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REVIEW / DERLEME

The Effects of Vitamin D on Benign Paroxysmal Positional Vertigo

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ABSTRACT

Benign paroxysmal positional vertigo (BPPV) is one of the most common peripheral vestibular disorders and is characterized by brief episodes of vertigo triggered by changes in head position, which occur as a result of the displacement of otoliths located in the inner ear. The majority of cases are idiopathic. In recent years, research has shown that disturbances in calcium metabolism, particularly vitamin D deficiency, may play a role in the development and recurrence of benign paroxysmal positional vertigo. Vitamin D plays a crucial role in regulating the calcium-phosphorus balance and preserving the structural integrity of otoconia. Within the scope of this study, recent scientific literature published in the past decade has been reviewed to evaluate the effects of vitamin D on benign paroxysmal positional vertigo. Studies investigating the potential interactions between these two variables have been conducted through a comprehensive literature review. These findings indicate that individuals with low vitamin D levels have a greater incidence of benign paroxysmal positional vertigo and that vitamin D supplementation may contribute to clinical improvement, particularly in recurrent cases. These findings emphasize that the vitamin D level should be considered an important factor in the management of BPPV; however, further research is needed to determine optimal treatment strategies and to fully understand the role of vitamin D in the pathophysiology of BPPV.
Keywords: Benign paroxysmal positional vertigo, otoconia, vitamin D deficiency.

D Vitamininin Benign Paroksismal Pozisyonel Vertigo Üzerine Etkisi

ÖZET

Benign paroksismal pozisyonel vertigo (BPPV), iç kulakta yer alan otolitlerin yer değiştirmesi sonucu ortaya çıkan ve başın pozisyonundaki değişimle tetiklenen kısa süreli vertigo ataklarıyla karakterize, en yaygın periferal vestibüler bozukluklardan biridir. Vakaların çoğu idiopatiktir. Son yıllarda yapılan araştırmalar, kalsiyum metabolizmasındaki bozuklukların, özellikle de D vitamini eksikliğinin, benign paroksismal pozisyonel vertigo gelişiminde ve nükslerinde rol oynayabileceğini ortaya koymuştur. D vitamini, kalsiyum-fosfor dengesinin düzenlenmesinde ve otokonyaların yapısal bütünlüğünün korunmasında kritik bir rol üstlenmektedir. Bu çalışma kapsamında, son on yıl içerisinde yayımlanmış güncel bilimsel kaynaklar incelenerek, D vitamininin benign paroksismal pozisyonel vertigo üzerindeki etkisine yönelik bulgular değerlendirilmiş; iki değişken arasındaki olası etkileşimleri araştıran çalışmalar kapsamlı bir literatür taraması ile bütüncül bir çerçevede ele alınmıştır. Çalışmalar, düşük D vitamini seviyelerine sahip bireylerde benign paroksismal pozisyonel vertigo sıklığının daha yüksek olduğunu ve D vitamini takviyesinin, özellikle tekrarlayan olgularda, klinik iyileşmeye katkı sağlayabileceğini göstermiştir. Sonuçlar, vitamin D düzeyinin BPPV yönetiminde dikkate alınması gereken önemli bir faktör olduğunu vurgulamaktadır; ancak optimal tedavi stratejilerini belirlemek ve vitamin D'nin BPPV patofizyolojisindeki rolünü tam olarak anlamak için daha fazla araştırmaya ihtiyaç vardır.
Anahtar Kelimeler: Benign paroksismal pozisyonel vertigo, otokonya, D vitamini eksikliği.

1. Introduction

Benign Paroxysmal Positional Vertigo is a peripheral vestibular disorder characterized by brief and sudden episodes of vertigo triggered by movements of the head in specific directions. The term “benign” indicates that the condition is not serious or life-threatening, whereas “positional” refers to the fact that symptoms are elicited by changes in head position (1,2). Common triggers in BPPV patients include changes in head position such as turning in bed, getting out of bed, bending forward, or looking upward. These episodes typically last between 10 and 30 seconds and occur in response to specific head positions (3). Accompanying symptoms may include nausea, a sensation of vomiting, and temporary imbalance (4).

