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Original Article

Vitamin D Insufficiency/Deficiency in Patients with Recurrent Benign Paroxysmal Positional Vertigo

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BACKGROUND: The aim of this study is to verify if (1) there is a link between hypovitaminosis D and benign paroxysmal positional vertigo, (2) the number of benign paroxysmal positional vertigo relapses decreases after vitamin D supplementation; and (3) benign paroxysmal positional vertigo response to physical therapy improves after hypovitaminosis D correction.

METHODS: We enrolled 26 patients with benign paroxysmal positional vertigo and 24 subjects, who never suffered from vertigo, as a control group. All benign paroxysmal positional vertigo patients underwent physical therapy, once a week, until benign paroxysmal positional vertigo resolution. All participants were subjected to a dosage of serum 25(OH) vitamin D. In patients with hypovitaminosis D, we prescribed cholecalciferol. After 3 months of therapy, all patients were asked to undergo a second dosage of serum 25(OH) vitamin D. For each patient, we counted the number of maneuvers required to resolve each episode of benign paroxysmal positional vertigo before and after vitamin D supplementation.

RESULTS: At T_0, both patients and controls had insufficient average vitamin D serum levels (23.18 and 23.73 ng/ml) without significant differences between groups (p 0.16). However, the percentage of patients who had a serum vitamin D deficiency before supplementation was higher than that of the control group (65.39% and 33.3%). The latter finding was statistically significant with a $P=0.002$. Before integration 100% of patients had a recurrent BPPV (average number of recurrences/pt: 9.31) while after supplementation only 5/16 pts (31.25%) had just 1 recurrence (average number of relapses/pt 0.31, $P=0.0003$). The average number of maneuvers before and after supplementation was 1.37 and 1.0 respectively ($P=0.6543$).

CONCLUSION: Our results suggest that (1) there is a relationship between vitamin D deficiency and the onset of BPPV, (2) hypovitaminosis correction is able to reduce both the number of patients relapsing and the number of relapses per patient, and (3) we have not found a significant effect of vitamin D supplementation as regards the responsivity of benign paroxysmal positional vertigo to physical therapy.

KEYWORDS: Benign paroxysmal positional vertigo, hypovitaminosis D, recurrent benign paroxysmal positional vertigo, vitamin D deficiency, vitamin D supplementation

INTRODUCTION

In the literature, several studies are reported on the association between benign paroxysmal positional vertigo (BPPV) and osteoporosis/osteopenia and between osteoporosis and vitamin D (vD) serum levels, but few studies link BPPV and vD values.

In 2003, Vibert et al[1] suggested a possible relationship between BPPV and osteoporosis, observing that 75% of women in menopausal age and with BPPV had osteopenia/osteoporosis on dual-energy x-ray absorptiometry (DEXA) of spine and hip. In 2009, Jang et al[2] reported the same association between idiopathic BPPV and reduced bone mass density (BMD); in addition, the authors observed that patients with reduced BMD had a greater number of relapses of BPPV and required a greater number of therapeutic maneuvers.

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In 2010, Mikulec et al[3] assessed the influence of "treated osteoporosis" in 260 women with and without BPPV observing a statistically significant negative association between BPPV and "treated osteoporosis."

Moreover, in recent years, Lundberg et al[4-6] noted the common characteristics between bone and otoconia biomineralization. These structures are in fact similar both in a matrix organization and in the majority of protein components. Furthermore, otoconia biomineralization is similar to that of bone and teeth and involves a tight regulation of organic matrix formation at specific sites and mineral crystals deposition in an ordered manner. In an experimental study, Vibert et al[7] discovered some typical ultrastructural alterations of otoconia in ovariectomized osteopenic/osteoporotic female adult rats; they found both a decrease in the density of debris and an increase in their size as compared to a control group of rats. These changes were interpreted by the authors as the result, in the utricle, of the alteration of calcium metabolism caused by osteoporosis.

The above-mentioned findings suggest that a BPPV could be induced by a disorder similar to that of osteoporosis through different mechanisms; first, the reduced calcium fixation could cause defects in remodeling of the internal structure of otoconia as well as of their adhesion to the otoconial membrane; on the other hand, an increase in the concentration of free calcium in the endolymph could induce a reduced ability to dissolve the dislodged otoconia affecting the electromechanical transduction.

