Auditory and Vestibular Research

Effects of Combined Vestibular Rehabilitation and 25-Hydroxyvitamin D Supplementation on Vestibular Function in Patients with Vestibular Neuritis: A Randomized Controlled Trial

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Highlights

- VRT plus vitamin D showed superior subjective improvement in DHI scores.
- Combined therapy significantly enhanced VOR gains across all affected canals.
- Adding Vitamin D boosted both subjective symptoms and objective VOR recovery.

Abstract

Background and Aim: Inflammatory processes play a key role in vestibular neuritis (VN) pathogenesis, and recent research has associated vitamin D deficiency with various audio-vestibular conditions, including reduced serum 25-hydroxyvitamin D [25(OH)D] concentrations in individuals with acute VN. Given its notable immunomodulatory and anti-inflammatory actions, vitamin D may serve as an adjunct to enhance VRT outcomes. The present study examined the impact of combining 25(OH)D supplementation with VRT on vestibular function among patients with VN.

Methods: In this randomized controlled trial, 41 patients with VN were allocated into two groups. The VRT group (n=20) received individualized vestibular rehabilitation for 30 minutes, three times daily for 12 weeks. The VRT+VitD group (n=21) received the same VRT protocol plus a weekly oral dose of 50,000 IU of 25(OH)D for 12 weeks. Outcomes were assessed pre- and post-intervention using the Dizziness Handicap Inventory (DHI), Vertigo Symptom Scale-short form (VSS-sf), and affected canal vestibulo-ocular reflex gains with video head impulse test, within- and between-groups.

Results: Both groups improved significantly across all measurements. The VRT+VitD group showed a normal serum level of vitamin D post-intervention (39.36±3.06; $p<0.001$) and greater improvements in subjective symptom reduction (DHI; $p=0.023$; partial $\eta^2=0.126$), and VOR gains ($p<0.001$ with partial $\eta^2=0.459$, 0.576, and 0.197 for lateral, anterior, and posterior canals, respectively) compared to the VRT group.

Conclusion: Correcting vitamin D deficiency supports and potentially enhances the effects of vestibular rehabilitation in patients with vestibular neuritis, although these outcomes should be interpreted with caution due to the study's limitations.
Keywords: Vestibular neuritis; vestibular rehabilitation; 25-hydroxyvitamin D; vestibulo-ocular reflex; inflammation.

Introduction

Vestibular neuritis (VN), an acute vestibular syndrome, represents the third most frequent peripheral vestibular disorder following benign paroxysmal positional vertigo (BPPV) and Meniere's disease (MD) [1]. Reported incidence rates of VN vary between 3.5 [2] to 162 [3] cases per 100,000 population. The condition appears more common in women [3] and typically affects individuals aged 30 to 60 years [4], with peak age distributions reported between 40 and 50 years [2].

Clinically, VN presents with sudden-onset, prolonged true rotational vertigo exceeding 24 hours, accompanied by postural instability, nausea, and vomiting, but without auditory or other neurological symptoms [1, 5]. Static symptoms often improve over weeks in most cases, but functional recovery may extend from months to as long as 10 years [1, 6, 7], highlighting the importance of vestibular rehabilitation for addressing persistent dynamic deficits beyond symptomatic treatments (such as vestibular suppressants) or targeted pharmacotherapy (including corticosteroids, antivirals, and vasodilators) [8-10]. Evidence from high-quality trials confirms that vestibular rehabilitation is both safe and efficacious for managing VN [10], with no reported adverse events related to VRT [11]. In contrast, certain adverse effects have been documented with corticosteroid therapy [12, 13].

Although the precise cause of VN is not fully understood, proposed mechanisms primarily involve viral infection, vascular ischemia, and immune-mediated processes [8]. A growing body of knowledge supports the idea that inflammation plays an important role in the induction and progression of VN. For instance, blood-based biomarkers (e.g., inner ear-specific protein (otolin-1)) and inflammatory markers (e.g., C-reactive protein, fibrinogen, D-dimers, leukocyte/neutrophil counts, and the neutrophil-to-lymphocyte ratio) are significantly elevated in patients with peripheral vestibular syndromes, including benign paroxysmal positional vertigo, vestibular neuritis, and Ménière's disease, compared to healthy subjects [14, 15]. Several studies have consistently shown significantly lower serum 25-hydroxyvitamin D (25(OH)D) levels in patients with acute VN compared to healthy individuals, suggesting a potential link between vitamin D deficiency and the onset or severity of VN [15-17]. Accordingly, Wu et al. conducted a prospective case-control study to evaluate serum 25(OH)D levels in patients with VN. They found that serum levels of 25(OH)D were significantly lower in patients with VN in the acute phase than in control patients [16]. Xiong et al. conducted a case-control study to investigate the expression level of serum 25(OH)D and its correlation in patients with VN, and concluded that lower serum levels of 25(OH)D are associated with VN [17].