At the core of the disorder lies the displacement of calcium carbonate crystals, known as otoconia, which detach from the utricle, one of the otolithic organs in the inner ear responsible for balance perception, and migrate into the semicircular canals, most commonly the posterior canal (5). These crystals cause abnormal endolymphatic fluid movements during head position changes, leading to the transmission of erroneous balance signals to the brain via the vestibular nerve. As a result, the individual experiences a sensation of spinning despite being in a stationary position (6,7).

BPPV most commonly affects individuals between the ages of 41 and 60 years, with the 51–60 years being the most affected, followed by individuals aged 61–80 years (8). BPPV is more

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prevalent in women than in men; consequently, it is hypothesized that gender may serve as a risk factor for its development (9).

Two major theories have been proposed for the pathophysiology of BPPV: canalithiasis and cupulolithiasis. According to canalithiasis theory, the dislodged otoconia from the utricle move freely within the endolymph of the semicircular canals. When the head position changes, the movement of these particles within the canal induces endolymph flow, which in turn leads to overstimulation of the vestibular nerve (10). In contrast, the cupulolithiasis theory posits that these otoliths adhere to the cupula located in the ampullary region of the canal. This increases the specific gravity of the cupula, causing it to move abnormally under the influence of gravity and generating inappropriate vestibular signals during head movements (6). Both mechanisms play a significant role in explaining the transient vertigo episodes that occur in relation to changes in head position (11).

Most cases of BPPV are primary (idiopathic), with no identifiable underlying cause. However, in some cases, inner ear conditions such as Ménière's disease or vestibular neuritis may lead to a secondary form of BPPV. Head or neck trauma and mechanical effects during dental or ear surgeries may also cause dislodgement of otoconia (8). Disorders affecting calcium metabolism and bone Health such as osteoporosis can compromise the structural integrity of otoconia, thereby increasing the risk of BPPV (12). Furthermore, systemic diseases such as hypertension and hyperlipidemia, which impair vascular health, may also disrupt inner ear circulation and contribute to the development of BPPV (13). Finally, vitamin D deficiency, which compromises the stability of otoconia, has been implicated as a contributing factor in the onset of BPPV, particularly among older adults (14).

Vitamin D plays a critical role in maintaining calcium homeostasis and the structural integrity of otoliths. Its deficiency may lead to the degeneration and displacement of otoconia, thereby predisposing individuals to the development of BPPV (15). Recent studies indicate that low vitamin D levels are associated with a greater frequency of BPPV recurrence, suggesting that sufficient vitamin D intake could reduce this risk (16-18). Within the scope of this review, we evaluate the current scientific literature from the past decade to examine the relationship between vitamin D and BPPV and to assess the potential effects of vitamin D supplementation on recurrence rates.

1.1. Materials and Methods

A comprehensive literature search was conducted to evaluate the current scientific evidence on the relationship between vitamin D and BPPV. The literature search was performed systematically via prominent scientific databases, including PubMed, Web of Science, Scopus, and Google Scholar. The search strategy utilized various combinations of keywords in English to ensure broad coverage of the relevant literature. The primary search terms included: "benign paroxysmal positional vertigo, BPPV, vitamin D, vitamin D deficiency". The conceptual framework illustrating the relationship between Vitamin D and BPPV (Figure 1) was created by the authors using Microsoft Word.

Only studies published within the last ten years were considered for this review and were compiled under the heading "Studies on the Subject." This time frame was chosen to reflect the most up-to-date scientific findings and methodologies. According to the inclusion criteria, priority was given to studies with a strong level of evidence, such as randomized controlled trials (RCTs), case-control studies, systematic reviews, and meta-analyses. The methodological rigor and quality of the selected studies were considered essential for a holistic evaluation of the literature. Conversely, case reports, letters to the editor, conference abstracts, and studies for which full-text articles were unavailable were excluded from the review. The studies identified through this systematic search were selected and summarized to examine the associations between the two variables, the potential effects of vitamin D supplementation on treatment outcomes, and the underlying pathophysiological mechanisms.