Besides this, we know that a correct otoconial formation requires a local increase in Ca^{2+} and carbonate concentration to begin crystallization on the proteinaceous core, while it is necessary to maintain a low Ca^{2+} concentration in the vestibular endolymph to prevent not required mineralization in the rest of the labyrinth. This critical balance is maintained by the epithelial calcium transport system expressed in the semicircular canal and cochlea.[8] Vitamin D, through its receptors in the inner ear, stimulates this system, upregulating the expression of its components.

Based on these concepts, Jeong et al[9] in 2013 hypothesized that low serum vD could be associated with the development of BPPV. In their study, they found that serum vD level was lower in patients with idiopathic BPPV compared with the control group, with an average difference of 4.5 ng/mL, and that this finding was independent of age, sex, body mass index (BMI), and decreased BMD. On the basis of their results, the authors suggested that vD deficiency could be a risk factor for BPPV. Bűki et al[10] investigated the relationship between BPPV and vD deficiency; the authors found that patients with idiopathic BPPV had low average vD serum levels (23 ng/mL). In addition, they identified 4 patients who had been having recurrent episodes of BPPV for several years before the examination; these patients had significantly lower average serum levels of vD than patients at the first episode of BPPV. Moreover, after vD supplementation, BPPV patients had not had recurrences in a follow-up period of at least 8 months. The association between BPPV and vD hypovitaminosis also aroused the interest of Talaat et al[11] in 2015. The authors compared 3 groups of subjects, the first consisting of patients with recurrent BPPV, the second of patients with BPPV at the first episode (and thus not recurrent), and the third (control group) of healthy volunteers. All subjects enrolled in the study were subjected to fasting early morning venous blood sampling to dose 25(OH) vD and to DEXA to assess bone mineral density. The data obtained showed 25(OH) vD levels significantly higher in the control group and, with respect to groups with BPPV, these values were lower in patients with recurrent BPPV.

Very recently, Jeong[12] and co-workers compared the recurrence rate of annual BPPV relapses between a group of patients treated with vD and calcium carbonate and others not treated. The authors found that intervention leads to a significant reduction of relapses per year as well as of the number of maneuvers needed to treat BPPV.

On the basis of the data reported in the literature, we conducted our study with 3 aims:

1. To verify if there is a link between serum vD levels and the onset of BPPV;
2. To verify if, in BPPV patients with low serum vD levels, the number of relapses decreases after vD supplementation;
3. To verify if, in BPPV relapsing patients, the correction of vD deficiency/insufficiency leads to a better response to physical therapy.

MAIN POINTS

- There is a link between hypovitaminosis D and the onset of benign paroxysmal positional vertigo (BPPV).
- Vitamin D deficiency is a risk factor for recurrent BPPV.
- Vitamin D supplementation reduces the number of BPPV recurrences.

MATERIALS AND METHODS

From March 2014 to November 2014, 26 patients with acute vertigo and nystagmus patterns of BPPV were enrolled as the study group, while 24 subjects served as the control group. Most BPPV patients (24 out of 26) had recurrent attacks; for these subjects, an accurate chronology of episodes was taken, including the timing of the first and subsequent attacks, the number, the frequency, and the duration of the recurrences as well as the number of maneuvers needed to resolve signs and symptoms. A "recurrence" was defined as a new BPPV episode occurring at least 2 weeks after verifying the complete resolution of the previous one. Patients excluded from the study were (1) those who declined to participate in the study, (2) whose age was under 18 years old, (3) those with a recent head trauma or oto-surgery, (4) those with an ongoing audiological or neuro-otological disease other than BPPV, (5) those with ongoing chemo and/or radiotherapy, (6) pregnant women, and (7) those treated with vD for more than 30 days.

The control group was made of patients waiting for functional surgery at the ear, nose, and throat Unit of the same Hospital (otological pathologies excluded) and who never suffered from vertigo.

After collecting a detailed history, all BPPV patients underwent a complete bedside neuro-otological examination, inclusive of the study of spontaneous/positional nystagmus, both with and without fixation (videoculoscopy or Frenzel's goggles); nystagmus was observed in the seated, supine, right and left side lateral positions,

head hanging position, and with both the Dix-Hallpike's tests. Moreover, gaze-evoked and rebound nystagmus as well as ocular motility were tested. Vestibulo-oculomotor reflex functional testings (head impulse, head shaking, and calorics) were also carried out. Similarly, auditory tests were performed in every patient.

