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Vitamin D Deficiency and Idiopathic Benign Paroxysmal Positional Vertigo: A Cross-sectional Study

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ABSTRACT

Background: Benign Paroxysmal Positional Vertigo (BPPV) is recognized as the most prevalent cause of peripheral vertigo. Vitamin D3 is crucial for bone mineralization and maintaining calcium homeostasis. The purpose of this study was to look into the relationship between vitamin D insufficiency and BPPV.

Methods: This case-control study involved 105 participants, 50 confirmed BPPV patients and 55 controls. All participants underwent ear, throat, and nose examinations. Vitamin D3 levels were measured in all subjects. The quality of life was assessed using DHI and the Visual Vertigo Analog Scale (VVAS).

Results: In the BPPV group (n=50), 34% had normal Vitamin D levels, 38% had Vitamin D deficiency, and 28% had Vitamin D insufficiency. Among healthy controls (n=55), 21.82% had normal Vitamin D levels, 56.36% had a deficiency, and 21.82% had insufficiency. Mean Vitamin D levels were 23.78±10.43 ng/ml in BPPV cases and 35.99±15.99 ng/ml in controls (p=0.001). The mean BMI was 25.12±3.02 kg/m² for BPPV and 24.15±2.89 kg/m² for controls (p<0.05). Higher VAS and DHI scores correlated with Vitamin D deficiency and insufficiency in BPPV cases.

Conclusion: This study provides robust evidence indicating a substantial association between decreased levels of Vitamin D and the occurrence of idiopathic BPPV. The findings suggest that individuals with lower concentrations of Vitamin D are at a higher risk of developing idiopathic BPPV, underscoring the potential role of Vitamin D in the pathophysiology of this vestibular disorder.

Key-words: Benign Paroxysmal Positional Vertigo, Dizziness Handicap Inventory, Vitamin D, Semicircular Canal, Visual Vertigo Analog Scale

INTRODUCTION

Benign Paroxysmal Positional Vertigo (BPPV) is one of the most frequently diagnosed conditions in vertigo clinics, accounting for 20% to 40% of patients. The annual incidence and lifetime prevalence of BPPV are 0.6% and 2.4%, respectively [1-3]. Vertigo is characterized by a false sensation of rotation, either of oneself or the surroundings, indicating the involvement of the vestibular system, specifically the semicircular canals. This can occur from the labyrinth to the vestibular cortex.

Approximately 45–54% of patients presenting to the hospital with dizziness experience vertigo [4,5]. Vertigo can be classified as either peripheral or central. Acute spontaneous vertigo arises from lesions in the peripheral vestibular system. It is associated with a sense of rotation of oneself or the surroundings, loss of balance exacerbated by head movements, and the absence of neurological symptoms [6].

BPPV is most commonly observed in individuals in their sixth and seventh decades of life, with elderly individuals

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at higher risk. In most cases, the etiology of BPPV is idiopathic. Secondary causes include aging, head trauma, prolonged bed rest, hypertension, inner ear disorders, hyperlipidemia, migraine, and stroke. BPPV resulting from trauma and vestibular neuritis shows a similar distribution between males and females.

However, idiopathic BPPV is more prevalent in females [7,8]. BPPV manifests as brief episodes of vertigo with nystagmus, exacerbated by changes in head position. Dix and Hallpike have described various head and neck positions that induce nystagmus, aiding in the clinical diagnosis of BPPV [9].

Otoconia, which are typically located in the otolithic membrane of the utricle and saccule, can become dislodged and move into the duct or cupula of the semicircular canal due to head movements, causing vertigo and nystagmus. This condition most commonly affects the posterior semicircular canal. Otoconia are calcium carbonate crystals composed of a central organic core with lower calcium levels and an inorganic peripheral zone with higher calcium concentration.

These crystals, embedded in the otolithic membrane, lie above the kinocilium of hair cells and are bound by proteinaceous filaments. The formation of otoconia and the maintenance of low calcium concentration in the endolymph are active processes [10-12]. Calcium metabolism also influences the synthesis and absorption of Vitamin D. Thus, our study's goal is to assess the relationship between vitamin D and BPPV.

MATERIALS AND METHODS

The current study was conducted following informed written consent. The study comprised 105 subjects divided into two groups: 50 patients diagnosed with BPPV and 55 healthy controls. The case group underwent comprehensive clinical evaluations, including the Dix Hallpike test and serum Vitamin D3 assessments. The control group consisted of age-matched and sex-matched individuals without a history of dizziness, vertigo, or imbalance within the past year.

Inclusion criteria- The case group's eligibility requirements were anyone between the ages of 18 and 80 who had a positive Dix Hallpike test result and clinical symptoms suggestive of benign paroxysmal positional vertigo.

