

## Original Research Article

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# A prospective analysis of vitamin D and recurrent benign paroxysmal positional vertigo

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## ABSTRACT

**Background:** The aim of the present study was to prospectively evaluate the role of vitamin D in the recurrence of Benign paroxysmal positional vertigo (BPPV). The BPPV is one of the commonest causes for peripheral vertigo. Many studies stated that BPPV can be associated with many co-morbidities and vitamin deficiencies.

**Methods:** In our study, we prospectively conducted an observational study in patients suffering from BPPV, from a study period of June 2015 to June 2018, to evaluate the role of vitamin D and calcium in those patients.

**Results:** Three forty eight patients between the age group of 40 to 82 years, suffering from BPPV were included in the study. It includes 235 females and 113 male patients. Out of these 108 patients had noticed vitamin D deficiency and were randomly divided into 2 groups in which group A received Vitamin D supplementation and Group B did not received vitamin D supplements. Calcium levels were found to be less in 15 patients between 7 and 10.5 mg/dl, however a larger sample is required for its correlation. All patients were followed up for 1 year.

**Conclusions:** The study showed a significant decrease of BPPV in those who received vitamin D supplementation.

**Keywords:** Benign paroxysmal positional vertigo, Vitamin D, Calcium, Recurrence

## INTRODUCTION

The Benign paroxysmal positional vertigo (BPPV) is one of the commonest cause for peripheral vertigo.<sup>1,2</sup> BPPV is due to inappropriate stimulation of vestibular labyrinthine structures in response to changes in head position, with respect to gravity, by the sequestered otoconia.<sup>3</sup> Many studies stated that BPPV can be associated with many co-morbidities and can affect the quality of life. Schuknecht's "cupulolithiasis" theory and Hall's "canalolithiasis" theory do not explain why BPPV increases in certain co-morbidities.<sup>4,5</sup>

The formation of otoconia in otolith organs like macula and cristae are very complex and a thorough understanding of the molecular mechanisms is required.

They are crystals of calcium carbonate and proteins in the otoconial membrane.

Vitamin D {1,25(OH)<sub>2</sub>D} is a steroid hormone.<sup>6</sup> It is synthesized endogenously in response to ultra violet radiation of the skin and from dietary sources like dairy products, fishes, egg yolks, animal fats and in very small amounts in vegetables and cereals. They are fat soluble vitamins. Vitamin D involves in the distribution of calcium and phosphate. Vitamin D increases the absorption of calcium from the intestine and the kidney. The maintenance of blood calcium is controlled by parathyroid hormone. Vitamin D has direct effect on the mechanism of deposition of calcium and phosphate in the bone, teeth and otoconial particle formation in the vestibular system.

Otoconia are found embedded in the gelatinous otoconial membrane in the macula. Size of otoconia is around 20 µg. It is composed of calcium carbonate and glycosylated protein. Vitamin D is essential for the development and maintains of otoconia and it may also induce changes in the otolithic organs. Increased calcium resorptions reduce the capacity to dissolve the dislodged otoconia owing to the increased concentration of free calcium in the endolymph. These undissolved otoconia in the endolymph cause BPPV. Human otoconia undergo degeneration regularly and accelerated otoconial degeneration seen in elderly people.<sup>7,8</sup> Waringhoff, Bayer et al found that the associated co-morbidities increases the otoconial degeneration.<sup>9</sup> Normal vitamin D requirement is 400-800 IU per day. Normal calcium requirement is 1 gm per day for adults. Above 40000 IU of vitamin D leads to vitamin D toxicity and hypercalcemia. Hypocalcemia may lead to impaired mineralization of bone and otoconial particle degeneration.

## METHODS

A prospective study was conducted in the Department of Otorhinolaryngology in Pushpagiri Institute of Medical Sciences from June 2015-June 2018. A total number of 348 patients were included in the study. Age of the patients ranges from 40 to 82 years. 108 patients (31%) showed vitamin D deficiency.

### Inclusion criteria

All the patients were clinically diagnosed with BPPV.

### Exclusion criteria

Patient with other forms of peripheral vertigo and central vertigo, individuals who suffered traumatic head injury, individuals who underwent middle ear surgery, pregnant and lactating women, patients who received

chemotherapy or other ototoxic drugs were excluded from the study.

Dix hall pike test was positive in all cases. 75% of the patient had right sided and 25 % left sided BPPV. Epley's repositioning manoeuvres were done for all cases. Serum vitamin D level estimation was done in all cases. 108 patients were noticed to have low serum vitamin D levels and they were divided into two groups randomly (as shown in figure 2). Group A were given vitamin D supplementation and group B were taken as control. All patients in the study group, supplemented with vitamin D were divided into 3groups according to their vitamin D levels

### Group 1

Vitamin D level is below 10 ng/ml.

### Group 2

Serum vitamin D level is 11-15 ng/ml.