1.2. Vitamin D: Synthesis and Function

Vitamin D is a crucial fat-soluble prohormone for human physiology (19). It is acquired through two pathways: exposure to sunlight (ultraviolet B) and diet (20). In the skin, vitamin D is synthesized from the 7-dehydrocholesterol molecule, forming cholecalciferol (vitamin D3) (21). Dietary sources include fatty fish, egg yolks, liver, and fortified foods (22). Both the inactively synthesized and ingested forms undergo a metabolic process to become biologically active (23). This process is completed first by 25-hydroxylation in the liver to form 25-hydroxyvitamin D (25-OH D), followed by 1-hydroxylation in the kidneys to yield its most active form, 1,25-dihydroxyvitamin D (calcitriol) (24).

The primary physiological role of vitamin D is to regulate calcium and phosphorus homeostasis. It protects the health of the skeletal system by enhancing intestinal calcium absorption and supporting bone mineralization (25). This explains why vitamin D is vital for maintaining the structural integrity of otoconia, which are made of calcium carbonate crystals and are responsible for balance perception in the inner ear. Its deficiency can lead to calcium metabolism disturbances, causing weakening and degeneration of theotoconia (12,14). This condition can facilitate the displacement of otoconia, predisposing individuals to the development of benign paroxysmal positional vertigo (BPPV) (15). Therefore, vitamin D is a critical factor not only for bone health but also for the health of the vestibular system (12,14,15).

1.3. The Relationship Between BPPV and Vitamin D

BPPV is a balance disorder resulting from the displacement of otoconia within the semicircular canals of the vestibular system. It is characterized by sudden and brief episodes of vertigo triggered by changes in head position. Otolithic organs, which are located in the inner ear, play a vital role in maintaining balance, and proper mineral balance is critical for their function. At this point, vitamin D plays an important role in preserving the structural integrity of otoliths by regulating calcium absorption in the body (1).

Vitamin D enhances calcium absorption from the intestines, stabilizes blood calcium levels, and supports bone health. This is achieved by upregulating calcium-binding proteins (calbindins) and calcium channels in intestinal cells, which facilitates the active transport of calcium into the bloodstream. Moreover, vitamin D deficiency may lead to imbalances in calcium metabolism, weakening the mineral structure of both bones and otoliths in the ear. This condition can result in the dislodgement of otoconia, especially in older individuals, allowing them to migrate into the semicircular canals and predisposing them to BPPV (16).

The role of vitamin D in the pathophysiology of BPPV is based on the theory that adequate vitamin D levels help maintain otoliths

in their correct position. A deficiency may weaken the otoliths and cause calcium crystals to become dislodged and migrate into the semicircular canals of the vestibular system. BPPV is then triggered when these displaced crystals move inappropriately in response to head position changes. This association suggests that vitamin D may have an important indirect effect on vestibular health (15).

The role of vitamin D is becoming increasingly important not only for bone health but also for vestibular system health. Therefore, maintaining adequate vitamin D levels in patients with BPPV has emerged as an important consideration in treatment. Monitoring vitamin D status may serve as a key strategy in reducing the risk of recurrence in the management of BPPV (16).

Recent studies have revealed that patients with BPPV often have low vitamin D levels. Vitamin D deficiency is also associated with bone diseases such as osteoporosis, which may help explain the increased prevalence of BPPV with increasing age. In summary, vitamin D deficiency may be a risk factor for BPPV, and supplementation with vitamin D in patients with BPPV could have a beneficial effect on treatment outcomes (16-18).

However, unlike previous studies that focused on individual findings or specific patient populations, this review provides a comprehensive synthesis of the literature from the past decade. By systematically examining both the pathophysiology of BPPV and the role of vitamin D deficiency in its development and recurrence, this study offers an updated and in-depth perspective. Moreover, highlighting methodological variations across studies and drawing attention to clinical implications contributes to a better understanding of how monitoring and correcting vitamin D levels may improve treatment outcomes in BPPV management.

Figure 1, prepared by the authors via Microsoft Word, illustrates the pathophysiological link between vitamin D deficiency and the recurrence of BPPV, as well as the potential preventive effect of vitamin D supplementation.