Exclusion criteria- Exclusion criteria included age outside the specified range (<18 or >80 years), pregnancy, and a history of head injury, ear infections, maxillary sinusitis, or chronic conditions such as renal, pulmonary, hematologic, gastrointestinal, or cardiovascular diseases. Additionally, participants using Vitamin D or calcium supplements or diagnosed with central vertigo were also excluded from the study.

Study Procedure- The BPPV patients diagnosed through clinical examination underwent Epley's maneuver post-diagnosis. VVAS and DHI were used to assess symptoms. The DHI comprises 25 questions categorizing physical, functional, and emotional impacts, scored as per patient responses ("yes," "sometimes," or "no"). Higher scores denote a bigger influence. The values range from 0 to 100. VVAS scores range from 0 to 10, reflecting symptom severity. Detailed histories, clinical examinations, and investigations were conducted as per the attached pro forma. 25-OH serum Using a commercially available ELISA kit, the levels of vitamin D3 were determined from aseptically obtained fasting blood samples obtained through venipuncture.

Statistical Analysis- Comparison of Vitamin D concentrations between the groups employed Epi Info™ for Windows version 7.0., analyzing data using mean, standard deviation (SD), and percentages. The p-values less than 0.05% were deemed statistically significant for differences.

RESULTS

The age distribution among BPPV cases (n=50) and healthy controls (n=55) is presented in Table 1. BPPV cases were categorized into four age groups: 21–34 years (12%), 35–50 years (22%), 51–64 years (54%), and >65 years (12%). In comparison, healthy controls were distributed as follows: 21–34 years (20%), 35–50 years (30.91%), 51–64 years (40%), and >65 years (10.91%). The majority of BPPV cases fell within the 51–64 years age group, comprising 54% of the sample, while healthy controls were most commonly aged 51–64 years, accounting for 40% of the control group.

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Table 1: Age distribution in BPPV cases and controls

Age group	BPPV cases (n=50)	Healthy Controls (n=55)
21–34 years, n (%)	6 (12)	11 (20.00)
35–50 years, n (%)	11 (22)	17 (30.91)
51–64 years, n (%)	27 (54)	22 (40.00)
>65 years, n (%)	6 (12)	6 (10.91)

The mean age of BPPV cases was 44.53 years (SD±13.75), similar to healthy controls at 45.60 years (SD±15.30), with no significant difference observed (p = 0.611). BPPV cases had a statistically higher mean BMI of 25.12 kg/m² (SD±3.02) compared to 24.15 kg/m² (SD±2.89) in healthy controls (p<0.05). Females comprised 60% of BPPV cases and 41.82% of healthy controls (p=0.451), while males constituted 40% of BPPV cases and 58.18% of healthy controls (p=0.542). Significant differences were observed in serum Vitamin D levels between BPPV cases and controls (p<0.01), with an increased incidence of deficiencies (<20 ng/ml) in BPPV cases compared to controls (Table 2).

Table 2: Baseline Clinical parameters in BPPV cases and controls

Parameters	BPPV cases (n=50)	Healthy Controls (n=55)	p-value
Age (years), mean±SD	44.53±13.75	45.60±15.30	0.611
BMI (kg/m²), mean±SD	25.12±3.02	24.15±2.89	<0.05
Females, n (%)	30 (60)	23 (41.82)	0.451
Males, n (%)	20 (40)	32 (58.18)	0.542
S. Vitamin D (ng/ml)		0	
Normal (30–100), n (%)	17 (34)	12 (21.82)	<0.01
Insufficiency (<30), n (%)	14 (28)	12 (21.82)	<0.01
Deficiency (<20), n (%)	19 (38)	31 (56.36)	<0.01

The distribution of semicircular canal involvement among BPPV cases (Table 3). The posterior canal was the most frequently involved (66%), followed by the horizontal canal (26%) and anterior canal (8%).

Table 3: Distribution of Semicircular canal involvement in BPPV cases

Canal involved	n	Percentage %
Anterior Canal	4	8
Horizontal Canal	13	26
Posterior Canal	33	66

The Pearson correlation coefficients between DHI score, VAS score, and serum Vitamin D levels. Strong positive correlations were found between DHI and VAS scores (r=0.876, p<0.01), indicating that higher DHI scores were associated with increased VAS scores (Table 4). Additionally, there was a significant negative correlation between DHI scores and serum Vitamin D levels (r=-0.716, p<0.01), suggesting that lower Vitamin D levels were associated with higher DHI scores. Similarly, VAS scores showed a strong negative correlation with serum Vitamin D levels (r=-0.816, p<0.01), indicating that lower Vitamin D levels correlated with higher perceived dizziness severity as measured by VAS (Table 4).