Similar associations exist with other vestibular pathologies, including BPPV [18, 19] and MD [20]. Positive therapeutic responses to vitamin D supplementation in BPPV [21, 22] and MD [23] indicate potential benefits for additional vestibular conditions. As a fat-soluble vitamin, vitamin D maintains circulating calcium and phosphate homeostasis while exerting substantial immunomodulatory and anti-inflammatory effects [24, 25]. Given its anti-inflammatory effects and the presence of vitamin D receptors in the inner ear, vitamin D may serve as a promising therapeutic option for VN.

Combining a new therapeutic agent with VRT could therefore offer a more efficacious treatment approach for VN while reducing potential adverse effects. Such advancements are essential for improving clinical management and patient outcomes. This study is the first randomized controlled trial to investigate the effects of 25(OH)D supplementation combined with VRT on the improvement of subjective symptoms and vestibular function in patients with vestibular neuritis.

Methods

A prospective, parallel-group, randomized controlled trial was conducted.

Participants

Forty-one patients diagnosed with acute unilateral vestibular neuritis were enrolled from the Ear, Nose and Throat Clinic at Amir-Alam Teaching Hospital. Diagnosis required acute, prolonged vertigo, unidirectional spontaneous nystagmus, positive head impulse test, and caloric testing showing ≥25% unilateral weakness, without auditory or central neurological involvement. Inclusion criteria include age 30–60 years, serum 25(OH)D level <20 ng/mL

(indicating deficiency) measured by blood collection and using the ELISA method, and commitment to protocol adherence. Exclusion criteria included prior vestibular rehabilitation or corticosteroid treatment for the current episode; coexisting neurological, musculoskeletal, visual, or metabolic conditions impacting balance or vitamin D metabolism; previous vestibular disorders, tinnitus, or hearing loss; medications interfering with vestibular compensation or vitamin D metabolism; and pregnancy, lactation, or menopause.

Patients were randomly allocated to one of two 12-week interventions. The sequence for random assignment was created using Random Allocation Software. To ensure balanced group sizes during the enrollment process, block randomization with blocks of four participants was employed. Allocation concealment was maintained through the use of sequentially numbered, sealed envelopes, which were opened by the researcher only after confirming participant eligibility and obtaining informed consent.

The VRT group (n=20) underwent an individualized home-based vestibular rehabilitation program, supervised through weekly video consultations and reinforced by bi-monthly clinic visits. The program consisted of gaze stabilization exercises (VOR ×1 and ×2 adaptation), habituation exercises customized to provocative movements, and balance/gait training, performed for 30 minutes three times daily. The VRT+VitD group (n=21) followed the identical VRT program plus weekly oral 50,000 IU vitamin D (cholecalciferol) supplementation for 12 weeks. They were asked to take the pill during lunch. Compliance was tracked via weekly pill counts and self-reported logs. Participants were instructed to report any symptoms suggestive of vitamin D toxicity, including signs of hypercalcemia (such as nausea, dehydration, and constipation) and hypercalciuria (such as polyuria and kidney stones) [26].

Outcome Measures

Assessments occurred at baseline and immediately following the 12-week intervention. The primary outcome was change in the Persian version of the Dizziness Handicap Inventory (DHI) total score, a 25-item questionnaire evaluating dizziness-related handicap (range 0–100) [27]. Secondary outcomes encompassed the Persian version of the Vertigo Symptom Scale-short form (VSS-sf) for vertigo and autonomic symptom frequency/severity (range 0-60, a score ≥12 indicates severe dizziness) [28], and vestibulo-ocular reflex (VOR) gain via video Head Impulse Test (vHIT), with mean gains calculated for each semicircular canal.

The psychometric properties, including face, content, discriminant, and construct validity, internal consistency, and reliability, of both DHI [27] and VSS-sf [28] were determined in the Iranian populations.

