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Vitamin D deficiency in benign paroxysmal positional vertigo: current evidence, molecular mechanisms, and clinical implications

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Abstract:

Benign paroxysmal positional vertigo (BPPV) is the most common peripheral vestibular disorder, accounting for approximately 20–30% of patients presenting with vertigo in specialized neurotology clinics. Although canalith repositioning maneuvers achieve rapid symptom resolution in most cases, recurrence remains a major clinical challenge, occurring in 15–50% of patients within the first years following treatment. This high recurrence rate suggests that BPPV should no longer be regarded solely as a mechanical disorder caused by displaced otoconia, but rather as the clinical manifestation of an underlying disturbance in otoconial homeostasis.

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Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common peripheral vestibular disorder, accounting for approximately 20–30% of patients presenting with vertigo in specialized neurotology clinics. Although canalith repositioning maneuvers achieve rapid symptom resolution in most cases, recurrence remains a major clinical challenge, occurring in 15–50% of patients within the first years following treatment. This high recurrence rate suggests that BPPV should no longer be regarded solely as a mechanical disorder caused by displaced otoconia, but rather as the clinical manifestation of an underlying disturbance in otoconial homeostasis.

Otoconia are dynamic biomineralized structures that undergo continuous remodeling throughout life. Their structural integrity depends on tightly regulated calcium metabolism, preservation of the organic protein matrix, and maintenance of a highly specialized endolymphatic microenvironment. Ageing, osteoporosis, estrogen deficiency, and disorders of calcium metabolism have all been implicated in disrupting this delicate balance, thereby increasing the susceptibility of otoconia to degeneration, fragmentation, and detachment from the utricular macula.

Among the metabolic factors currently attracting increasing attention, vitamin D has emerged as a key regulator linking systemic calcium metabolism to vestibular physiology. Beyond its classical role in skeletal mineralization and intestinal calcium absorption, vitamin D directly regulates the expression of epithelial calcium transport proteins within the inner ear, including transient receptor potential vanilloid channels (TRPV5 and TRPV6) and calcium-binding proteins that are essential for maintaining endolymphatic calcium homeostasis. Dysregulation of these molecular pathways may impair otoconial biomineralization, accelerate age-related degeneration, and compromise the physiological turnover of otoconia.

Over the past decade, growing experimental, histopathological, and clinical evidence has strengthened the hypothesis that vitamin D deficiency contributes to both the development and recurrence of BPPV. Several observational studies have consistently demonstrated lower serum 25-hydroxyvitamin D [25(OH)D] concentrations in patients with recurrent BPPV, while randomized controlled trials have suggested that vitamin D supplementation may significantly reduce recurrence in vitamin D-deficient individuals. Nevertheless, conflicting epidemiological findings and methodological heterogeneity continue to prevent definitive conclusions regarding causality.

This review critically summarizes the current evidence regarding the association between vitamin D deficiency and BPPV, with particular emphasis on the molecular mechanisms governing otoconial homeostasis, the available clinical evidence, and the potential implications for disease prevention and long-term management.

Molecular Mechanisms Linking Vitamin D Deficiency to BPPV

Although BPPV has traditionally been explained by the mechanical displacement of otoconia into one or more semicircular canals, this concept does not explain why otoconia detach from the utricle or why recurrence is particularly frequent in elderly individuals, postmenopausal women, and patients with osteoporosis. Increasing evidence indicates that BPPV represents the final clinical manifestation of progressive otoconial degeneration secondary to impaired vestibular calcium homeostasis.

Otoconia are highly specialized extracellular biominerals composed predominantly of calcium carbonate crystals organized around a complex organic matrix rich in otoconin-90 (OC90), the principal structural glycoprotein responsible for crystal nucleation, growth, and long-term mechanical stability. Proper formation and lifelong maintenance of otoconia require a precisely regulated endolymphatic calcium concentration, making vestibular calcium homeostasis a critical determinant of otoconial integrity.

Maintenance of this highly specialized microenvironment depends on an epithelial calcium transport system composed of apical calcium channels (TRPV5 and TRPV6), intracellular calcium-binding proteins (calbindin-D9K and calbindin-D28K), plasma membrane calcium ATPases, and sodium-calcium exchangers. These transporters collectively regulate calcium trafficking between vestibular epithelial cells and the endolymph, thereby preserving the ionic conditions required for continuous otoconial remodeling.

