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ORIGINAL RESEARCH

Role of Vitamin D Deficiency in Benign Paroxysmal Positional Vertigo

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ABSTRACT

Background: To evaluate the role of vitamin D deficiency in benign paroxysmal positional vertigo.

Materials and Methods: One hundred and twenty eight patients of benign paroxysmal positional vertigo of both genders were included. Serum 25-hydroxyvitamin D (25-OHD) was measured using ELISA method and concentration less than 20 ng/ml was considered as deficiency. Patients were divided into 3 groups. Group I (treatment group -with serum 25-OHD deficiency) received Epley maneuver + supplemental vitamin D and group II (nontreatment group - with serum 25-OHD deficiency) received only Epley maneuver therapy. Group III (non-treatment group - with normal vitamin D level) also received only Epley maneuver therapy.

Results: Group I comprised of 18 males and 26 females, group II 20 males and 22 females and group III 21 males and 21 females. The mean(\pm SD) vitamin D level at baseline, 2 months and 6 months in group I was 11.4 ± 1.6 mg/ml, 34.1 ± 4.16 mg/ml and 35.9 ± 1.8 mg/ml respectively. In group II was 10.9 ± 1.4 mg/ml, 10.5 ± 2.01 mg/ml and 11.6 ± 3.2 mg/ml. In group III was 33.5 ± 1.6 mg/ml, 34.8 ± 3.2 mg/ml and 35.2 ± 4.1 mg/ml respectively. The difference was statistically significant (P< 0.05). It was found that the intensity of BPPV decreased in group I, increased in group II and decreased in group III over the period of 2 months and 6 months. The difference was found statistically significant (P< 0.05).

Conclusion: The correction of vitamin D deficiency in BPPV provides additional benefit to rehabilitation therapy regarding duration of improvement. Supplementation of vitamin D should be considered in patients with frequent attacks of BPPV, especially when serum vitamin D is subnormal.

Keywords: benign paroxysmal positional vertigo, VVAS, vitamin D.

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INTRODUCTION

Among the vestibular disorders, benign paroxysmal positional vertigo (BPPV) is the most common cause of recurrent vertigo. [1] The unexpected occurrence of benign paroxysmal positional vertigo is serious concern in patients. [2]

Its incidence rate among population is approximately 10%. It is more common around age of sixty. Dislodgement of calcium carbonate crystals from the utricle into the semicircular canals is one of the leading concepts of pathogenesis of BPPV. Vitamin D plays a major role in calcium metabolism which may affect the calcium carbonate crystals density and matrix. The link between vitamin D and BPPV was first described in 2013. Since then, several studies across the globe have reported a higher prevalence of vitamin D deficiency in patients with BPPV than in general population. A possible explanation for this is low sunshine exposure resulting from immobilization due to vertigo. Studies regarding the beneficial effect of vitamin D therapy on muscle strength have been observed in the past. Improvement of postural balance is an important component of treatment in older patients with dizziness and imbalance. Restoration of serum vitamin D improves muscle strength in lower limbs and is expected to improve balance and fall. This study was conducted to evaluate the role of vit D in benign paroxysmal positional vertigo.

MATERIALS & METHODS

This Prospective and Comparative Study was conducted in the Department of Otorhinolaryngology and Head & Neck Surgery, S.M.G.S Hospital, Government Medical College Jammu, over a period of 18 months w.e.f January 2021 to June 2022, on 128 patients of benign paroxysmal positional vertigo, after obtaining informed written consent from the participants.

The study was conducted after taking prior approval from the Institutional Ethical Committee.

Inclusion criteria was all patients between age group 20-70 years who had history of at least two or more attacks of BPPV over 6 months. Exclusion criteria was patients with history of head trauma, ear surgery, long term steroid intake, vitamin D supplementation, infectious diseases of the ear, maxillary sinuses and patients with chronic renal, pulmonary, hematologic, gastrointestinal, cardiovascular diseases. To exclude confounding factors only patients who were diagnosed with unilateral posterior semi-circular canal BPPV were included.

