Original Article
Iranian Journal of Otorhinolaryngology, Vol.38(2), Serial No.145, Mar-2026

Effect of Vitamin D Deficiency on Incidence and Relapse of Benign Paroxysmal Positional Vertigo

Manwinder Singh Walia[1], Nitika Tuli[1], Arshaque Hussain[1], Sandeep Bansal[1], *Ruchika Bhagat[1]

Abstract

Introduction:

Benign Paroxysmal Positional Vertigo (BPPV) is a peripheral labyrinth disease and is a prevalent cause of dizziness with a lifetime frequency of roughly 10%. Otoconia found in the otolith organ contain calcium carbonate and protein. Derangement in calcium homeostasis due to vitamin D is implicated in both the onset and recurrence of BPPV, and supplementation could play a role in preventing or alleviating the condition.

Materials and Methods:

This prospective study enrolled a total of 60 patients with posterior semicircular canal BPPV. Subjects were divided into Group A with vitamin D<20ng/ml (45) and Group B with vitamin D>20ng/ml (15). Group A on day 1 received Canal Repositioning Manoeuvre (CRM) and vitamin D supplementation. Group B on day 1 received CRM only. Patients were followed up at 1,2,3, and 6 months and reassessed for vitamin D levels and relapse of BPPV.

Results:

Out of 60 subjects, 14 showed relapse, 23.3%. Out of 45 subjects in Group A, 13 showed relapse (28.9%). Among 15 patients in Group B, only 1 patient showed relapse (6.7%). Vitamin D levels in the supplementation group normalized before 3 months of follow-up. After 3 months, no relapse was noted in either of the groups. Hence, vitamin D-deficient BPPV patients showed a significantly a higher rate of relapse (p value 0.039).

Conclusion:

This study demonstrates a high incidence of vitamin D deficiency among patients with BPPV. Also, patients with low vitamin D levels have higher rate of relapse of BPPV, and correction of vitamin D levels leads to a decrease in relapse frequency.

***Keywords*:** Benign Paroxysmal Positional Vertigo (BPPV), Vitamin D deficiency, Canalith repositioning manoeuvre (CRM), Nystagmus, Epley's manoeuvre, Dix-Hallpike test.

Received date: 30 Aug 2025
Accepted date: 06 Jan 2026

*__Please cite this article;__ *Walia MS, Tuli N, Hussain A, Bansal S, Bhagat R. Effect of Vitamin D Deficiency on Incidence and Relapse of Benign Paroxysmal Positional Vertigo. Iran J Otorhinolaryngol. 2026:38(2): 99-108.*
Doi: 10.22038/ijorl.2026.90851.4032

[1]*Deportment of ENT, Government multi-Speciality Hospital-16, Chandigarh, India.*
__Corresponding author:__
E-mail: Ruchikabhagat62@gmail.com

Introduction

Benign Paroxysmal Positional Vertigo (BPPV) is a peripheral labyrinth disease and is a prevalent cause of dizziness among the general population, with a lifetime frequency of roughly 10% (1,2). BPPV is characterized by vertigo or sudden-onset of dizziness brought on by certain shifts in the patient's head posture, nausea, and/or nystagmus, which is typically positional (3), Vertigo that comes on suddenly and passes quickly is one of the hallmarks of BPPV. BPPV can occur at any age; however, it is more common in middle-aged or older people. BPPV can also afflict children (4), BPPV affects females at a rate two times higher than that of males (5). The posterior semi-circular canal is damaged by BPPV in the vast majority of cases (90-91 percent); however, the horizontal and anterior semi-circular canals can also be affected (6). The presence of positional vertigo, which occurs for short intervals along with nystagmus occurring on positional testing after one month of execution of a successful Canalith repositioning manoeuvre (CRM), has been classified as BPPV recurrence (7), Recent research has shown that BPPV is associated with various comorbid conditions such as raised blood pressure or blood sugars, hyper or hypothyroidism, increased blood lipid levels, and osteoporosis (8).

BPPV occurs when loosened otoconia detach from the utricular macula and float in the semicircular canals, which causes these to become sensitive to gravity (5). These otoconia are divided into separate zones: the central and peripheral zones. The central is usually organic and has a lower calcium level, while the peripheral is mostly inorganic and has a greater calcium level (9). Several investigations have revealed a link between BPPV and impaired calcium metabolism in the inner ear endolymph. Osteoporosis, defined by low bone mass and accelerated turnover of bone, was discovered more commonly in women who had recurrent BPPV. These women were mostly middle-aged or older. A shortage in serum vitamin D may be associated with recurrent BPPV, and treatment with vitamin D may be able to reduce the chance of recurrence in some patients (9,10). Because otoconia found in otolith organ contains calcium carbonate and protein, any derangement in calcium homeostasis may lead to structural and/or functional changes in otoconia (11). So, vitamin D can be linked to the conservation of the function of the otolith organ. In a study, mutant Vitamin D receptor mice were found to have many vestibular dysfunctions (12). In osteopenic/osteoporotic female adult rats, the size of otoconia increases while density decreases (13). The width and density of the globular material increase in animals with vitamin D deficiency, while vitamin D treatment mitigates these alterations (14).