Vitamin D plays a pivotal role in this process through activation of the vitamin D receptor (VDR), which stimulates transcription of several calcium transport proteins, particularly TRPV5. Experimental studies have demonstrated that vitamin D deficiency markedly reduces TRPV5 expression within the vestibular epithelium, leading to impaired calcium transport, disruption of endolymphatic calcium homeostasis, and defective otoconial biomineralization. Consequently, insufficient vitamin D may compromise both the formation and maintenance of otoconia.

In addition to regulating calcium transport, vitamin D appears to influence the molecular architecture of otoconia through indirect regulation of OC90 expression. Experimental models lacking adequate OC90 develop enlarged, poorly mineralized, mechanically unstable otoconia characterized by abnormal crystal organization. Ultrastructural analyses of ageing human temporal bones have consistently demonstrated progressive degeneration of otoconia, including central fissures, surface erosion, fragmentation, and dissolution of calcium carbonate crystals. These morphological abnormalities significantly reduce mechanical stability and increase the likelihood of spontaneous otoconial detachment from the utricular macula.

Ageing further amplifies these pathological processes through multiple complementary mechanisms. Progressive deterioration of the gelatinous otoconial membrane weakens the adhesive forces responsible for anchoring otoconia to the sensory epithelium, while a gradual decline in otoconial number and regenerative capacity reduces the ability of the vestibular system to maintain structural integrity. Histological studies have shown that these degenerative changes become particularly pronounced after the fifth decade of life, paralleling the marked increase in BPPV incidence observed in epidemiological studies.

Hormonal factors further contribute to this process. Estrogen deficiency following menopause accelerates systemic bone resorption, alters calcium metabolism, and may directly impair vestibular biomineralization. Experimental ovariectomy models consistently demonstrate enlarged, hypomineralized otoconia with reduced density and abnormal morphology, providing a biological explanation for the higher prevalence and recurrence of BPPV among postmenopausal women.

More recently, vestibular dark cells have emerged as key regulators of otoconial homeostasis. Besides maintaining endolymph composition through active ion transport, these metabolically active epithelial cells appear to participate in the physiological degradation of detached otoconial debris. Disturbances in endolymphatic calcium concentration may reduce their resorptive capacity, allowing free otoconial fragments to persist within the vestibular labyrinth and repeatedly migrate into the semicircular canals, thereby contributing to disease recurrence despite successful repositioning maneuvers.

Collectively, current evidence supports a multifactorial pathogenic model in which vitamin D deficiency acts as a biological modifier rather than an isolated etiological factor. By disrupting calcium transport, impairing otoconial biomineralization, weakening the structural cohesion of the otolithic membrane, and reducing physiological clearance of detached otoconia, hypovitaminosis D may substantially increase susceptibility to both the development and recurrence of BPPV. This integrated model provides a strong biological rationale for considering correction of vitamin D deficiency as a potential adjunctive strategy for reducing disease recurrence, particularly in high-risk populations such as older adults, postmenopausal women, and patients with osteoporosis.

The growing body of evidence linking vitamin D deficiency to BPPV has substantially expanded our understanding of this

common vestibular disorder. Rather than representing an isolated mechanical event, BPPV should increasingly be viewed as the consequence of progressive degeneration of the otolithic system, resulting from the interaction of ageing, impaired calcium metabolism, hormonal changes, and alterations in vestibular biomineralization. Within this multifactorial framework, vitamin D deficiency appears to function as a biological modifier that increases susceptibility to otoconial instability and disease recurrence rather than serving as a single causative factor.

One of the major strengths of the current evidence is the remarkable consistency between experimental observations and clinical findings. Animal models have demonstrated that disruption of vitamin D signaling leads to abnormal otoconial morphology, impaired biomineralization, and structural fragility. Likewise, histopathological analyses of ageing human temporal bones have consistently revealed fragmentation, surface erosion, and progressive degeneration of otoconia. These structural alterations closely mirror the clinical observation that recurrent BPPV predominantly affects elderly individuals, postmenopausal women, and patients with osteoporosis. The convergence of molecular, histological, and epidemiological evidence therefore provides strong biological plausibility for the proposed association.