A vHIT system (GN Otometrics, Denmark) was employed to assess VOR function in all semicircular canals. Each participant received around twenty rapid and unpredictable head impulses with an amplitude of 10–20° while maintaining gaze on a stationary visual target. Impulses applied in the yaw plane were used to evaluate the horizontal semicircular canals. To assess the left anterior–right posterior and right anterior–left posterior canal planes, the head was turned approximately 35–45° toward the corresponding side, and impulses were then delivered in the pitch plane. A reduced VOR gain was defined as values below 0.8 for the horizontal canals and below 0.7 for the vertical canals [29].

Statistical Analysis

Analyses were conducted using SPSS Statistics (Version 17.0, IBM Corp., USA). Normality was evaluated with the Shapiro-Wilk test. Intention-to-treat principles were applied. Baseline between-group differences were assessed via standardized differences (>0.2 indicating significant imbalance). Intervention effects were examined using two-way mixed-model ANOVA with Group (VRT vs. VRT+VitD) and Time (pre vs. post) as factors for DHI and VSS-sf scores. Analysis of Covariance was applied for VOR gains since there were significant differences between the groups in terms of baseline values. The baseline values were considered as the covariant factor. All assumptions required for conducting both ANOVA and ANCOVA, including normality of residuals, homogeneity of variances, linearity between covariates and outcomes, and homogeneity of regression slopes, were examined and met. Statistical significance was set at $p < 0.05$.

Results

Demographic characteristics of participants in both groups are presented in Table 1.

There were no significant differences between groups at baseline in terms of sex, age, serum 25(OH)D levels, and subjective scores (DHI, VSS-sf). However, the VRT+VitD group exhibited lower baseline VOR gains in

affected canals. The standardized differences for lateral, anterior, and posterior canals are 0.3, 0.5, 0.7, respectively, which all are >0.2.

In the VRT+VitD group, serum 25(OH)D levels increased significantly from 17.37 ± 3.45 to 39.36 ± 3.06 ng/mL ($p<0.001$). In contrast, the VRT group showed no significant change, with levels rising from 17.23 ± 4.01 to 18.81 ± 3.92 ng/mL ($p>0.05$). This resulted in a significant between-group difference ($p<0.001$).

The ANOVA showed that both groups demonstrated improved subjective scores ($p<0.001$; Table 2), yet the VRT+VitD group achieved greater DHI reduction ($F_{(1,38)} = 5.607$; $p = 0.023$, partial $\eta^2=0.126$) but no significant group-by-time interaction emerged for VSS-sf ($F_{(1,38)}=1.202$, $p>0.05$).

There were significant VOR gain improvements for affected lateral, anterior, and posterior semicircular canals in both groups ($p<0.001$; Table3). Nevertheless, after adjusting for baseline values, ANCOVA showed a significant effect of group on post intervention VOR gains for affected lateral, anterior, and posterior semicircular canals in favor of VRT+VitD group (lateral: $F_{(1,38)}=32.289$, $p < 0.001$, partial $\eta^2=0.459$; anterior: $F_{(1,38)}=51.712$, $p < 0.001$, partial $\eta^2=0.576$; posterior: $F_{(1,38)}=9.302$, $p = 0.004$, partial $\eta^2=0.197$).

Discussion

This randomized controlled trial evaluated the additional benefit of high-dose 25(OH)D supplementation alongside standardized vestibular rehabilitation in patients with acute vestibular neuritis and established vitamin D deficiency. Key results demonstrate that combined treatment outperformed VRT alone in normalizing serum vitamin D, alleviating subjective handicap (as measured by DHI scores), and restoring vestibulo-ocular reflex (VOR) function (as measured by vHIT). Effective vitamin D repletion in the supplemented group verifies protocol adherence and provides mechanistic support for the clinical gains observed.

The more pronounced DHI score improvement in the VRT+VitD group indicates that vitamin D correction adds significant improvement beyond VRT's established benefits [10, 11]. This holds clinical relevance, given that 30–50% of VN patients experience ongoing dizziness and imbalance that impair quality of life [6, 7].