Vitamin D deficiency is implicated in both the onset and recurrence of BPPV, and supplementation could play a role in preventing or alleviating the condition (15,16). The Epley manoeuvre is widely used to treat posterior canal BPPV (17,18). However, BPPV is known to recur, and identifying and addressing risk factors is crucial for preventing relapse. This study aims to explore the role of vitamin D in the pathophysiology and recurrence of BPPV. We aim to assess serum concentrations of vitamin D in cases of BPPV, to determine if low levels of vitamin D can be regarded as a cause of BPPV relapse, and to determine whether supplementation of vitamin D reduces the risk of BPPV relapse.

Materials and Methods

The prospective hospital-based study was conducted in the Department of Otorhinolaryngology at the Government Multispecialty Hospital, Sector 16, Chandigarh, over a period of two years. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from the patient.

Study subjects

Patients with signs and symptoms of BPPV who were willing to take part in this study and fulfilled the inclusion criteria were recruited. Patients with only posterior canal BPPV were recruited. The study is prospective observational (two exposure groups: Vit-D < 20 vs ≥ 20; No untreated control for ethical reasons. We enrolled consecutive eligible patients presenting with BPPV during the study period. This method reduces the risk of the researcher selectively assigning patients to a particular group based on their suspected outcome. Patients were allocated to the "deficient" (group A) or "sufficient"(group B) group solely based on a pre-defined, objective

laboratory cutoff value for serum vitamin D. This allocation was based on a blood test result, not on clinical judgment, which minimizes the potential for subjective patient assignment. The unequal group sizes are a direct reflection of the prevalence of vitamin D deficiency within our study's recruitment population and timeframe. A prevalence of 2.4% was used to arrive at a sample size of 60.

Inclusion criteria:

Patients from all age groups were included. Posterior semicircular Benign paroxysmal positional vertigo (BPPV), was diagnosed based on the patient's history (repeated bouts of positional vertigo triggered by turning over or when lying down in the supine position, and each attack lasts for less than one minute). Dix Hallpike test, Spontaneous nystagmus, Gaze induced nystagmus, Post head shaking nystagmus, ocular motility tests, and was confirmed with VNG.

Exclusion criteria:

1. Patients are taking a dose of vitamin D.
2. Patients taking any medication that affects vitamin D metabolism, such as antiepileptic drugs like Carbamazepine, Phenobarbital, Phenytoin, Fosphenytoin, Primidone, and Rifampin.
3. Disorders that impair vitamin D metabolism, such as obesity, diabetes mellitus, hypertension, cancers, and multiple sclerosis.
4. Patients with conditions such as Meniere's disease, vestibular neuritis, or sudden sensorineural hearing loss-which can often present with vertigo-were excluded based on abnormal audiological findings.

Methodology

Complete history taking was done. It covered personal and medical histories, as well as specifics of the patient's symptoms, such as the duration of each vertigo attack, the factors that provoked it, and the causes that precipitated it. In addition to it, accompanying symptoms such as nausea and vomiting were examined. Basic audiological measurement, which consisted of pure tone audiometry using a two-channel audiometer, the MAICO MA-42, was done. Videonystagmography (VNG) test battery, including spontaneous nystagmus, gaze-induced nystagmus, post-head-shaking nystagmus, ocular motility tests, and Dix-Hallpike test, were used to diagnose and review the patients of BPPV. CRM were performed, which included Epley's manoeuvre for posterior semi-circular canal BPPV.

All CRM procedures were performed exclusively by a single, highly experienced clinician: the principal Investigator, Head of the Department of ENT. By centralizing the procedure with one expert operator, the inter-operator differences were effectively eliminated. This approach was implemented specifically to ensure a high degree of interobserver agreement and to minimize diagnostic variability throughout the study. Furthermore, all cases were reviewed in a weekly team meeting to achieve a consensus on diagnosis and relapse classification, reinforcing the consistency of our clinical assessments.

These procedures were performed after BPPV was confirmed and the affected side canal, as well as the underlying pathology (canalolithiaisis or cupulolithiasis), was identified. Serum 25-hydroxyvitamin D3 (25-OH D3) concentration was evaluated using the ELISA technique on a serum sample. The normal vitamin D reference range for an adult is 20-40 ng/ml. BPPV patients were then divided into two groups based on vitamin D levels. Group A with low vitamin D levels (<20ng/ml) and Group B with normal levels of vitamin D (>20ng/ml). Cases with low Vitamin D levels were supplemented with vitamin D. Both groups were followed up to compare levels of serum vitamin D and relapse of BPPV. Vitamin D supplement was given in the form of cholecalciferol granule sachets (60,000 IU/gm) weekly for six weeks or till their serum vitamin D level reached 20 ng/ml.