Nevertheless, biological plausibility alone does not establish causality. Despite the growing number of observational studies reporting lower serum vitamin D concentrations among patients with recurrent BPPV, several investigations have failed to confirm this association after adjustment for confounding variables. Such discrepancies likely reflect differences in study populations, laboratory assays, vitamin D threshold definitions, seasonal variation, geographic latitude, and baseline skeletal health. Moreover, serum 25(OH)D concentration represents only one component of calcium metabolism and may not accurately reflect local vitamin D activity within the vestibular labyrinth. Future studies incorporating additional biomarkers—including parathyroid hormone, bone turnover markers, fibroblast growth factor-23, and vitamin D receptor polymorphisms—may provide a more comprehensive understanding of vestibular calcium regulation.

Another important consideration is that vitamin D deficiency rarely occurs in isolation. Patients with recurrent BPPV frequently present with osteoporosis, sarcopenia, reduced physical activity, frailty, diabetes mellitus, chronic kidney disease, or estrogen deficiency, all of which may independently influence otoconial integrity. Consequently, vitamin D deficiency may represent one component of a broader metabolic phenotype characterized by impaired calcium homeostasis rather than acting as an independent pathogenic determinant. This concept is supported by epidemiological studies demonstrating a strong overlap between osteoporotic disease and recurrent BPPV, suggesting that systemic skeletal health and vestibular biomineralization are closely interconnected.

Current therapeutic evidence also deserves careful interpretation. Randomized controlled trials evaluating vitamin D supplementation have generally demonstrated a reduction in recurrence rates among vitamin D-deficient patients; however, treatment protocols remain heterogeneous regarding dosage, duration, calcium co-supplementation, and target serum concentrations. Furthermore, most available trials evaluated recurrence as the primary endpoint without assessing structural recovery of otoconia or long-term vestibular function. Consequently, although supplementation appears to be a safe and inexpensive strategy in deficient individuals, the optimal therapeutic regimen and the serum vitamin D threshold required for vestibular protection remain to be established.

The emerging concept of BPPV as a metabolic vestibular disorder has important implications for future research. Advances in molecular biology have identified several promising pathways involved in otoconial homeostasis, including OC90, otolin-1, TRPV5/TRPV6 channels, vitamin D receptor signaling, and vestibular dark-cell function. These molecular targets may ultimately provide novel biomarkers for identifying individuals at increased risk of recurrence and could pave the way for targeted preventive therapies aimed at preserving otoconial integrity rather than merely treating canalith displacement after symptom onset.

Future investigations should prioritize large multicenter prospective studies with standardized definitions of vitamin D deficiency, uniform supplementation protocols, and extended follow-up periods. Integration of biochemical markers, high-resolution vestibular imaging, genetic susceptibility analyses, and functional vestibular assessment may further clarify the complex relationship between systemic calcium metabolism and vestibular disease. Such an approach would help distinguish simple epidemiological association from true biological causation and facilitate the development of evidence-based preventive strategies.

Future Perspectives

The expanding understanding of otoconial biology is progressively transforming the conceptual framework of BPPV. Rather than being regarded exclusively as a mechanical disorder caused by displaced otoconia, BPPV is increasingly recognized as a multifactorial disease involving age-related degeneration, altered calcium metabolism, hormonal imbalance, and impaired vestibular biomineralization. This paradigm shift opens new opportunities for both translational research and preventive therapeutic strategies.

One of the most promising research directions concerns the identification of reliable biomarkers reflecting otoconial metabolism. Several molecules, including otolin-1, otoconin-90 (OC90), osteopontin, and other extracellular matrix proteins, have recently emerged as potential indicators of otoconial degeneration. Combined with serum 25-hydroxyvitamin D [25(OH)D] measurements and bone turnover markers, these biomarkers may enable early identification of individuals at increased risk of recurrent BPPV before the onset of clinical manifestations.

Advances in molecular biology have also highlighted the importance of calcium transport pathways within the vestibular labyrinth. The vitamin D receptor (VDR), transient receptor potential vanilloid channels (TRPV5 and TRPV6), calcium ATPases, and intracellular calcium-binding proteins constitute interconnected regulatory networks that maintain endolymphatic calcium homeostasis. A better understanding of these molecular mechanisms may facilitate the development of targeted therapies aimed at preserving otoconial integrity rather than simply repositioning displaced particles.