From an objective standpoint, enhanced VOR gain recovery across all semicircular canals in the combination group suggests favorable influences on peripheral vestibular structures and central adaptation mechanisms. Although VRT facilitates neuroplasticity and compensation, the findings imply that vitamin D amplifies these processes. Vitamin D is a secosteroid hormone that is primarily produced in the skin and subsequently activated through hydroxylation in the liver and kidneys to form 1,25-dihydroxyvitamin D3, the biologically active form [25]. This active metabolite binds to the nuclear vitamin D receptor (VDR), forming a complex that functions as a transcription factor and regulates genes involved in cell proliferation, differentiation, and immune regulation [24]. One of the most important actions of vitamin D is its inhibitory effect on the adaptive immune system and inflammatory responses. It reduces the expression and release of pro-inflammatory cytokines such as TNF-α, IL-1β, IL-6, and IL-8, while enhancing the production of anti-inflammatory cytokines including IL-4, IL-5, and IL-10 [30-32]. These immunomodulatory effects are mainly mediated through the nuclear VDR, which is widely expressed in immune cells such as regulatory T cells, dendritic cells, macrophages, neutrophils, and B lymphocytes. Beyond the immune system, VDR has also been identified in inner ear structures, including the epithelium of the crista ampullaris, the membranous semicircular canals, and adjacent osteocytes [33, 34]. The presence of VDR in these vestibular structures suggests that vitamin D may play a role in maintaining vestibular and auditory function. Experimental evidence supports this assumption, as animals lacking the vitamin D receptor exhibit balance impairments in various motor and vestibular tests [35]. Moreover, it has been proposed that sufficient levels of vitamin D may suppress local autoimmune or inflammatory reactions triggered by prior viral infections, like its anti-inflammatory effects observed in other chronic inflammatory conditions [36]. Taken together, these findings indicate that vitamin D may influence vestibular function through both immunomodulatory and receptor-mediated mechanisms. Thus, deficiency correction probably reduced the neuroinflammatory burden typical of acute VN, fostering an environment conducive to neural recovery and strengthening VRT-induced compensation. This aligns with inner ear vitamin D receptor expression [33, 34] and mirrors the logic behind corticosteroid use [37].

These results align with and build upon evidence connecting vitamin D status to vestibular disorders. Deficiency is an established contributor to BPPV onset and recurrence [18, 19]. Case-control research has documented lower 25(OH) D in VN patients versus controls [16, 17]. The current trial provides the first randomized evidence that targeted repletion yields significantly better clinical outcomes. Inflammatory patterns in VN share features with

other neuroinflammatory states modulated by vitamin D [30, 33, 34, 38], suggesting supplementation could target shared pathways across select vestibular conditions.

Several limitations should be considered when interpreting these results. Participants were not blinded to group allocation, which could introduce performance or expectation bias. Future studies could minimize this risk by using a blinded assessor or implementing a placebo intervention for the control group. Although participants were aware of their group assignment, the secondary outcome measures, vHIT-derived vestibulo-ocular reflex (VOR) gain, are objective, instrument-based assessments that are less susceptible to subjective influence or reporting bias, thereby reducing the potential impact of this limitation. The study population was limited to patients with vitamin D deficiency, and therefore, the findings may not be generalizable to patients with normal baseline vitamin D levels. Because vitamin D status can influence neuromuscular function and vestibular adaptation, patients with normal baseline vitamin D levels might respond differently to vestibular rehabilitation combined with supplementation. Therefore, caution should be exercised when extrapolating these results to populations without vitamin D deficiency. Additionally, the follow-up duration was restricted to 12 weeks, precluding conclusions regarding long-term outcomes or the prevention of chronic vestibular symptoms. Despite appropriate randomization procedures, a chance imbalance in baseline VOR gain between groups was observed, which should be taken into account when interpreting group comparisons.

Future research could further explore the long-term effects of combining vitamin D supplementation with vestibular rehabilitation, as well as investigate the impact of different vitamin D doses in patients with varying degrees of deficiency. Such studies would help clarify optimal treatment strategies and determine whether the benefits observed in this study are sustained over time or vary according to baseline vitamin D status.