Study procedure and follow-up

Patients were assigned to one of two groups: group A, who had vitamin D levels that were less than 20 ng/ml and were given more vitamin D in addition to having CRM performed on them; and group B, whose vitamin D levels were greater than 20 ng/ml, and only CRM was performed on them.

The patients were asked to come back between 48 and 72 hours to see the resolution of symptoms. The disappearance of symptoms along with the absence of nystagmus in the Dix-Hallpike test was seen as evidence of a full recovery. The patients were further followed up at 1month, 2 months, 3 months, and 6 months.

The total duration of the follow-up period was six months.

During this time, clinical evaluation was performed again in the form of repeat history taking, positional testing, and blood vitamin D level testing for the group that had low levels of vitamin D before vitamin D supplementation. The levels of vitamin D were monitored until they were found to be higher than 20ng/ml. Vertigo returning after a complete recovery and being able to detect nystagmus with Frenzel glasses were both regarded as indicators of a relapse of BPPV.

A BPPV relapse was defined as a recurrence of characteristic positional vertigo, objectively confirmed by the presence of diagnostic positional nystagmus (via Dix-Hallpike or Supine Roll test), occurring ≥ 30 days after a successfully treated initial episode (confirmed by resolution of symptoms and a negative positional test) (19).

Over the course of a period of six months, the number of BPPV attacks that occurred in each group was counted, examined, and documented. While longer follow-ups can provide additional insights into long-term patterns, our chosen period is unequivocally valid for assessing the primary effect of Vitamin D deficiency on early BPPV relapse.

Statistical analysis

Following the loading of the data into the Microsoft Excel spreadsheet, the statistical analysis was performed using the SPSS version 25.0 program. Information about quantitative factors, such as numerical data, was provided as the mean and standard deviation, whereas information regarding qualitative variables, such as categorical categories, was presented as the frequency and percentage of each category. The Student t-test was used to compare the mean values of the two groups, while the chi-square test was used to assess the frequency differences between the two groups.

The analysis consists of a frequency table, a bar chart, a pie chart, an association of variables based on a Chi-square test, and Yates corrections for a 2x2 contingency table or technique pooling, as well as a Fisher's exact test, if any cell frequency is less than five (for higher order tables than a 2x2 table). Measures of central location (mean and median) and measures of dispersion were utilized in order to arrive at an estimate for each and every quantitative variable (standard deviation). The Kolmogorov–Smirnov tests of normality were used to investigate the consistency of the data. When looking at data having a normal distribution, the mean was analyzed in relation to parametric tests. In the case of data that was not distributed normally, a comparison was made between the Median and the non-parametric. If the p-value was lower than 0.05, then it was considered to have statistical relevance.

Results

A total of 60 patients were enrolled and analysed in the study. The majority of the subjects belonged to the 41-45 years (26.7%) age group. Mean age was 43.62±12.90 years. (Figure. 1)

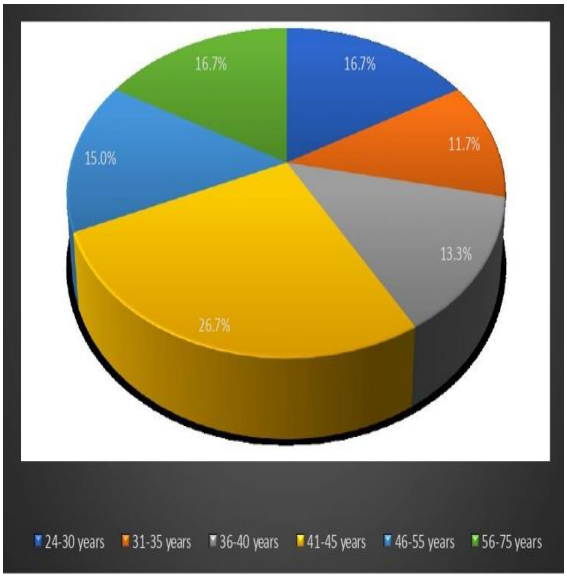

Fig 1. Age distribution of study subject

The difference between the mean Vitamin D level on 1st day, 1st month, and 2nd month was significantly greater among the group with supplementation (with vitamin D less than 20ng/ml) as compared to the group without supplementation (with vitamin D more than 20 ng/ml). Thereafter, there was no significant difference in mean Vitamin D level at the 3rd month and 6th month between subjects with no supplementation and subjects with supplementation (Table. 1).

Hence, the vitamin D levels in the supplementation group nearly normalized before 3 months of follow-up.