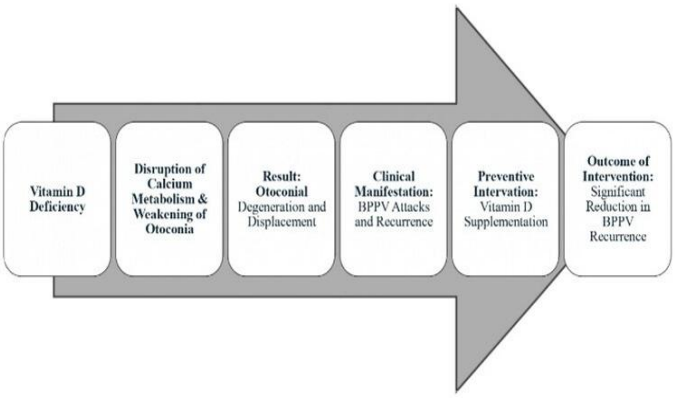

Figure 1. The relationship between Vitamin D and BPPV: Pathophysiology to clinical management

1.4. Studies on the Subject

In a case-control study, Sheikhzadeh et al. (18) investigated the effect of normalizing serum vitamin D levels on the recurrence of BPPV. The study included 54 patients, divided into a treatment group that received vitamin D supplementation and a control group that did not. At baseline, the mean vitamin D levels were comparable in both groups (approximately 11 ng/mL). After two months of supplementation, the treatment group's vitamin D level in the treatment group significantly increased to 34.2 ng/mL, whereas the vitamin D level in the control group remained unchanged. During the follow-up, the recurrence rate in the treated group was 14.8%, which was significantly lower than the 96.3% reported in the control group. These findings suggest that normalizing serum vitamin D levels may be an effective strategy to reduce BPPV recurrence.

Kong et al. (26) conducted a randomized, double-blind, placebo-controlled study with a one-year follow-up to investigate the effect of vitamin D supplementation on the recurrence of BPPV. In this study, only vitamin D supplementation was administered to patients with low serum 25-OH vitamin D levels (<20 ng/mL), without the co-administration of calcium. The mean serum vitamin D level in the vitamin D group was initially 13.3 ng/mL, and it significantly increased to 28.2 ng/mL at the six-month mark and 29.4 ng/mL at one year. This increase was statistically significant compared with that in the placebo group. The study's main finding ot he study, which was based on Kaplan-Meier survival analysis, was a significant reduction in BPPV recurrence events in the vitamin D group compared with the placebo group at both six months and one year. Furthermore, the recurrence rate in the vitamin D group was substantially lower than that in the placebo group, with rates of 5.3% versus 26.7% at six months and 9.5% versus 44.4% at one year.

A multicenter randomized controlled trial by Jeong et al. (17) investigated the role of vitamin D and calcium supplementation in preventing BPPV recurrence within a larger patient population. The study, which included a total of 1,050 patients (518 in the intervention group, 532 in the observation group), included individuals whose serum vitamin D levels were less than 20 ng/mL. The findings revealed that the annual recurrence rate in the intervention group (0.83) was significantly lower than that in the observation group (1.10). Furthermore, the proportion of patients who experienced recurrence was lower in the intervention group (37.8%) than in the observation group (46.7%). These results support the idea that vitamin D and calcium supplementation may be beneficial in preventing BPPV recurrence.

A case-control study by Talaat et al. (15) compared vitamin D levels in BPPV patients with those in healthy controls. The study included 80 BPPV patients and 100 healthy controls. The mean vitamin D level in the BPPV group was significantly lower (14.2 ng/mL) than that in the control group (22.5 ng/mL). These findings suggest that low vitamin D levels may be a risk factor not only for developing BPPV but also for its recurrence, as vitamin D levels were found to be even lower in patients with recurrent BPPV than in those with non-recurrent BPPV.