Data such as name, age, gender etc. was recorded. A thorough clinical examination, and laboratory tests were performed. Intensity of BPPV episodes in the patients was assessed subjectively and expressed as VVAS score (0- 10), where 0 indicated no vertigo and 10 indicated severe attacks of vertigo. Serum 25-hydroxyvitamin D (25-OHD) was measured using ELISA method and concentrations less than 20 ng/ml were considered as deficiency.

Patients were divided into 2 groups. Group I was treatment group (with serum 25-OHD deficiency) which received Epley maneuver + supplemental vitamin D) and group II was non-treatment group – with vitamin D deficiency which received Epley maneuver therapy alone. Group III (non-treatment group - with normal vitamin D level) also received only Epley maneuver therapy.

All patients received rehabilitation treatment using Epley's method once a week for one month. Vitamin D was administered at 60000 IU orally, weekly for two months and then 60000 IU orally, monthly over the 6-months. The results were compiled and subjected for statistical analysis using Mann Whitney U test. P value less than 0.05 was considered statistically significant. SPSS software version 28 was applied for analysis.

RESULTS

Table I Patients distribution according to Gender

Groups	Group I (treatment) n=44	Group II (non- treatment) n=42	Group III (control) n=42
Status	Epley maneuver + supplemental vitamin D	* *	Epley maneuver therapy alone.
M:F	18:26	20:22	21:21

Group I comprised of 18 males and 26 females, group II 20 males and 22 females and group III 21 males and 21 females (Table I).

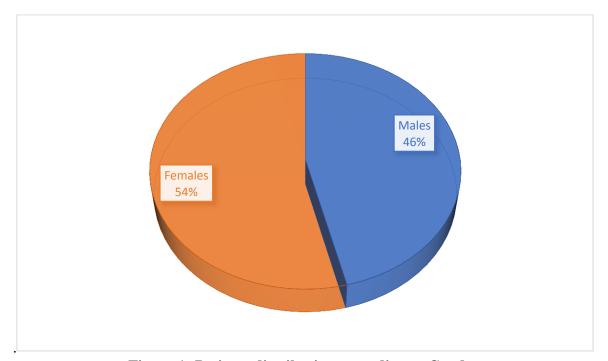


Figure 1: Patients distribution according to Gender

Table II Group-wise serum vit D levels in patients with BPPV at baseline, 2 months and 6 months in all groups

Groups	Baseline	2 months	6 months	P value
	Mean ±SD	Mean± SD	Mean ±SD	
Group I	11.4±1.6	34.1±4.16	35.9±1.8	0.05
Group II	10.9±1.4	10.5±2.01	11.6±3.2	0.04
Group III	33.5±1.6	34.8±3.2	35.2±4.1	0.91

The mean vitamin D level at baseline, 2 months and 6 months in group I was 11.4 mg/ml, 34.1 mg/ml and 35.9 mg/ml respectively. In group II was 10.9 mg/ml, 10.5 mg/ml and 11.1 mg/ml. In group III was 33.5 mg/ml, 34.8 mg/ml and 36.2 mg/ml respectively. The difference was significant (P< 0.05) (Table II).

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Table III Influence of supplemental vitamin D on intensity of benign paroxysmal positional vertigo(Mean VVAS score)

Groups	Baseline	2 months	6 months	P value
Group I	7.4	0.42	0.23	0.04
Group II	7.8	0.21	6.8	0.02
Group III	6.4	0.27	0.31	0.05

It was found that the intensity of BPPV episodes decreased in group I, increased in group II and decreased in group III over 2 months and 6 months follow-up period. The difference was found to be significant (P< 0.05) (Table III).