Benign paroxysmal positional vertigo was treated with a suitable physical therapy; at the follow-up visits, 1 week apart, the therapeutic maneuvers were repeated until resolution.

During the follow-up period, BPPV patients were instructed to contact us for a check-up if having symptoms of recurrence; anyhow, they all were interviewed by telephone at the end of the follow-up period to verify the absence of further recurrences.

Body mass index[13] was calculated for subjects of both groups.

At the enrollment (T_0), all subjects (study and control group) underwent a fasting, early morning, venous blood sampling in order to dose serum levels of 25(OH) vD, parathyroid hormone, calcium, magnesium, phosphate, transaminases, and creatinine. According to the guidelines on the prevention and treatment of vD hypovitaminosis, serum levels of 25(OH) vD were considered deficient if lower than 20 ng/mL, insufficient if comprised between 20 ng/mL and 30 ng/mL, and sufficient if higher than 30 ng/mL.[14]

In case of hypovitaminosis D, controls were referred to the general practitioner for further therapy.

In study patients, supplementation with cholecalciferol (vD3) was initiated when serum levels were below the norm, and continued 3 months, as follows: (1) deficiency: 50.000 IU once a week for 2 weeks, then 25.000 IU once a week for 2 weeks, then 7.000 IU once a week as maintenance; (2) insufficiency: 7.000-8.000 IU once a week. Patients were also informed about vD-rich foods.

After 3 months' treatment (T_1), patients were contacted by phone to plan an additional blood sampling, in order to revalue serum 25(OH) vD, thus verifying the adequate hypovitaminosis correction, and serum calcium levels, to exclude hypercalcemia.

Moreover, for each patient with relapsing BPPV and vD deficiency/insufficiency, the number of maneuvers required to resolve each BPPV episode, before and after vD supplementation, were counted.

The follow-up period ranged from 4 months to 8 months; during such a period, vD intake was suggested if still needed.

According to the study protocol, written informed consent was obtained in accordance with the Declaration of Helsinki.

For statistical analysis, the averages were compared using Student's *t*-test. Chi-square test was used to compare percentages. Odds ratio (OR) was used to test the correlation between BPPV recurrences and vD hypovitaminosis. A P value $\leq .05$ indicated a statistical significance. The computer program used was Statistical Package for the Social Sciences.

RESULTS

BPPV Group

The study group included 6 males and 20 females (male: female ratio: 1 : 3.3), with an age ranging from 39 years to 79 years (average 61.85). The age of the maximum incidence was 60/70 and 70/80 for women and men, respectively (Figure 1).

The average BMI was 25.03.

Main comorbidities were hypertension (14/26, 53.85%), dyslipidemia (11/26, 42.31%), osteoporosis (6/26, 23.08%), and diabetes mellitus (3/26, 11.54%).

Fifteen out of 26 patients (57,69%) had a normal hearing; 9 subjects (34,61%) had presbycusis, 1 had bilateral sensorineural hearing loss, and 1 had unilateral sensorineural hearing loss of the contralateral ear.

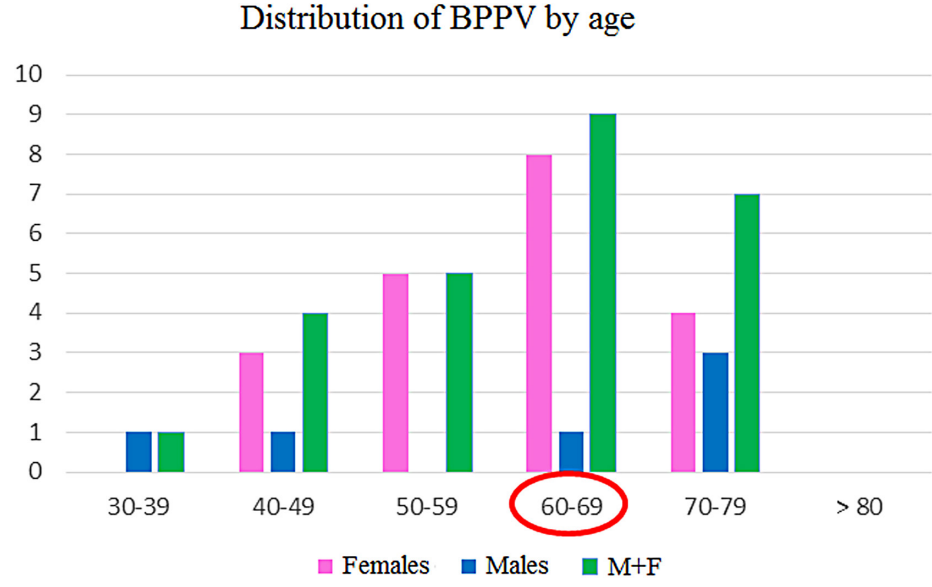

Figure 1. Distribution of BPPV by age. BPPV, benign paroxysmal positional vertigo.

