e-ISSN: 0976-822X, p-ISSN: 2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2023; 15(8); 78-81

Original Research Article

Association of Vitamin D Deficiency with Positional Vertigo: A Case Control Study

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Received: 12-05-2023 / Revised: 18-06-2023 / Accepted: 27-07-2023
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Conflict of interest: Nil

Abstract

Background and Objectives: Benign Paroxysmal Positional Vertigo (BPPV) stands as the most prevalent etiology of peripheral vertigo within the medical context. An essential component in bone mineralization and calcium homeostasis, Vitamin D3 bears crucial significance in physiological processes. Moreover, Vitamin D3 has been implicated in the pathogenesis of BPPV, thereby suggesting its potential as a viable therapeutic intervention. The primary objective of this study was to assess the potential association between BPPV and Vitamin D deficiency.

Materials and Methods: An observational case-control study involving a cohort of 60 subjects, comprising 30 confirmed cases diagnosed with BPPV) and 30 control individuals. All participants underwent comprehensive examinations of the ear, throat, and nose to ensure accurate identification and classification. The assessment of Vitamin D3 levels was conducted in all subjects.

Results: Among the BPPV cases, 35% exhibited normal levels of Vitamin D, while 37.5% presented with Vitamin D deficiency, and 27.5% demonstrated Vitamin D insufficiency. In the control group, 55% exhibited normal Vitamin D levels, whereas 22.5% each displayed Vitamin D insufficiency and deficiency. The mean concentration of Vitamin D in the BPPV case group was found to be 23.78 ± 10.43, whereas in the control group, it was 35.99 ± 15.99. This disparity between the two groups was statistically significant.

Conclusion: There was a significant association between reduced Vitamin D concentration and idiopathic BPPV. This highlights the potential relevance of Vitamin D as a contributing factor in the pathogenesis of BPPV and suggests its potential role as a therapeutic target for managing this condition.

Keywords: Benign Paroxysmal Positional Vertigo, Vitamin D, Cholecalciferol, Semicircular canal.

Introduction

Benign Paroxysmal Positional Vertigo (BPPV) constitutes a prevalent diagnosis in vertigo clinics, accounting for 20% to 40% of patient cases [1, 2]. The annual incidence and lifetime prevalence of BPPV are reported to be 0.6% and 2.4%, respectively. Vertigo, defined as a perception of rotational movement either of oneself or the surrounding environment, indicates the involvement of the vestibular system, specifically the semicircular canals. The origin of vertigo may vary from the labyrinth to the vestibular cortex [3, 4]. Notably, approximately 50% of patients presenting with dizziness at hospitals are diagnosed with vertigo [5]. Vertigo can be classified into peripheral or central types, with acute spontaneous vertigo typically associated with lesions in the peripheral vestibular system, manifesting as a sense of rotation of oneself or the surroundings, loss of balance, and exacerbation with head movements, but without neurological symptoms [6].

BPPV predominantly occurs in individuals aged in their sixth and seventh decade, with the elderly being at a higher risk [2]. While most cases of BPPV have an idiopathic etiology, secondary causes include aging, head injury, prolonged rest, hypertension, inner ear disorders, hyperlipidemia, migraine, and stroke. Notably, BPPV arising from trauma and vestibular neuritis shows similar distribution between males and females, whereas idiopathic BPPV is more frequently observed in females [7-9].

Clinically, BPPV is characterized by brief vertigo attacks accompanied by nystagmus, which worsen

with changes in head position. Dix and Hallpike's maneuver aids in the clinical diagnosis of BPPV by inducing specific head and neck positions to elicit nystagmus. The underlying mechanism involves the displacement of otoconia (calcium carbonate crystals) normally present in the otolithic membrane of the utricle and saccule into the duct or cupula of the semicircular canal due to head position changes in the plane of the canal. This displacement triggers vertigo and nystagmus, with the posterior semicircular canal being the most commonly involved [10-15]. Given the critical role of calcium metabolism in regulating Vitamin D synthesis and absorption, the present study aimed to explore the association between Vitamin D and BPPV at a tertiary care hospital in India.

Material & Methods

The study's case group comprised subjects who met specific inclusion criteria, including an age range of 18 to 80 years, clinical symptoms suggestive of BPPV, and a positive Dix Hallpike test. Conversely, both the case and control groups were subject to exclusion criteria, which encompassed pregnant participants, individuals with a history of head injury, ear infections, maxillary sinusitis, or chronic renal, pulmonary, hematologic, gastrointestinal, or cardiovascular diseases. Furthermore, subjects taking Vitamin D and calcium supplements were excluded from the study, as were those diagnosed with central vertigo.

Detailed history, clinical examination, and investigations were recorded using a designated proforma. Serum 25-OH Vitamin D3 levels were measured in all subjects using a commercially available ELISA kit method, with fasting blood samples collected via venipuncture from the antecubital vein. The study also compared Vitamin D concentrations between the case and control groups, and statistical analysis was conducted using Statistical Package for the Social Sciences (SPSS) Version 21.0 Software for Windows. The data were analyzed in terms of mean, standard deviation (SD), and percentages. A P-value of less than 0.05% was considered statistically significant.