DISCUSSION

BPPV is most common peripheral vestibular disorder and presents as brief episodic positionally provoked vertigo. Calcium metabolism is involved in synthesis and absorption of otoconia and vitamin D is required for the calcium metabolism. Disturbance in the metabolism of both vitamin D and calcium is probably the key element of the pathogenesis of BPPV. [8,9] Vitamin D level and deposition of calcium crystals affect the otoconia matrix and density similar to its effect on bone structures. [10,11] Vitamin D insufficiency is correlated with the severity of BPPV and its recurrence. In fact, the recurrence attacks of BPPV may decrease with vitamin D supplementation. [12,13] This study was undertaken to assess the role of vit D in patients of benign paroxysmal positional vertigo. In our Study (n=128), male: female ratio was 1:1.2. Group I comprised of 18 males and 26 females, group II had 20 males and 22 females and group III had 21 males and 21 females. Females were more susceptible to BPPV than males in both groups, and there was no statistically significant difference for age or sex distribution in either group. In our study it was observed that the disease was more common in women than in men . Adult female patients seem to be more prone to the occurrence of BPPV, as the BPPV prevalence in female could be correlated to hormonal variations and associated demineralization and metabolic changes. The latter condition could reduce bone mineral density, facilitating the detachment of otoconia from the utricular neuroepithelium. Similar results were seen in a study by Elmoursy M (2021).[18-21]

In our study, the mean vitamin D level at baseline, 2 months and 6 months in group I was 11.4 mg/ml, 34.1 mg/ml and 35.9 mg/ml respectively. In group II they were 10.9 mg/ml, 10.5 mg/ml and 11.1 mg/ml respectively. In group III they were 33.5 mg/ml, 34.8 mg/ml and 36.2 mg/ml respectively. The results were statistically significant indicating the association of vit D in BPPV. Kasem MME (2021) in their study of 50 patients with first attack of BPPV compared serum calcium and serum Vitamin D3 levels and found statistically significant difference in Serum Vit D3 Levels in BPPV groups with mean Vit D levels of 22.8±11.26 ng/ml. Similar results were also seen by Jeong et al who found lower serum values of vit D in BPPV.

Abdelmaksoud et al^[15] assessed the relation between recurrent attacks of BPPV and Vitamin D deficiency. In their case control study (n=40) Patients were divided into two groups; group A (20 patients) received Vitamin D supplementation in addition to canal repositioning maneuver and group B (20 patients) treated by canal repositioning maneuver only. Follow up of all patients for 6 months, neuro-otological assessment was repeated and recurrent attacks were recorded. Serum vitamin D was repeated after 6 months. This study included 14 males and 26 females age ranged from 35 to 61 years, Average serum vit D level at the first visit was $(12.4 \pm 2 \text{ ng/ml})$ for group A, and $(12.2 \pm 1.7 \text{ ng/ml})$ for group B indicating that all

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patients had low serum level of vit D (below 20 ng/ml). Recurrent BPPV episodes, were significantly lower in group A than that of group indicating a statistically significant relationship between BPPV recurrence and low serum Vitamin D, whereas a study by Rhim et al(2020) found no significant effect on recurrence of BPPV in patients with vitamin D deficiency.

In our study, the intensity of BPPV (as assessed by VVAS score) decreased in group I, increased in group II and decreased in group III over 2 months and 6 months period after repositional manoeuvre. The difference was found to be significant (P< 0.05). Sheikhzadeh et al^[14] also determined the influence of vitamin D supplementation on intensity of BPPV. In his study, after two months of treatment, in both vitamin D treated and non-treated groups the intensity of BPPV decreased significantly as compared with control (P=0.001 for both groups) but at endpoint, the intensity of BPPV aggravated and regressed to the baseline value in vitamin D deficient non-treated group (P=0.001) whereas, in vitamin D treated group, improvement of BPPV remained stable and unchanged over the study period.

Talat et al^[16] in a follow-up study of vitamin D deficient patients demonstrated that raising serum 25-OHD > 10 ng/ml by treatment significantly decreased the recurrences as well as the number of BPPV attacks as compared with those who had less than 10 ng/ml increment. Jeong et al^[17] showed that in patients with serum vitamin D between 10-20 ng/ml, the risk of BPPV increases 3.8 times, whereas, in patients with serum vitamin D less than 10 ng/ml, the risk increases by odds of 23.

CONCLUSION

The correction of vitamin D deficiency in BPPV provides additional benefit to rehabilitation therapy regarding duration of improvement. Supplementation of vitamin D should be considered in patients with frequent attacks of BPPV, especially when serum vitamin D is subnormal.

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