Al-Rawi et al. (16) investigated the relationship between benign paroxysmal positional vertigo (BPPV) and vitamin D deficiency. The study included 40 patients diagnosed with BPPV and 80 control subjects. Serum vitamin D levels were significantly lower in the BPPV group than in the control group (15.46 ng/mL vs. 23.60 ng/mL). Moreover, the likelihood of developing BPPV was greater among individuals with vitamin D deficiency. Among BPPV patients, those who experienced recurrence had significantly lower vitamin D levels, a difference that was statistically significant (12.62 ng/mL vs. 18.3 ng/mL). These findings suggest that vitamin D deficiency may play a significant role in the occurrence and recurrence of BPPV.

Bener et al. (27) investigated the relationships between serum vitamin D and calcium levels and BPPV using a case-control study

design. The study involved 177 patients diagnosed with BPPV and 656 control subjects. The findings revealed a statistically significant difference in the serum vitamin D and calcium levels between the two groups. Low levels of both vitamin D and calcium were identified as potential risk factors for BPPV. The study concluded that supplementation with vitamin D and calcium could be considered a preventative measure in BPPV patients.

A meta-analysis conducted by Yang et al. (28) evaluated the impact of vitamin D supplementation on recurrence rates in patients with vitamin D deficiency and idiopathic benign paroxysmal positional vertigo (BPPV). The analysis included seven studies with a total of 1333 participants (602 in the vitamin D supplementation groups and 731 in the control groups). The results revealed that patients receiving vitamin D supplementation had a significantly lower recurrence rate than controls did, corresponding to a 59% reduction in BPPV recurrence. The study revealed a Risk Ratio (RR) of 0.41 for the supplementation group, indicating a nearly twofold decrease in the risk of recurrence. These findings suggest that correcting vitamin D deficiency may be an effective strategy to reduce BPPV recurrence and that low vitamin D levels are a significant risk factor for the disease recurrence.

In a retrospective study, Altın and Ertuğrul (29) analyzed vitamin D and B12 deficiencies in 131 patients diagnosed with either acute or subclinical BPPV. Vitamin D deficiency was present in 35.9% of patients, and those individuals experienced more frequent recurrences within the first year. No significant association was found between B12 deficiency and BPPV recurrence.

Özşimşek and Karaçay (30) compared serum 25-hydroxyvitamin D (25-OH vitamin D) and calcium levels between idiopathic BPPV patients and a healthy control group without vertigo symptoms. The study included 409 BPPV patients and 338 controls. The average 25-OH vitamin D level was 15.74 ng/mL in the BPPV group and 17.91 ng/mL in the control group.

These findings emphasize the association between low vitamin D levels and the development of BPPV. A summary of the studies related to the association between BPPV and vitamin D is presented in Table 1

Table 1. Summary of the studies on the subject.