Table 1. Mean Vitamin D level among subjects with and without Supplementation

Time interval	Vitamin D Supplementation	Mean	Std. Deviation	95% Confidence Interval	Mean Difference	t-test value	p-value
1st day	No	25.39	2.50	24.01 – 26.77	15.20	19.929	.001*
	Yes	10.19	2.58	9.41 -10.97			
1 month	No	25.39	2.50	24.01 – 26.77	10.39	13.005	.001*
	Yes	15.00	2.73	14.18 – 15.82			
2 months	No	25.39	2.50	24.01 – 26.77	6.12	6.997	.001*
	Yes	19.27	3.06	18.35 – 20.19			
3 months	No	25.39	2.50	24.01 – 26.77	3.25	1.611	0.201
	Yes	22.14	2.32	21.44 – 22.84			
6 months	No	25.39	2.50	24.01 – 26.77	2.41	1.309	0.289
	Yes	22.98	1.63	22.49 – 23.47			

Out of 60 BPPV subjects, 14 showed relapse (23.3%). A total of 45 BPPV patients (Group A) had day 1 vitamin D levels below 20ng/ml. In Group A (vitamin D < 20ng/ml), 13 patients showed relapse, with a relapse frequency of 28.9% (95% CI: 15.64% - 42.14%). In Group B (vitamin D > 20ng/ml), only 1 patient showed relapse with relapse frequency being 6.7% (95% CI: 0.17% - 31.96%). Hence, in Group A (vitamin D-deficient), BPPV patients showed a significantly higher rate of relapse than in Group B (p value 0.039) (Table 2)

Table 2. Day 1 Vitamin D level among subjects with and without relapse

Vitamin D level	Relapse	
	No	Yes
< 20 ng/ml (Group A)	32	13
	71.1%	28.9%
≥ 20 ng/ml (Group B)	14	1
	93.3%	6.7%
p-value = 0.039*		

Of the 13 patients with day 1 vitamin D levels below 20 ng/ml (Group A) who showed relapse, 11 patients showed relapse in <2 months, and 2 patients showed relapse in <3 months (95% CI: 54.97% - 98.05%). In Group B, only one patient showed relapse, which was within 2 months. After 3 months, no relapse was noted in either of the groups (95% CI: 0.17% - 31.96%; p value = 0.039) (Table 3).

Table 3. Describing the study groups as per the Time of relapse and the Vitamin D level at the time of relapse

Time of Relapse	Level of Vitamin D (ng/ml)	No. of patients
<2 months	<20	11
<3 months	<20	2
<2 months	>20	1

It can be concluded from the table below that a higher relapse rate (64.3%, 95% CI: 35.1 - 87.2%) was seen in patients among Group A with the lowest level of vitamin D on day 1(<10 ng/ml). Amongst patients in Group B (>20 ng/ml on day 1), the relapse rate was quite low (7.1%, 95% CI: 0.2-33.9%; p value = 0.0028) (Table 4).

Table 4. Describing the study groups as per levels of vitamin D on day 1 and the percentage of relapse.

Vitamin D level (Day 1)	Number	No. of patients
<10	9	64.3%
10-20	4	28.6%
>20	1	7.1%

Discussion

Vertigo is the sensation that there is movement when there isn't any movement present. This sensation might be described as either swaying, tilting, spinning, or an unstable feeling. Vertigo has traditionally been classified into two types: peripheral and central. Although some symptoms of peripheral and central causes of vertigo overlap, peripheral vertigo is caused mostly by issues with the inner ear's vestibular system or the nerve. More than half of all cases of peripheral vertigo are brought on by a condition known as BPPV (20,21).

It has been shown that the risk of developing BPPV is significantly increased between the ages of 41 and 60, particularly in females (22). In our study, the maximum number of subjects belonged to the 41-45 years (26.7%) age group with a mean age of 43.62±12.90 years. This pattern is coherent with the findings by Resuli et al. (mean age of BPPV patients was 43.70±15.44 years (range: 17-87 years) and Thomas et al. (mean age being 44.39 years) (23,24). Our study demonstrated a female: male ratio of 2:1 among the BPPV patients enrolled. This is similar to findings by Resuli et al. and Thomas et al. (23,24). Changes in hormone levels with age contribute to the development of osteoporosis and osteopenia in females.

In ovariectomized osteopenic/osteoporotic female adult rats, one experimental investigation discovered ultrastructural variations of the otoconia. These modifications included changes in the otoconia's aspect, size, and density (13). This process might be one factor that contributes to the increased occurrence of BPPV in females. However, BPPV may also occur in people of different ages and can occur in males; whether or not osteoporosis has a role in the incidence of BPPV in any of these groups is yet unsolved (3). In this investigation, the mean Vitamin D level on the 1st day, 1st month, and 2nd month was significantly higher among males (19.70±6.79, 21.91±5.13, and 23.80±3.88, respectively) compared to females (11.13±5.37, 15.44±3.82, and 19.30±3.05, respectively). Thereafter, it showed a non-significant difference in the 3rd and 6th months, as all patients with vitamin D deficiency were supplemented with vitamin D. This finding of a higher incidence of vitamin D deficiency among females compared to males is in line with findings by previous studies (23,24).

Our study demonstrated a high vitamin D deficiency rate (75%) in BPPV patients. This is much higher than reported by Ceylan and Kanmaz (33%), Yamanaka et al (14%), and Bigelow et al (26.2%). (25-27).