### Group 3

Serum vitamin D level were 16-20 ng/ml.

All the patients were followed up for one year.

Data was entered in MS-excel 2007 and data was analyzed using SPSS software Trail version 22. Results were displayed in numbers, percentages only.

## RESULTS

Vitamin D level above 20 ng/ml is taken as normal. Out of those 348 patients, 235 patients (67%) were females and 113 patients (32%) were males (Table 1 shows age and sex distribution of patients).

**Table 1: Age and sex distribution of the patients.**

Age group (in years)	Number	Male	Female
40-50	107	48	98
51-60	118	34	76
61-70	72	15	35
>71	51	16	26
Total	348	113	235
%		32	67
Vitamin D deficiency	108	35 (32%)	73 (67%)

**Table 2: Number of patients with vitamin D deficiencies.**

No. of patients	Deficient in vitamin D	No. of male patient with vitamin D deficiency	No. of female patient with vitamin D deficiency
348	108	35	73
%	31	32	67

**Table 3: Serum vitamin D levels after three months.**

	Group A	Group B	Group C
<b>Supplementation group (ng/ml)</b>	12-14	16-18	18-24
<b>Non supplementation group (ng/ml)</b>	9	9	13

**Table 4: Serum vitamin D levels after one year.**

	Group A	Group B	Group C
<b>Supplementation group (ng/ml)</b>	15-17	19-21	26-32

**Table 5: Rate of recurrence after one year.**

	No. of patients	Recurrences	%
<b>Vitamin D supplementation group</b>	27	0	Nil
<b>Vitamin D non supplementation group</b>	27	18	66

Group A, B and C of the supplementation group were given 800 IU of vitamin D and 1 gm of calcium for 3 months. After 3 months the serum vitamin D were estimated and found that the level of vitamin D increases to 10-14 ng/ml in group A, 16-18 ng/ml in group B and 18-24 ng/ml in group C. After that all the patients in the vitamin D supplementation group group A, B and C were given 400 IU of vitamin D and 1 gm of calcium for 9 months. Vitamin D level in group A is increased to 15-17 ng/ml, Group B 19-21 ng/ml and Group C 26-32 ng/ml (as shown in Table 3 and 4).

The patients in the group 1 and group 2 showed recurrences at 6th month were as patients in group 3 were symptom free for 1 year. 32 out of 54 patients in the non-supplementation groups shows recurrences and their serum levels of vitamin D increases by 2-3 ng/ml, might be by ultraviolet radiation or from dietary sources during the follow up period (as shown in Table 5).

## DISCUSSION

Vertigo is a common symptom with a life time prevalence of about 30% affecting the day today activities. Multiple systems are involved in the maintenance of balance. By proper history and clinical examination 90% of cases can be diagnosed without any laboratory investigations or imaging techniques. Management includes vestibular exercises, otoconial repositioning manoeuvres and specific pharmacotherapies. Vertigo is more common in the elderly compared to young as vestibular system degenerates with age.<sup>7</sup> Peripheral vestibular disorder is the cause for true vertigo and it is usually associated with nausea and vomiting. The vertigo aggravated with the change in head positions and lasting for few seconds is BPPV. Vertigo tends to be recurrent in the vast majority of the affected persons. Elderly people have many other co-morbidities and many vitamin deficiencies.<sup>9</sup> So correction of these co-morbidities and vitamin deficiencies can reduce the recurrence rate of BPPV dramatically. Recurrence of BPPV is defined as relapse

of vertigo even after a successful treatment, like otoconial repositioning manoeuvres.

### *Role of serum Vitamin D levels in the pathogenesis of BPPV*

The prevalence of vitamin D deficiency in our patients diagnosed with BPPV was as high as 31%. The recurrence among patients with vitamin D deficiency was 66%. Normal vitamin D level is above 20 ng/ml.

### *Classification of serum Vitamin D level*

- Deficient - <20 ng/ml
- Insufficient - 20-29 ng/ml
- Sufficient - 30-100 ng/ml
- Potential Toxicity - >100 ng/ml

The formation and degeneration of the otolith particles is highly complex and a thorough molecular and genetic knowledge is required for its analysis. They are a combination of calcium carbonate crystals and proteins in the otoconial membrane in the summit of the hair cells. Those particles that getting dislodged from otolith organs like macula and cristae and will wander in the semi-circular canals, most commonly the posterior semi-circular canal. This movement with various head positions can induce BPPV. Absent or delayed degeneration of the particles is occurring in BPPV. Calcium metabolism is involved in synthesis and absorption of otoconia and vitamin D is required for the calcium metabolism. 15 patients in our study showed hypocalcemia, however a larger study is required for its correlation.

