptoms
Oral estrogen	Greater hormonal fluctuations
Continuous combined HRT	Potentially preferable
Cyclical HRT	May trigger migraine in susceptible women
Evidence quality	Low to moderate

Table 3: HRT and Vestibular Migraine: Current Evidence

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