This may be due to the higher prevalence of deficiency in India, where studies indicate that 70-100% of the population is deficient. Contributing factors include limited sun exposure and a lack of vitamin D fortification in food (28). According to Harinarayan et al, only 31% of the Indian population has normal Vitamin D levels, with urban populations showing lower levels than rural ones, possibly due to dietary differences and lower intake of Vitamin D-rich foods (29).

Karatas A et al. suggested that the rates of osteoporosis and vitamin D deficiency found in BPPV patients were reasonably high, but there was no significant difference in mean vitamin D levels of BPPV patients and controls (30). One theory that explains the relation of vitamin D and BPPV states that there is a complex system of calcium-binding proteins and an epithelial calcium transport mechanism present in the membranous labyrinth. Vitamin D deficiency causes downregulation of calcium transport, and thus it leads to defects in otoconia production and an increase in otolith detachment (31).

In the present investigation, the overall relapse rate was 23.3%. According to the findings of Vibert et al, the rate of relapse of BPPV was 27 percent. The majority of patients had a relapse within the first six months (32).

In our study, all relapses occurred before 3 months of follow-up. Abdelmaksoud et al. stated that the recurrence rates of BPPV are reported to be between 30 and 50 percent.

These rates are often connected with female sex, elderly age, ear problems, chronic diseases, and a lack of vitamin D (33). Rhim et al. reported that BPPV patients exhibit recurrence rates of roughly 20 percent after one year and nearly 50 percent after five years (34). In our study, relapse was significantly more common among subjects with Vitamin D level < 20 ng/ml (28.9%) compared to subjects with Vitamin D level ≥ 20 ng/ml (6.7%) on the day of relapse (p value 0.039). These findings strengthen the relationship that has been found in the literature, suggesting low levels of vitamin D predisposing to BPPV.

According to Talaat et al, it was hypothesized that low levels of vitamin D were not only connected to the development of BPPV but that low levels of vitamin D also had a role in the recurrence of the condition. The return of the disease was attributed in part to having insufficient amounts of vitamin D (35).

Dessouky et al examined blood levels of 25-hydroxyvitamin D3 and calcium total & ionized in BPPV cases and compared the difference in serum levels of vitamin D3 in patients who had recurrence versus patients who did not have recurrence. The recurrence group had lower levels of vitamin D, total calcium, and ionized calcium in their serum than the non-recurrence group did (36).

The researchers Jeong et al conducted a meta-analysis to evaluate the therapeutic effects of vitamin D administration, both with and without calcium, with the purpose of avoiding BPPV recurrences. The researchers came to the conclusion that taking vitamin D supplements might be beneficial for the secondary prevention of BPPV (37).

Pecci et al found that there is a relationship between benign paroxysmal positional vertigo and hypo-vitaminosis D, as well as the fact that the occurrence of benign paroxysmal positional vertigo relapses decreases following vitamin D administration (38) Ioanna Saganaki and Paris Binos conducted a literature study on the numerous variables that increase the likelihood of BPPV recurrence. They came to the conclusion that postmenopausal women who have recurring episodes of BPPV should also undergo treatment for osteoporosis and a vitamin D deficit if they are to receive treatment (39).

In our study, all the relapses occurred within the first 3 months of follow-up, and the mean vitamin D levels of the Vitamin D-deficient group normalized and became comparable to the non-deficient group before 3 months of follow up. Hence, we can indirectly infer that with the treatment of vitamin D deficiency, there was a reduction in relapses.

Conclusion On Significant Comparisons After Month 3:

The most important finding is that no significant difference in relapse rates exists between the groups after Month 3 because relapses completely ceased. The significant comparisons

are confined to the initial period:

- The relapse rate was significantly higher in Group A than in Group B during the first 3 months.
- The mean Vitamin D level was significantly different between the groups at Day 1, Month 1, and Month 2.

After Month 3, the significant differences disappear for both the biochemical (vitamin D levels) and clinical (relapse incidence) outcomes. This convergence-where normalized vitamin D levels are associated with a complete cessation of relapses-strongly supports the inference that correcting the vitamin D deficiency was responsible for reducing the relapse risk.Due to ethical reasons and as suggested by the ethical committee, we could not deny treatment in the form of vitamin D to patients with vitamin D levels less than 20 ng/ml. Thus, this study lacks a control group to which cases who were supplemented with vitamin D could be compared. Abdelmaksoud et al investigated the connection between BPPV recurring episodes and a lack of vitamin D. Forty patients participated in the case-control research. In the group that took vitamin D supplements, the incidence of recurrent BPPV was much lower (33). We acknowledge that the convenience sampling method, necessitated by BPPV's acute presentation, may limit generalizability to the broader population. However, consecutive recruitment from our tertiary clinic ensures that the cohort is representative of patients actively seeking specialized care for this condition.

The unequal group sizes (45 vs. 15) directly reflect the high prevalence of Vitamin D deficiency in our clinical population, an epidemiologically expected finding. While the smaller group reduces power for subgroup

analyses, our primary statistical tests are robust to this imbalance. The significant association observed suggests a strong effect unlikely to be negated by the sample size disparity.