Eighteen patients out of 26 (69.23%) showed a paroxysmal positional nystagmus due to the posterior semicircular canal (PSC) lithiasis, 10 of the right ear (38.46%), and 8 of the left one (30.77%). Seven subjects (26.92%) had an involvement of the lateral semicircular canal (LSC), of the right ear in 5 (19.23%), and of the left ear in 2 (7.69%). Only 1 patient (3.85%) had a bilateral BPPV involving the left PSC and right LSC.

Control Group

This group included 15 males and 9 females with an age ranging from 18 years to 76 years (average 44.17).

The average BMI in this group was 24.32.

Vitamin D Levels T_0

The average level of vD in the study group was 20.18 ng/mL, lower in men (16.17 ng/mL) than in women (21.38 ng/mL, Figure 2). Vitamin D values were deficient in 17/26 patients (65.39%), insufficient in 6/26 patients (23.08%), and in the normal range in the remaining 3 patients (11.53%, Figure 3). One of the 2 patients manifesting the first BPPV episode had normal vD levels (36.3 ng/mL) and the other one showed an insufficiency (25.6 ng/mL). Among the 24 patients with a recurrent BPPV, only 2 (8.34%) had normal vD levels, 5 (20.83%) had vD insufficiency, and 17 (70.83%) had a deficiency.

In the control group, the average vD level was 23.73 ng/mL, higher in men (25.6 ng/mL) than in women (20.6 ng/mL, Figure 2). Vitamin D values were deficient in 8 patients (33.33%), insufficient in 12 (50%), and included in the normal range in 4 cases (16.67%, Figure 3).

Vitamin D Levels T_1

All BPPV patients with deficient (17/26, 65.39%) or insufficient (6/26, 23.08%) vD levels were supplemented by cholecalciferol oral administration in a suitable dose. Three patients showing normal vD values did not receive any corrective therapy.

After 3 months' treatment, 7 patients out of the 23 contacted, refused to undergo another sampling because of personal/family/working reasons. Considering the remaining 16 patients, vD serum levels before supplementation were deficient in 13 (81.25%) and insufficient in 3 (18.75%). After supplementation, among the 13 deficient patients, 6 (46.15%) reached normal vD values and 7 (53.85%) only increased them, thus remaining below the normal range, but passing from the state of deficiency to that of insufficiency. Noteworthy, 2 of the latter patients had post-treatment values very close to the normality (29.4 ng/mL and 28.1 ng/mL).

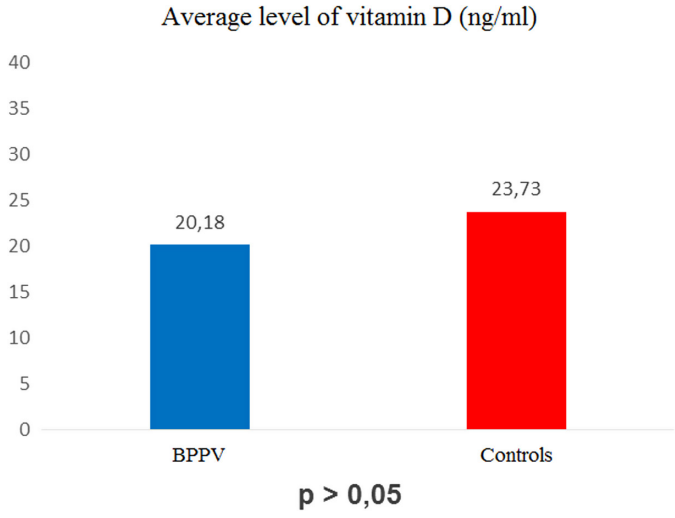

Figure 2. Average level of vD (ng/mL). vD, vitamin D.

