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ORIGINAL ARTICLE

Vitamin D deficiency and benign paroxysmal positioning vertigo

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ABSTRACT

Objectives: Benign paroxysmal positioning vertigo (BPPV) has significant recurrence rates, mainly in older patients. The etiopathology of recurrent BPPV is possibly related to low serum vitamin D levels. Finding a therapeutic weapon will help with these complicated cases, reducing disability, falling risk and also health care costs.

Study design: *Clinical trial*: one-year duration.

Setting: Ten patients with diagnosis of BPPV made by history and physical examination and at least two episodes of documented BPPV in the previous two years and chronic complaints of dizziness. Neurologic and other otological diseases were excluded for these patients.

Subjects and methods: Vitamin D was evaluated by measuring serum 25-hydroxyvitamin D (25-OHD). Levels below 20 ng/mL were considered deficiency and levels between 20 and 30 ng/mL were considered insufficiency. Half of the patients (treatment group) started a treatment with cholecalciferol while the remaining patients were the control group. All of the patients were reevaluated every three months.

Results: All patients of the treatment group did not have any subsequent episode of positional vertigo, dizziness complaints or nystagmus evoked by provocative manoeuvers. At reevaluations, the mean value of serum 25-OHD for the treatment group had increased noticeably. It was also significantly higher than the mean value of control group. All patients of control group had positional vertigo episodes, as well as positional nystagmus at office reevaluations.

Conclusion: These results support the need to systematically measure and correct vitamin D levels in patients with recurrent BPPV.

Abbreviations: BPPV: benign paroxysmal positioning vertigo; 25-OHD: 25-hydroxyvitamin D

KEYWORDS
Benign paroxysmal positioning vertigo; vertigo; cholecalciferol; vitamin D

Introduction

Benign paroxysmal positioning vertigo (BPPV) is the most frequent cause of peripheral vertigo, accounting for approximately 20–30% of diagnosis in specialized dizziness clinics [1–3]. Studies have shown a cumulative incidence of 10% until 80 years [1] and significant recurrence rates, even after successful treatment manoeuvers (nearly 50% in 5 years), mainly in older people [4,5]. This risk of BPPV episodes in older people constitutes a very probable cause of falls, leading to diminished quality of life [2].

The fact that otoliths are calcium carbonate crystals [6] and have reduced density in rats with induced osteoporosis [7] raises the hypothesis that vitamin D deficiency can increase the risk of recurrence of BPPV. In a study with 32 consecutive women with BPPV, 24 had osteoporosis. The ratio of BPPV is two women for one man. Obviously, we know that lack of vitamin D is associated with osteoporosis [2,8]. Can we close this triangle [2]?

This study aims to analyse if vitamin D supplementation affects the number of BPPV recurrences in patients with previous episodes of this disease.

Methods

The authors selected 10 consecutive patients, all of them women, observed by the first author at emergency care, during the months of October and November of 2016. These must have an history of at least two episodes of BPPV, in the previous two years, requiring emergency care. They presented nystagmus at Dix-Hallpike or McClure-Pagnini manoeuvers (compatible with canalolithiasis), solved with only one Epley,

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Table 1. Results from 'treatment group'.

Patient ID	Age	Canal	Initial 25-OHD	3th month 25-OHD	6th month 25-OHD	9th month 25-OHD	12th month 25-OHD	BPPV episodes
1	66	P	10	31	28	24	29	0
2	82	P	8	47	33	40	49	0
3	76	P	11	28	33	29	34	0
4	65	P	18	30	29	27	30	0
5	66	L	23	33	35	31	34	0

P: posterior; L: lateral.

Table 2. Results from 'control group'.

Patient ID	Age	Canal	25-OHD	BPPV episodes
6	57	P	11	1
7	62	P	16	2
8	52	P	9	3
9	55	P	22	1
10	59	A	8	2

P: posterior; A: anterior.

Gufoni or Yacovino manoeuver. Other otological and neurological causes of vertigo were excluded.

Vitamin D was evaluated by measuring serum 25-hydroxyvitamin D (25-OHD) levels: levels below 20 ng/mL were considered deficiency and levels between 20 and 30 ng/mL were considered insufficiency.

The authors divided the sample into two groups, which were named 'TREATMENT GROUP' and 'CONTROL GROUP'. Stratified sampling was used according to the semicircular canal affected: eight patients presented BPPV of the posterior canal, so each group had four cases of BPPV of the posterior canal. One patient of the treatment group had BPPV of the lateral canal, while one patient of the control group had BPPV of the anterior canal.