Thus, while these factors may constrain broad generalizability, they enhance the internal validity and clinical relevance of our findings for the typical BPPV patient demographic in a specialized care setting. This has been explicitly addressed in the manuscript's limitations.

Conclusion

Our study highlights a notable association between vitamin D deficiency and benign paroxysmal positional vertigo (BPPV), revealing both a high prevalence of deficiency among affected individuals and a link between low vitamin D levels and increased recurrence. Encouragingly, correction of these deficiencies appears to reduce relapse frequency.

While larger, controlled studies are needed to confirm these findings and establish an association, the known broader benefits of vitamin D combined with its potential role in managing BPPV support the routine measurement and supplementation of 25(OH)D levels in these patients. Given the extensive impact of BPPV, even modest improvements in recurrence rates through supplementation could provide symptom relief for a substantial number of individuals.

The novelty of our study is not just in confirming an association, but in providing high-quality, prospective evidence from a rigorously controlled study that positions vitamin D deficiency as a central, treatable contributor to the BPPV disease process and its recurrence. Furthermore, we provide novel data on the pronounced effect size of this association despite significant confounding comorbidities, suggesting that vitamin D deficiency is a potent and independent risk factor worthy of targeted intervention.

This paves the way for a change in clinical practice towards a more preventive and holistic management approach, rather than a purely reactive one.

Limitations

1. The absence of an untreated control group (it was a deliberate choice in our study, based on ethical considerations for patient care).

2. We acknowledge the presence of uncontrolled confounders, including age, sex, comorbidities (osteoporosis, thyroid, and renal disease), seasonal variation in vitamin D levels, and baseline vertigo severity. While these factors could influence the results in our study, we believed prioritizing a clear and feasible primary objective: to track the association between baseline vitamin D levels and BPPV outcomes. Systematically controlling the extensive list of potential confounders would have required a
significantly larger sample size and more complex resources, which were beyond the scope of this initial investigation.

3. Finally, the unequal sizes between the vitamin D-deficient and sufficient groups, which in our study are a direct reflection of the high prevalence of deficiency within the recruitment population during the study period.

References

1. Maslovara. S. Butkovic Soldo, S. Sestak, A., Milinkovic, K. Rogic-Namacinski. J. & Soldo, A. (2018) chapters 25 (OH) D3 levels, incidence and recurrence of different clinical forms of benign paroxysmal positional vertigo. Brazilian Journal of Otorhinolaryngology, 84, 453-459. DOI: 10.1016/j. bjort 2017.05.007, PubMed: 28655524.

2. Mohsin, F.D., Alharbawi, A.F. & Alraho, T.S. (2019) Benign paroxysmal positional vertigo and vitamin D deficiency. Journal of Pharmaceutical Innovation, 8, 49-52.

3. Yu, S., Liu, F., Cheng, Z. & Wang, Q. (2014) Association between osteoporosis and benign paroxysmal positional vertigo. A systematic review BMC Neurology. 14, 110. DOI: 10.1186/1471-2377-14-110, PubMed: 24886504.

4. Jang, Y.S. & Kang. M.K. (2009) Relationship between bone mineral density and clinical features in women with idiopathic benign paroxysmal positional vertigo. Otology and Neurotology, 30, 95-100. DOI: 10.1097/MAO.0b013e31818f5777,
PubMed: 19008769.

5. Parham, K., Leonard, G., Feinn, R.S., Lafreniere, D. & Kenny, A.M. (2013) Prospective clinical investigation of the relationship between idiopathic benign paroxysmal positional vertigo and bone turnover: A pilot study. Laryngoscope, 123, 2834-2839. DOI: 10.1002/lary 24162, PubMed: 23674177.

6. Kansu, L., Avci, S., Yilmaz, 1. & Ozluoglu, L.N. (2010) Long-term follow-up of patients with posterior canal benign paroxysmal positional vertigo. Acta Oto-Laryngologica, 130, 1009-1012. DOI: 10.3109/00016481003629333, PubMed: 20297928.

7. Kim, S.Y., Han, S.H., Kim, Y.H. & Park, M.H. (2017) Clinical features of recurrence and osteoporotic changes in benign paroxysmal positional vertigo. Auris, Nasus, Larynx, 44, 156-161. DOI: 10.1016/j.anl.2016.06.006, PubMed: 27423924.

8. Sreenivas, V., Sima, N.H. & Philip, S. (2021) The role of comorbidities in benign paroxysmal positional vertigo, Ear, Nose, and Throat Journal, 100. NP225-NP230. DOI: 10. 1177/0145561319878546, PubMed: 31565984.

9. Büki, B., Ecker, M., Jünger, H. & Lundberg, Y.W. (2013) Vitamin D deficiency and benign paroxysmal positioning vertigo. Medical Hypotheses. 80.201-204. DOI: 10.1016/j.mehy.2012.11.029, PubMed: 23245911.

10. Al-Mohaimeed, A., Khan, N., Naeem, Z. & Al-Mogbel, E. (2012) Vitamin D status among women in middle east. Journal of Health Sciences, 2, 49-56.