Authors (Year)	Type of study	Study sample	Vitamin D level (ng/ml)	Possible effects	Supplementation details (Dose/duration)
Talaat et al. (2015) (15)	Case-Control	80 patients (BPPV), 100 healthy volunteers (control)	BPPV patients had significantly lower 25-hydroxyvitamin D levels than the control group	The study found that very low vitamin D levels are associated with recurrence.	NR
Al-rawi et al. (2024) (16)	Retrospective Case-Control	40 (St. Gr.) 80 (Co. Gr.)	St. Gr.: 15.46 ± 6.14 Co. Gr.: 23.60 ± 12.58	Vitamin D deficiency is associated with the occurrence and recurrence of BPPV.	NR
Jeong et al. (2020) (17)	Randomized controlled trial	518 (Intervention Gr.), 532 (Observation Gr.)	low initially, normal after supplementation	Proportion of Patients with Recurrence: Lower in the intervention group (37.8%) than in the observation group (46.7%)	400 IU of vitamin D and 500 mg of calcium carbonate, twice a day, for 1 year.
Sheikhzadeh et al. (2016) (18)	Case-Control	27 (St. Gr.) 27 (Co. Gr.)	St Gr. (Initial): 10.7, (After Supp.): 34.2 Co. Gr. (Initial): 11.4, (After Supp.): 10.6	St. Gr.: 14.8% recurrence Co. Gr.: 96.3% recurrence	50.000 IU vitamin D weekly for 2 months. Follow-up: 6 months
Kong et al. (2024) (26)	Randomized Controlled Trial (Double-Blind)	20 (St. Gr.), 18 (Placebo Gr.)	St. Gr. (Initial): 13.3 ± 3.9, (After 1 year): 29.4 ± 10.9 Placebo Gr. (Initial): 13.8 ± 3.7, (After 1 year): 13.8 ± 3.7	Recurrence Rate (After 1 year): St. Gr. 9.5%, Placebo Gr. 44.4%	7000 IU of vitamin D weekly for 1 year (without calcium supplementation)
Bener et al. (2024) (27)	Case-Control	177 (St. Gr.) 656 (Co. Gr.)	BPPV patients had significantly lower serum vitamin D and calcium levels compared to the control group.	Low serum levels of vitamin D and calcium are associated with BPPV.	NR
Yang et al. (2021) (28)	Meta-Analysis	7 studies (602 patients, 731 Co. Gr.)	Patients with vitamin D deficiency (<20 ng/mL) were included.	A 59% reduction in the recurrence rate was observed in the supplementation group.	Variable. Different studies used different doses and durations.
Altın & Ertuğrul (2023) (29)	Retrospective	131 BPPV patients	Numerical values (ng/mL) were not provided in the article.	In patients with vitamin D deficiency, BPPV attacks recurred more frequently within the first year.	NR
Özşimşek & Karaçay (2022) (30)	Retrospective Case-Control	409 (St. Gr.) 338 (Co. Gr.)	St. Gr.: 15.74 Co. Gr.: 17.91	Low vitamin D levels are associated with the occurrence of BPPV. There was no significant difference in calcium levels.	NR

St. Gr.: Study Group, Co. Gr.: Control Group, IU: International Unit, NR: Not Reported

2. Conclusion and Recommendation

Benign paroxysmal positional vertigo (BPPV) is a common peripheral vestibular disorder characterized by benign, short-lived vertigo attacks triggered by head movements, which can significantly impair quality of life. The underlying mechanism of the disorder is based on abnormal stimulation of the semicircular canals resulting from the displacement of the otoconia. Although the exact etiology remains unclear, BPPV is particularly prevalent among older adults and tends to recur, leading to a long-term physical and psychological burden on affected individuals.

In recent years, increasing scientific interest has emerged regarding the potential influence of vitamin D levels on BPPV. Vitamin D plays a critical role not only in bone health but also in calcium metabolism and the functional integrity of the vestibular system. Deficiency of vitamin D, which is involved in the mechanisms maintaining the structural integrity of the otolithic organs, is considered a potential contributing factor to the degeneration of otoconia, thereby facilitating the development of BPPV.

A substantial portion of the literature has demonstrated a significant association between vitamin D deficiency and BPPV. Low vitamin D levels have frequently been reported in individuals with recurrent BPPV episodes. Furthermore, some studies have suggested that vitamin D supplementation may reduce the frequency of these episodes. These findings imply that vitamin D may act not only as a risk indicator but also as a potential protective factor. Accordingly, monitoring vitamin D levels in BPPV patients and providing appropriate supplementation in cases of deficiency may represent a beneficial strategy for improving disease outcomes.

3. Contribution to the Field

This review makes a valuable and distinctive contribution to the literature by thoroughly examining research on the relationship between BPPV and vitamin D levels published over the past decade. Unlike prior studies, which often focus on specific patient populations or individual findings, this work explores the pathophysiology of BPPV alongside the role of vitamin D deficiency in both the onset and recurrence of the condition from a broad perspective. By evaluating methodological variations across studies and highlighting their clinical implications, this review offers a more detailed and comprehensive understanding of how vitamin D status may influence BPPV development and management. In particular, the emphasis on the potential of vitamin D supplementation to reduce recurrence rates in BPPV patients underscores the clinical importance of monitoring and correcting vitamin D deficiency. This comprehensive approach not only clarifies existing controversies but also provides actionable insights for clinicians and directions for future research, reinforcing the importance of vitamin D assessment in BPPV treatment protocols.

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Conflict of Interest

There is no conflict of interest regarding any person and/or institution.

Authorship Contribution

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