Relapses After Treatment

At the end of the study period, among the 16 patients who received supplementation, 11 (68.75%) did not relapse; the remaining 5 (31.25%), all women and with recurrent BPPV, relapsed (Figure 4). Among latter cases, the average number of relapses was 5.70 ± 3.49 before supplementation and 1.00 ± 0 after treatment ($P = .0168$). Relapses occurred during the first weeks of treatment in 2 patients. T_1 vD levels were under the normal value in all 5 relapsing patients, except one.

Among the 11 non-relapsing patients, the average number of relapses was 10.95 ± 10.09 before supplementation and, as a definition, 0 after it ($P = .0018$).

Number of Maneuvers

In the 5 relapsing patients, the number of maneuvers required to resolve BPPV was calculated, before and after supplementation. Before vD intake, patients had 16 episodes overall, requiring 22 maneuvers, with a ratio of 1 : 37 maneuvers per episode. After supplementation, subjects developed only 5 episodes as a whole (1 each), demanding 5 maneuvers, with a ratio of 1 : 00 maneuver per episode ($P = .6543$).

DISCUSSION

The first goal of our study was to establish whether vD deficiency or insufficiency contributes to BPPV pathophysiology. The link existing between vD and osteoporosis[15] as well as between osteoporosis and BPPV[1-3] has been widely reported in the literature. Conversely, very little is known about a possible connection between vD and BPPV.

Analysis of demographic data aligns with literature,[16,17] confirming the higher incidence of BPPV in the female sex and the most frequent age of appearance of the pathology (considering both genders).[16] In our experience, controls have a lower average age (44.17 years) with respect to the study cohort (61.85 years); nevertheless, this difference does not influence vD basal levels, being such values not statistically different between the 2 groups (20.18 ± 9.26 ng/mL for BPPV group and 23.73 ± 8.42 ng/mL for control group, $P = .16$).

Also BMI is substantially comparable between BPPV patients (25.03 ± 3.98) and controls (24.32 ± 3.40; $P = .50$). Our data does not agree with those reported by others,[9] stating that patients with BPPV have an increase in BMI. The substantial overlap of BMI between the 2 groups would exclude the possibility of interference from confounding factors just as obesity, given vD lipid solubility. On the other hand, our sample is not large enough in allowing definite considerations on this issue.

Our data overlap with those of literature also regarding comorbidities of BPPV,[18] such as hypertension, dyslipidemia, and diabetes mellitus. Among comorbidities, osteoporosis has also been documented

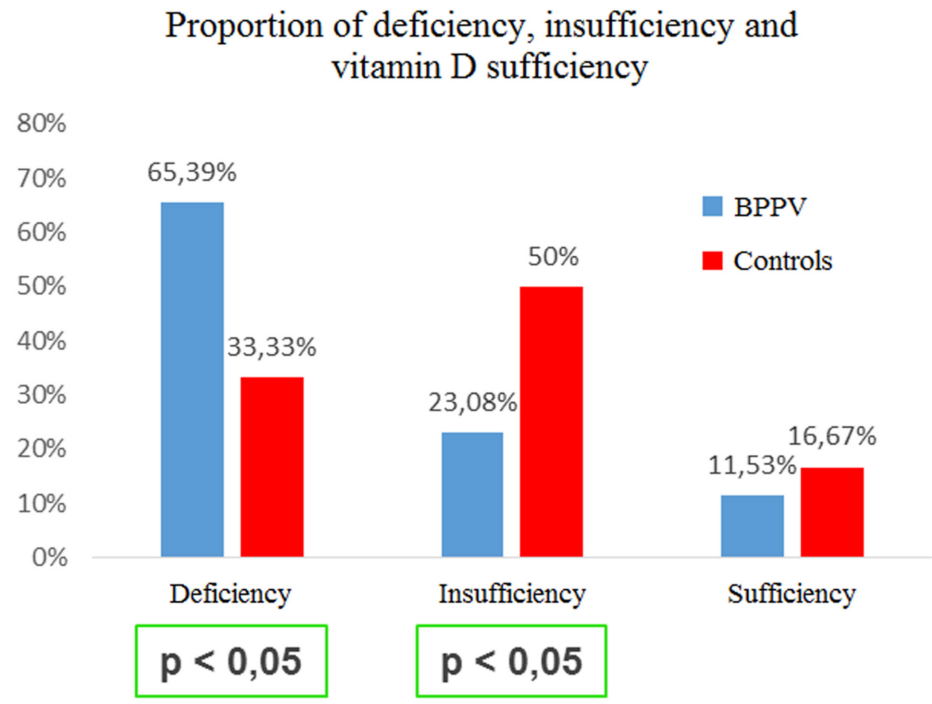

Figure 3. Proportion of deficiency, insufficiency, and vD sufficiency. vD, vitamin D.