Follow-up reevaluations were performed every three months, during one year: in the treatment group, serum 25-OHD was measured at every reevaluation. Since the control group was not treated for supplementation, no further measurements were made.

The treatment group was supplemented with cholecalciferol according to Hospital Pedro Hispano Endocrinology recommendations: eight oral drops per day if there was deficiency (ensuing 5000 IU of vitamin D per day) and only one pill per month (assuring 800 IU of vitamin D per day) if there was only insufficiency. All patients on this group had normal renal function (based on serum creatinine) and no history of nephrolithiasis. Since this strategy of supplementation is supported by Portuguese General Health Direction and by 'Vitamin D Portuguese Statement' (released by Portuguese Endocrinology and Internal Medicine Societies), this work was exempted from ethics committee approval.

Results

The authors used two-tailed Student's *t* tests (95% confidence level) for independent samples.

Both groups showed identical initial mean values of serum 25-OHD: 14.0 ng/mL in the treatment group, 13.2 ng/mL in the control group.

In treatment group, chronic dizziness complaints disappeared completely and there were no more vertigo episodes and nystagmus at Dix-Hallpike and McClure-Pagnini manoeuvers. Mean value of 25-OHD level was 33.8 ng/mL at 3rd month, 31.8 ng/mL at 6th month, 30.6 ng/mL at 9th month and 35.2 ng/mL at 12th month. These values are significantly different, comparing to the initial mean values of this group (p values equal to .002, .001, .002 and .002, respectively) and of the control group (p values equal to .001, .001, .002 and .001, respectively).

On the contrary, all of the patients on the control group still had positional vertigo episodes with needing of emergency care and chronic dizziness complaints. They also showed positional nystagmus at reevaluations.

Moreover, the mean age for treatment group (71 years-old) was significantly higher compared to the control group (57 years-old): $p = .011$).

Results are summed up on Tables 1 and 2.

Discussion

A recent study showed a negative correlation between vitamin D and otolin-1 levels. The latter is a secreted glycoprotein which is a marker of otolithic degradation [9]. Another fact favouring the role of vitamin D is the seasonal variability of median number of patients presenting episodes of BPPV: in a retrospective survey at United Kingdom, it was significantly higher in the months associated with lower serum vitamin D levels (March, April and May) than in the rest of the year [4].

Patients of both groups also presented osteoporosis and hypocalcaemia, reinforcing the possible relationship between vitamin D deficiency and recurrent BPPV. In fact, the reduced density of otoliths due to the calcium deficiency, which can be caused by vitamin D deficiency, rises the likelihood of otolithic displacement from utricle and saccule to the

semicircular canals leading to canalolithiasis [3]. Furthermore, a paper from Croatian authors showed that vitamin D levels were significantly lower in patients with canalolithiasis comparing to those with cupulolithiasis [10]. Hence, the idea that vitamin D deficiency can, ultimately, reduce the density of otoliths is reinforced, because canalolithiasis is triggered by a difference of density between otoliths and endolymph [11]. However, repeated BPPV episodes may lead to immobilization, and possibly aggravating osteoporosis/osteopenia [12]. Thus, it is already not completely clear if recurrent BPPV and osteoporosis/osteopenia share a common pathway or, simply, the latter is a consequence of the former.

All patients on the treatment group were 65 or more years-old while patients on the control group were younger. In general population, incidence of BPPV is higher among elderly (more than 65 years of age) [13]. In spite of that, none of the patients on the treatment group had episodes of BPPV, reinforcing the positive effect of vitamin D supplementation.

Our study has two main limitations. The sample had only 10 patients, because all patients were evaluated by only one otolaryngologist (the first author). On the other hand, there was no second control group with patients without recurrent BPPV, because vitamin D deficiency/insufficiency is so frequent that one would expect a mean serum value similar to the other groups. For the same reason, there also was no second control group with patients with recurrent BPPV and normal 25-OHD levels.

In conclusion, our results are one more support to investigate the possible protective role of vitamin D supplementation against recurrence of BPPV and build a powerful and relatively cheap therapeutic weapon for some complicated cases of old patients with repeated episodes. BPPV is very incident and prevalent so even an improvement in a small proportion of cases represents a large amount of patients and, therefore, an important annual cost care saving. Moreover, vitamin D can improve muscular balance and force (by calcium increasing), resulting in a lower fall risk [2,14].

Disclosure statement

No potential conflict of interest was reported by the authors.

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