11. Büki, B., Jünger, H., Zhang, Y. & Lundberg, Y.W. (2019) The price of immune responses and the role of vitamin D in the inner ear. Otology and Neurotology, 40, 701-709. DOI: 10.1097/ MA0. 0000000000002258, PubMed: 31194714.

12. Minasyan, A., Keisala, T., Zou, J., Zhang, Y., Toppila, E., Syvälä, H., Lou, Y.R., Kalueff, A.V., Pyykkö, I. & Tuohimaa, P. (2009) Vestibular dysfunction.in vitamin D receptor mutant mice. Journal of Steroid Biochemistry and Molecular Biology, 114, 161-166. DOI: 10.1016/j. jsbmb. 2009.01.020, PubMed: 19429446.

13. Vibert, D., Sans, A., Kompis, M., Travo, C., Muhlbauer, R.C., Tschudi, I., Boukhaddaoui, H. & Häuster. R. (2008) Ultrastructural changes in otoconia of osteoporotic rats. Audiology and Neuro-Otology, 13, 293-301. DOI 10.1159/000124277, PubMed: 18391565.

14. Song, P., Zhao, X., Xu, Y., Zhao, Z., Liu, Y. & Gao, Q. (2021) Morphological effect of vitamin D deficiency on globular substances in mice. Otology and Neurotology, 42, e1313-e1317. DOI: 10.1097/ MA0.0000000000003229, PubMed: 34121084.

15. Sanyelbhaa, H. & Sanyelbhaa, A. (2015) Vestibular-evoked myogenic potentials and subjective visual vertical testing in patients with vitamin D deficiency/insufficiency. European Archives of Oto-Rhino-Laryngology, 272, 3233-3239, DOI: 10.1007/ s00405-014-3395-6, PubMed: 25411075.

16. Sheikhzadeh, M. Lotfi, Y., Mousavi, A., Heidari. B. & Bakhshi. E. (2016) The effect of serum vitamin D normalization in preventing recurrences of benign paroxysmal positional vertigo: A case-control study. Caspian Journal of Internal Medicine, 7, 173-177. PubMed: 27757201.

17. Helminski, J.O. Zee, D.S., Janssen, I. & Hain, T.C. (2010) Effectiveness of particle repositioning maneuvers in the treatment of benign paroxysmal positional vertigo: A systematic review. Physical Therapy, 90, 663-678. DOI: 10.2522/ptj.20090071, PubMed: 20338918.

18. Prim-Espada, M.P. De Diego-Sastre, J.I. & Pérez-Fernández, E. (2010) [Meta-analysis on the efficacy of Epley's manoeuvre in benign paroxysmal positional vertigo]. Neurologia, 25, 295-299. DOI: 10.1016/j.nri.2010.01.004, PubMed: 20643039.

19. Sfakianaki I, Binos P, Karkos P, Dimas GG, Psillas G. (2021) Risk Factors for Recurrence of Benign Paroxysmal Positional Vertigo. A Clinical Review. Journal of Clinical Medicine, 10(19):4372. DOI: 10.3390/jcm10194372. PMID: 34640391; PMCID: PMC8509726.

20. Muncie, H.L., Sirmans, S.M. & James, E. (2017) Dizziness: Approach to evaluation and management. American Family Physician, 95, 154-162 PubMed: 28145669.

21. Grill, E., Strupp, M., Müller, M. & Jahn, K. (2014) Health services utilization of patients with vertigo in primary care: A retrospective cohort study. Journal of Neurology, 261, 1492-1498. DOI: 10.1007/s00415-014-7367-y. PubMed: 24817192.

22. Caldas, M.A., Ganança, C.F., Ganança, F.F., Ganança, M.M. & Caovilla, H.H. (2009) Clinical features of benign paroxysmal positional vertigo. Brazilian Journal of Otorhinolaryngology, 75, 502-506. DOI: 10.1016/S1808-8694(15)30487-0, PubMed: 19784417.

23. Seyed Resuli, A., Bedir, A. & Özgür. A. (2022) The relationship between benign paroxysmal positional vertigo and vitamin D. Cureus. 14, 26068 DOI: 10.7759/cureus 26068, PubMed: 35747111.

24. Thomas, R.J., Goutham, M.K., Bhat. V.S., Kamath, S.D., Aroor, R. & Bhandary, S.K. (2022) Association of serum calcium and vitamin D with benign paroxysmal positional vertigo. International Archives of Otorhinolaryngology, 26, e365-e369. DOI: 10.1055/5-0041-1724093, PubMed: 35846804.

25. Ceylan, S.M. & Kanmaz. M.A. (2020) Vitamin D influence on the clinical course of benign paroxysmal positional vertigo: A retrospective cohort study. An International Journal of Otorhinolaryngology Clinics, 12, 43-47. DOI: 10.5005/jp-journals-10003-1359.