Results

The prevalence of BPPV was found to be higher among individuals aged between 50 and 65 years as shown in Table 1.

Table 1: Age wise distribution of BPPV patients

Age group	Cases		Controls	
	n	%	n	%
21–34	4	13.33	6	20.00
35–50	7	23.33	9	30.00
51–65	15	50.00	12	40.00
>65	4	13.33	3	10.00
Total	30	100.00	30	100.00

Notably, there was a preponderance of females in the study group. However, statistical analysis revealed that the difference in prevalence between the two gender groups was not deemed significant. The difference in Vitamin D levels between the study and control groups was found to be highly significant. These results indicate a substantial association between reduced Vitamin D concentrations and the occurrence of BPPV, highlighting the potential role of Vitamin D deficiency as a contributing factor in the pathogenesis of this vestibular disorder. It was found that there was a statistically significant difference in body mass index (BMI) between the study group and the control group [Table 2].

Table 2: Clinico-demographic variables in study patients

Variable	Cases	Controls	P value
Age (in years) [mean±SD]	48.20±13.90	41.80±14.60	0.31
Female gender	16 (52.50)	14 (47.50)	0.40
Male gender	14 (47.50)	16 (52.50)	0.40
BMI (kg/m2), [mean±SD]	26.80±2.95	24.60±2.50	<0.05
Serum Vitamin D levels (ng/ml)			
30–100 (Normal)	12	7	<0.05
<30 (Insufficiency)	10	7	<0.05
<20 (Deficiency)	8	16	<0.05

The study results indicated that no significant relationship was observed between BPPV and smoking, alcohol consumption, or comorbidities such as diabetes mellitus and hypertension. Among the BPPV patients, unilateral BPPV was the most common presentation. The posterior canal was the most commonly affected followed by the horizontal canal and the anterior canal [Table3].

Table 3: Involvement of semicircular canals in study patients

SCC involved	n	%
Posterior	20	66.67
Horizontal	8	26.67
Anterior	2	6.67
Total	30	100.00

Discussion

Benign Paroxysmal Positional Vertigo (BPPV) represents the most prevalent peripheral vestibular disorder, predominantly characterized by vertigo arising from the inner ear. This condition stands as a primary cause of vertigo, constituting 13.7% of cases, with an estimated annual incidence of approximately 100 per 100,000 individuals within the population [16, 17].

No statistically significant difference in age and sex distribution was observed between the cases and control groups. Similarly, in the study by Jeong et al., a cohort of 100 consecutive patients was investigated, comprising 63 females and 37 males. The study found no statistically significant difference in age and sex distribution among the participants [18]. Our findings indicated that the posterior canal is the most frequently affected by BPPV. Honrubia et al. [19] also reported the prevalence of BPPV subtypes in their clinic, with posterior canal BPPV (p-BPPV) accounting for 93% of cases, horizontal canal BPPV (h-BPPV) for 5%, and anterior canal BPPV (a-BPPV) for 2% [20, 21]. Several factors contribute to the propensity for particle accumulation in the posterior canal. These include its inferior position relative to the utricle when in a supine position, the size of the common crus of the posterior and superior semicircular canals, and its dependent position in both erect and supine postures. Consequently, particles tend to become entrapped in the posterior canal, while any debris that might have entered the superior canal is more likely to descend back into the utricle [22].

Our study revealed a statistically significant difference in BMI values between the two groups ($P < 0.05$). This finding aligns with the study conducted by Jeong et al., which also reported a significant difference ($P = 0.001$) in BMI between the patient and control groups [18]. The link between Vitamin D and BMI is multifaceted. Vitamin D induces a rise in serum parathyroid hormone as a compensatory mechanism, leading to increased calcium uptake by adipocytes. This, in turn, contributes to lipogenesis and promotes weight gain. Additionally, obesity can cause hypovitaminosis D due to increased uptake of 25-Hydroxyvitamin D by adipose tissue. These complex interactions highlight the potential role of Vitamin D in both weight regulation and its interplay with adiposity [23-26].

Numerous studies have indicated that the regulation of calcium concentration in the endolymph involves various transporter channels, including plasma membrane Ca2+ pumps, Na+/Ca2+ exchangers, and epithelial Ca2+ channels (calbindins). These calcium channels' functionality is further sustained through the expression of specific Ca2+ binding proteins, namely calbindin-D9k and calbindin-D28k. Notably, 1, 25-dihydroxyvitamin D3 plays a crucial role in upregulating the expression of these proteins, as well as in calcium homeostasis [27]. Consequently, a disruption in calcium metabolism due to Vitamin D deficiency may lead to the development of BPPV. As such, Vitamin D deficiency emerges as a potential contributing factor in the onset of BPPV. These findings emphasize the significance of Vitamin D in maintaining calcium homeostasis and its plausible association with BPPV pathogenesis [28, 29].

Conclusion

Our study revealed a significant association between low Vitamin D levels and the majority of BPPV patients. High BMI may also contribute to BPPV pathogenesis, but further multicenter studies are required to clarify its exact role. In conclusion, there is a direct correlation between Vitamin D concentration and the incidence of BPPV.

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