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Table 4: Pearson Correlation between various variables

Variables	DHI score	VAS	Vit D levels
DHI score			
r-value	1	0.876	-0.716
p-value	<0.01	<0.01	<0.01
VAS			
r-value	0.876	1	-0.816
p-value	<0.01	<0.01	<0.01
Vit D levels			
r-value	-0.716	-0.816	1
p-value	<0.01	<0.01	<0.01

DISCUSSION

This study involved 105 participants, comprising 50 cases and 55 controls, with no statistically notable variation observed in age and gender between these groups. Jeong *et al*. [13] conducted a study with 100 consecutive patients, including 63 females and 37 males, showing no statistically significant differences in age and sex distribution.

In our study, out of 50 patients, 33 had posterior canal BPPV, 13 had horizontal canal BPPV, and 4 had anterior canal involvement. Honrubia *et al.* [14] reported the prevalence of posterior, horizontal, and anterior canal BPPV as 93%, 5%, and 2%, respectively. Particle buildup in the posterior canal is caused by several factors, including its dependent position in both erect and supine postures, its location below the utricle when supine, and the size of the common crus of the posterior and superior semicircular canals. The likelihood of particles returning to the utricle increases as they enter the superior canal.

We found a significant difference in BMI between cases and controls. Jeong *et al*. [13] also found a significant difference (P=0.001) in BMI between groups A and B. The mean BMI in the patient group was 24.9±3.4, while in the control group, it was 23.3±3.6. Obesity decreases serum Vitamin D levels, leading to increased serum parathyroid hormone as a compensatory mechanism, which enhances calcium influx uptake by adipocytes, contributing to lipogenesis and promoting weight gain. Obesity also leads to hypovitaminosis D due to increased uptake of 25-Hydroxyvitamin D by adipose tissue [15-17].

In our study, the mean Vitamin D concentration was significantly lower in the patient group, consistent with findings from previous studies [18,19].

BPPV results from the displacement of otolithic debris from the otoconial bed [20]. Since calcium carbonate is the primary component of otolithic debris, normal concentrations of Ca^{2+} and carbonate (CO_3-) are necessary for crystal formation on the proteinaceous core [21]. Lower Ca^{2+} levels in the endolymph are crucial for regulating mineralization throughout the endolymph [22]. Because increased calcium resorption lowers endolymphic Ca^{2+} levels, it is less effective at dissolving dislodged otoconia. [23]. Studies have demonstrated that calcium concentration in the endolymph is controlled by various transport channels, including plasma membrane Ca2+ pumps, Na+/Ca^{2+} exchangers, and epithelial Ca^{2+} channels (calbindins) [22]. The expression of calcium-binding proteins such as calbindin-D9k and calbindin-D28k further maintains the function of these calcium channels. 1,25-dihydroxyvitamin D3 plays a role in upregulating the expression of these proteins and in maintaining calcium homeostasis [22]. Therefore, disturbances in calcium metabolism due to Vitamin D deficiency may contribute to the development of BPPV. Thus, Vitamin D deficiency may be considered a contributing factor in the onset of BPPV [23,24].

In our study, most patients exhibited moderate DHI and VVAS scores. In a study by De Abreu e Silva Grigol *et al*. [25], which included 91 patients, 72 (79.1%) were females and 19 (20.9%) were males, with a mean age ranging from 23 to 86 years and a mean age of 52.5 years. The mean VVAS score was 5.2, and the mean DHI score was 43.9, indicating moderate impairment. In another study by Garcia *et al*. [26], which focused on an age group of 18–60 years with peripheral vestibular disorders, a mean DHI score between 52 and 57 points was observed, also indicating moderate impairment. These studies align

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with our findings, where most patients with dizziness reported moderate scores on the DHI and VVAS scales. When examining the correlation between DHI and VAS scores with Vitamin D concentration in our study population, patients with high DHI and VVAS scores were found to have insufficient or deficient levels of Vitamin D. This similarity between the clinical severity of vertigo symptoms and low serum Vitamin D levels suggests a significant correlation between DHI scores and Vitamin D levels.

CONCLUSIONS

This study provides robust evidence indicating a substantial association between decreased Vitamin D levels and idiopathic BPPV occurrence. The findings suggest that individuals with lower concentrations of Vitamin D are at a higher risk of developing idiopathic BPPV, underscoring the potential role of Vitamin D in the pathophysiology of this vestibular disorder. This association highlights the need for further research to explore the underlying mechanisms and to evaluate the potential benefits of Vitamin D supplementation as a preventive or therapeutic strategy for idiopathic BPPV.

Future clinical trials are warranted to confirm these findings and to establish guidelines for Vitamin D supplementation in the prevention and treatment of idiopathic BPPV.

CONTRIBUTION OF AUTHORS

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