26. Bigelow, R.T. & Carey, J.P. (2020) Randomized controlled trial in support of vitamin D and calcium supplementation for BPPV. Neurology, 95, 371-372. DOI: 10.1212/WNL.0000000000010349, PubMed: 32759203.

27. Yamanaka, T., Shirota, S., Sawai, Y., Murai. T., Fujita, N. & Hosoi, H. (2013) Osteoporosis as a risk factor for the recurrence of benign paroxysmal positional vertigo. Laryngoscope, 123, 2813-2816Bigelow. DOI: 10.1002/ary 24099, PubMed: 23568754 RT.

28. GR & Gupta, A. (2014) Vitamin D deficiency in India: Prevalence, causalities and interventions. Nutrients, 6, 729-775. DOI: 10.3390/nu6020729. PubMed: 24566435.

29. Harinarayan, C.V., Ramalakshmi, T., Prasad, U.V., Sudhakar, D., Srinivasarao, P.V.L.N., Sarma. K.V.S. & Kumar, E.G.T. (2007) High prevalence of low dietary calcium, high phytate consumption, and vitamin D deficiency in healthy south Indians. American Journal of Clinical Nutrition, 85, 1062-67. DOI: 10.1093/ ajcn/ 85.4.1062, PubMed: 17413106.

30. Karatas. A. Acar Yuceant. G. Yüce. T. Hacı. C., Cebi, I.T. & Salviz. M. (2017) Association of benign paroxysmal positional vertigo with osteoporosis and vitamin D deficiency: A case-controlled study. Journal of International Advanced Otology. 13, 259-265. DOI: 10.5152/lao 2016.2640, PubMed: 28274898.

31. Libonati, G.A., Leone, A., Martellucci, S., Gallo, A., Albera, R., Lucisano, S., Bavazzano, M., Chiarella, G., Viola, P., Galletti, F. Freni, F. Ciodaro, F. Marcelli. V. Tortoriello, G., di Santillo, L.S., Picciotti, P.M., Galli, J., Vitale, S., Quaranta. N., Cavallaro, G., Gamba, P., Teggi, R., Cangiano, I. Faralli, M., Barboni, A., Messina, A. & Graziano, G. (2022) Prevention of recurrent benign paroxysmal positional vertigo: The role of combined supplementation with vitamin D and antioxidants. Audiology Research, 12, 445-456. DOI: 10.3390/audiolres12040045. PubMed: 36004953.

32. Vibert, D., Kompis, M. & Häusler, R. (2003) Benign paroxysmal positional vertigo in older women may be related to osteoporosis and osteopenia Annals of Otology, Rhinology, and Laryngology, 112, 885-889, DOI: 10.1177/000348940311201010, PubMed: 14587980.

33. Abdelmaksoud, A.A, Fahim, D.F.M., Bazeed, S.E.S., Alemam, M.F. & Aref, Z.F. (2021) Relation between vitamin D deficiency and benign paroxysmal positional vertigo. Scientific Reports, 11, 16855. DOI: 10.1038 /s41598 -021 -96445-x, PubMed: 34413436.

34. Rhim. Gl. (2019) Serum vitamin D and long-term outcomes of benign paroxysmal positional vertigo. Clinical and Experimental Otorhinolaryngology. 12. 273-278. DOI: 10.21053/ceo.2018.00381, PubMed: 30813712.

35. Talaat, H.S., Abuhadied, G., Talaat, A.S. & Abdelaal, M.S.S. (2015) Low bone mineral density and vitamin D deficiency in patients with benign positional paroxysmal vertigo. European Archives of Oto-Rhino-Laryngology, 272, 2249-2253. DOI: 10.1007/s00405-014-3175-3, PubMed: 24973969.

36. El Dessouky. T. Basiouny, E., Awad, A. & Abd Elkareem, R. (2022) Serum vitamin D and Recurrent Benign Paroxysmal Positional Vertigo. Egyptian Journal of Medical Research, 3.7-17. DOI: 10.21608/ejmr.2022.216112.

37. Jeong. S.H. Lee. SU & Kim, IS. (2022) Prevention of recurrent benign paroxysmal positional vertigo with vitamin D supplementation: A meta-analysis. Journal of Neurology, 269, 619-626. DOI: 10.1007/s00415-020-09952-8, PubMed: 32767116.

38. Pecci, R., Mandala, M., Marcari, A., Bertolai, R., Vannucchi, P., Santimone. R. Bentivegna, L., Di Giustino, F., Mengucci, A., Vanni, S. Pollastrı, F. & Giannoni, B. (2022) Vitamin D insufficiency/ deficiency in patients with recurrent benign paroxysmal positional vertigo. Journal of International Advanced Otology, 18. 158-166. DOI: 10.5152/iao.2022.21269. PubMed: 35418365.

39. Sfakianaki, I. Binos. P. Karkos, P., Dimas, G.G. & Psillas, G. (2021) Risk factors for recurrence of benign paroxysmal positional vertigo. A clinical review. Journal of Clinical Medicine, 10, 4372. DOI: 10.3390/jcm10194372, PubMed: 34640391.