Conclusion

This randomized controlled trial supports the hypothesis that high-dose 25-hydroxyvitamin D supplementation improves vestibular rehabilitation outcomes in patients with vestibular neuritis and concurrent vitamin D deficiency. The addition of vitamin D supplementation led to marked improvements in rehabilitation outcomes. The combined intervention resulted in significantly better outcomes in biochemical restoration (serum vitamin D levels), symptom relief (as measured by DHI scores), and VOR recovery compared to rehabilitation alone. These benefits are likely attributed to the inflammatory nature of vestibular neuritis and the well-established anti-inflammatory and immunomodulatory roles of vitamin D. Vitamin D supplementation may reduce neuro inflammation, promote neural healing, and enhance central adaptation through vestibular rehabilitation therapy. These findings could inform clinical practice by suggesting that vitamin D supplementation, when combined with vestibular rehabilitation, may provide an effective treatment strategy for patients with vestibular neuritis and vitamin D deficiency. Future studies with larger sample sizes and longer follow-up durations are needed to confirm these findings and explore the long-term benefits of vitamin D supplementation in vestibular neuritis treatment.

Ethical Considerations

The study protocol received approval from the Ethical Committee of Tehran University of Medical Sciences (IR.TUMS.NFM.REC.1403.040), and all participants provided written informed consent before enrollment. The trial was registered with the Iranian Registry of Clinical Trials (IRCT20240512061714N1).

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Authors' contributions

MAK: Data acquisition, drafting the manuscript; MAG: Conceptualization, design of the study, study supervision, critical revision of the manuscript; ET: Conceptualization, design of the study, study supervision, critical revision of the manuscript; KM: Technical and material support; NY: Selection and evaluation of patients for recruitment; AF: Technical and material support; MAS: Data acquisition.

Conflict of interest

The authors have no conflict of interest, financial or otherwise, to declare.

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Table 1. Demographic and clinical characteristics of participants in the study groups (n=41)

Characteristic	VRT + VitD (n = 21)	VRT (n = 20)
Age (years), mean ± SD	41.81 ± 10.97	48.15 ± 10.83
Sex, Female/Male, n	11 / 10	11 / 9
Symptom Duration (months), median [IQR]	5 [5–6]	6 [5–6]
Affected Side, n (%)		
Left	11 (52.4)	11 (55.0)
Right	10 (47.6)	9 (45.0)
Serum 25-hydroxyvitamin D (ng/mL), mean ± SD	17.37 ± 3.45	17.23 ± 4.01

VRT, vestibular rehabilitation therapy; VitD, Vitamin D; SD, standard deviation; IQR, interquartile range.

Table 2. Mean (standard deviation) of subjective symptom scores in the study groups before and after intervention.

	VRT+VitD (n = 21)			VRT (n = 20)		
Questionnaire	Before, mean ± SD	After, mean ± SD	Mean Diff (95% CI) p-value	Before, mean ± SD	After, mean ± SD	Mean Diff (95% CI) p-value
DHI	67.33 ± 10.22	7.90 ± 1.84	-59.43 (-64.00, -54.85) <0.001	65.00 ± 8.03	12.10 ± 2.38	-52.90 (-56.32, -49.71) <0.001
VSS-sf	22.33 ± 4.04	3.90 ± 1.51	-18.43 (-20.61, -16.23) <0.001	21.80 ± 6.93	5.30 ± 1.41	-16.50 (-19.48, -13.51) <0.001

VRT, vestibular rehabilitation therapy; VitD, Vitamin D; CI, confidence interval; DHI, Dizziness Handicap Inventory; VSS-sf, Vertigo Symptom Scale-short form.

Table 3. Mean (standard deviation) vestibulo-ocular reflex gain on the affected side in the study groups before and after intervention

	VRT + Vitamin D (n = 21)			VRT (n = 20)		
Semicircular Canal	Before, mean ± SD	After, mean ± SD	Mean Difference (95% CI) p-value	Before, mean ± SD	After, mean ± SD	Mean Difference (95% CI) p-value
Lateral	0.41 ± 0.10	0.91 ± 0.06	0.50 (0.45, 0.55) <0.001	0.44 ± 0.10	0.80 ± 0.06	0.36 (0.29, 0.41) <0.001
Anterior	0.55 ± 0.10	0.83 ± 0.03	0.28 (0.23, 0.30) <0.001	0.60 ± 0.10	0.75 ± 0.04	0.15 (0.11, 0.18) <0.001
Posterior	0.77 ± 0.02	0.80 ± 0.03	0.03 (0.02, 0.04) <0.001	0.73 ± 0.02	0.74 ± 0.02	0.01 (0.006, 0.01) <0.001

VRT, vestibular rehabilitation therapy; VitD, Vitamin D; CI, confidence interval; VOR, vestibulo-ocular reflex.