The Jeong et al were studied 100 patients with idiopathic BPPV and 192 controls found that serum vitamin D levels as significantly lower in the BPPV patients as compared to the controls.<sup>10</sup> There are many studies which reports that vitamin D deficiency is a risk factor for recurrence in BPPV.<sup>11</sup> Belabuki et al in his study evaluate the relation between the vitamin D deficiency and BPPV

had found that vertigo improves after vitamin D supplementation.<sup>12</sup> Taalat et al in their study found that recurrence rate of BPPV following treatment for severe vitamin D deficiency and found that there is a significant decrease in the recurrence rate following improvement in serum 25 hydroxy vitamin D3.<sup>13</sup> Vitamin D plays a crucial role in the homeostasis of calcium and phosphorus. Otoconia, like bone are the result of ordered deposit of inorganic calcium carbonate (calcium phosphate in bone) crystallizes onto a preformed frame work of organic matrix.<sup>14-16</sup> The calcium for otoconia is obtained is from endolymph.

## CONCLUSION

The prevalence of vitamin D deficiency is high in the general population (31%). The present study was aimed to study the relationship between BPPV and vitamin D deficiency. We studied 108 patients who were diagnosed with BPPV and vitamin D deficiency were followed for one year and found that vitamin D deficiency is a definite co-morbidity for the development and recurrence of idiopathic BPPV. Hence patient presenting with BPPV should be evaluated for serum vitamin D levels along with otoconial repositioning manoeuvres since vitamin D plays a crucial role on the mechanism of formation and maintenance of otoconial particles in the vestibular system.

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## REFERENCES

1. Al-Asadi J, Al-Lami Q. Prevalence and risk factors of benign paroxysmal positional vertigo among patients with dizziness. *Br J Med Medical Res*. 2015;7(9):754-61.
2. De Stefano A, Dispensa F, Suarez H, Perez-Fernandez N, Manrique-Huarte R, Ban JH, et al. A multicentre observational study on the role of co-morbidities in the recurrent episodes of benign paroxysmal positional vertigo. *Auris Nasus Larynx*. 2014;14:31-6.
3. Bhattacharyya N, Gubbels SP, Schwartz SR, Edlow J, El-Kashlan H, Fife T, et al. Clinical practice guideline. Benign Paroxysmal positional vertigo (Update). *Otolaryngol Head Neck Surg*. 2017;156(3S):1-47.
4. Buki B, Ecker M, Junger H, Lundberg YW. Vitamin D deficiency and benign paroxysmal positional vertigo. *Medical Hypotheses*. 2013;80(2):201-4.
5. Halmagyi GM, Thurtell MJ, Curthoys IS. Vertigo: clinical syndromes. In: Gleeson M, ed. *Scott-Browns Otorhinolaryngology, Head and Neck Surgery*. 7ed. London, UK: Taylor and Francis Ltd; 2008: 3760-3763.
6. Jameson JL, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J. *Harrison's Principles of Internal Medicine*. United States: McGraw Hill; 2018.
7. Hotson JR, Baloh RW. Acute vestibular syndrome. *N Engl J Med*. 1998;339(10):680-5.
8. Jang Y, Hwang C, Shin J, Bae W, Kim L. Age related changes on the morphology of the otoconia. *Otolaryngoscope*. 2006;996-1001.
9. Karatas A, Yuceant GA, Yuce T, Haci C, Cebil T, Salviz M. Association of benign paroxysmal positional vertigo with osteoporosis and vitamin D deficiency. *J Int Advan Otol*. 2017;13(2):259-65.
10. Lundberg Y, Xu Y, Thiessen K, Kramer K. Mechanism of otoconia and otolith development. *Develop Dynamics*. 2014;244(3):239-53.
11. Parham K, Leonard G, Feinn R, Laferriere D, Kenny A. Prospective clinical investigation of the relationship between idiopathic benign paroxysmal positional vertigo and bone turnover. *Laryngoscope*. 2013;123(11):2834-9.
12. Picciotti PM, Lucidi D, Corso ED, Meucci D, Sergi B, Plaudetti G. Co-morbidities and recurrence of benign paroxysmal vertigo. *Int J Audiol*. 2016;55(5):279-84.
13. Schuknecht H. Cupulolithiasis: *Archives of Otolaryngology-Head and Neck Surgery*. 1969;90(6):765-78.
14. Jeong SH, Kim JS, Shin JW, Kim S, Lee H, Lee AY, et al. Decreased serum vitamin D in idiopathic benign paroxysmal position vertigo. *J Neurol*. 2012;260(3):832-8.
15. Talaat HS, Kabel AM, Khaliel LH, Abuhadied G, El-Naga HA, Talaat AS. Reduction of recurrence rate of benign paroxysmal positional vertigo by treatment of severe vitamin D deficiency. *Auris Nasus Larynx*. 2016;43(3):237-41.
16. Waringhoff JC, Bayer O, Ferrari U, Straube A. Co-morbidities of vertiginous disease. *BMC Neurol*. 2009;9:29.

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