Another promising field concerns the genetic susceptibility to BPPV. Although the disease has traditionally been considered idiopathic, accumulating evidence suggests that genetic polymorphisms affecting calcium metabolism, extracellular matrix organization, vitamin D signaling, and bone remodeling may influence both disease susceptibility and recurrence. Large-scale

genomic studies are therefore warranted to identify individuals genetically predisposed to otoconial degeneration and to support the development of personalized preventive strategies.

Emerging imaging technologies may further improve our understanding of vestibular pathology. Ultra-high-resolution magnetic resonance imaging, three-dimensional micro-computed tomography, and advanced histopathological techniques could provide unprecedented insights into otoconial ultrastructure and vestibular degeneration. Integration of structural imaging with molecular biomarkers may ultimately allow in vivo assessment of otoconial integrity, overcoming one of the major limitations of current clinical research.

From a therapeutic perspective, future management of BPPV should evolve from a purely mechanical approach toward a comprehensive metabolic strategy. Canalith repositioning maneuvers remain the cornerstone of acute treatment; however, identification and correction of modifiable metabolic risk factors—including vitamin D deficiency, osteoporosis, reduced bone mineral density, and endocrine disorders—may substantially reduce recurrence rates. Such an integrated approach is particularly relevant for elderly individuals, postmenopausal women, and patients with recurrent disease, in whom metabolic abnormalities frequently coexist.

Future randomized controlled trials should therefore move beyond evaluating recurrence alone and incorporate mechanistic outcomes, including changes in biochemical markers, vestibular function, quality of life, and long-term otoconial stability. Standardization of vitamin D deficiency thresholds, supplementation protocols, and follow-up duration will be essential to improve comparability across studies and strengthen the certainty of current evidence.

Clinical Implications

Current evidence supports a practical and clinically relevant approach to patients presenting with recurrent BPPV. While routine vitamin D screening cannot yet be recommended for every patient with a first episode, assessment of serum 25(OH)D should be considered in individuals at increased risk of recurrence, particularly older adults, postmenopausal women, patients with osteoporosis or osteopenia, individuals with previous fragility fractures, and those experiencing multiple recurrent episodes despite appropriate canalith repositioning maneuvers.

In patients with confirmed hypovitaminosis D, correction of vitamin D deficiency according to current osteoporosis and endocrine guidelines represents a rational adjunctive intervention. Although supplementation should not replace repositioning maneuvers, restoration of physiological calcium homeostasis may improve long-term vestibular stability by limiting progressive otoconial degeneration and reducing the likelihood of recurrent canalithiasis.

The management of recurrent BPPV should therefore extend beyond symptom resolution and incorporate assessment of systemic metabolic health. Such an approach promotes a transition from reactive treatment toward individualized prevention, consistent with the principles of precision medicine.

Conclusion

Vitamin D deficiency has emerged as one of the most extensively investigated metabolic factors associated with BPPV. Experimental studies demonstrate that disruption of vitamin D-dependent calcium regulation impairs otoconial biomineralization and structural integrity, whereas clinical investigations consistently report an increased prevalence of hypovitaminosis D among patients with recurrent disease. Together, these findings support a biologically plausible relationship between systemic calcium homeostasis and vestibular function.

Nevertheless, the currently available evidence remains insufficient to establish vitamin D deficiency as an independent etiological factor. Instead, hypovitaminosis D should be regarded as a clinically relevant and potentially modifiable risk factor acting in concert with ageing, osteoporosis, estrogen deficiency, and other disturbances of calcium metabolism. This multifactorial model provides a coherent explanation for the high recurrence rate observed in susceptible populations and supports a broader metabolic interpretation of BPPV pathophysiology.

From a clinical standpoint, targeted assessment and correction of vitamin D deficiency represent simple, safe, and cost-effective interventions that may complement conventional canalith repositioning maneuvers in appropriately selected patients. Although additional high-quality multicenter randomized trials are required to establish optimal screening strategies and supplementation protocols, the available evidence already supports integrating metabolic evaluation into the long-term management of recurrent BPPV.

Future advances in molecular biology, biomarker discovery, vestibular imaging, and precision medicine are expected to redefine the management of BPPV. Moving beyond symptomatic treatment toward preservation of otoconial homeostasis may ultimately reduce disease recurrence and improve long-term vestibular health.

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