in our BPPV patients, being diagnosed in 6/26 (23.08%) subjects, all suffering from recurrent BPPV. In our cohort, osteoporosis could be age-correlated, since BPPV patients' mean age was higher than that of controls (61.85 ± 11.61 and 44.17 ± 18.05, respectively, $P = .0001$); moreover, the association between osteoporosis and BPPV has been underlined by most authors.[3] Conversely, Yamanaka et al[19] did not find a direct correlation between BPPV onset and osteoporosis, but they reported that BPPV, once developed, relapsed more frequently in osteoporotic subjects. This finding suggests that a reduction in bone mass can be closely involved at least in the pathogenetic mechanism of BPPV recurrences.

None of the patients had a hearing loss that could theoretically be associated with BPPV; actually, the only subject showing a unilateral sensorineural hearing loss had a contralateral BPPV.

Also in our experience, the PSC and the right side were more frequently involved by BPPV; right side in 15/25 (60%), PSC in 18/25 (72%), excluding the patient with bilateral VPPB.

The majority of patients (24/26, 92.31%) had suffered from many BPPV episodes, with an average number of recurrences of 7.44 ± 8.13; only 2 of the selected patients had BPPV for the first time.

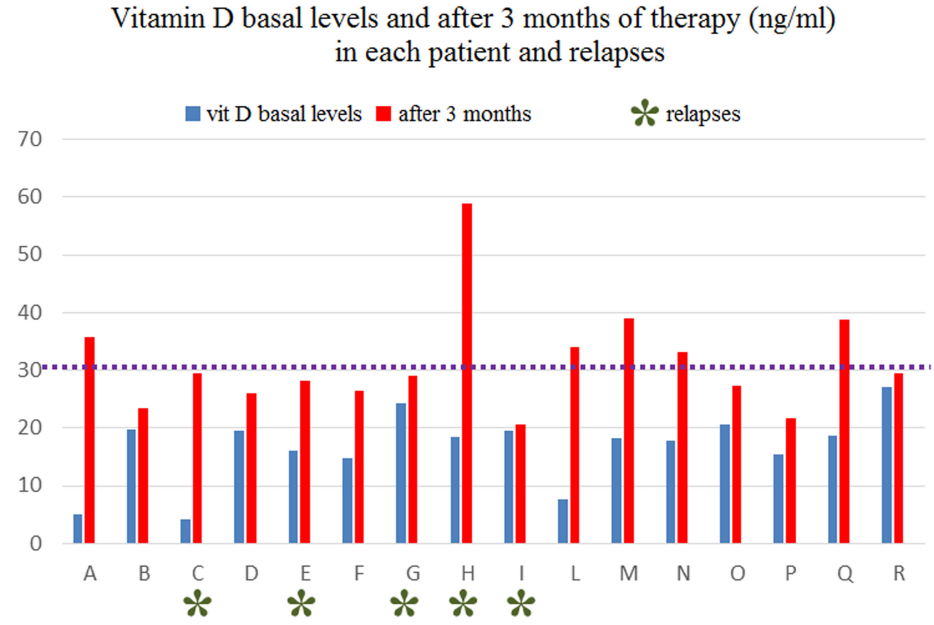

Figure 4. Vitamin D basal levels and after 3 months of therapy (ng/mL) in each patient and relapses.

T_0 average vD values in patients as well as in controls were lower than normal (20.18 ± 9.26 ng/mL and 23.73 ± 8.42 ng/mL, respectively), thus meaning that also non-BPPV subjects basically had a vD insufficiency; it could be argued that factors other than BPPV may be related to vD paucity. The latter causes may include sunlight exposure and/or diet.[13]

Vitamin D serum levels were lower in patients with respect to controls, but in our experience, this difference was not statistically significant ($P = .16$). Our data are similar to those reported by Büki et al.[10]

Given the above consideration, it would seem that vD levels do not affect BPPV onset at all. Nevertheless, in BPPV group we found a higher incidence of T_0 vD deficiency compared to that encountered in the control group (65.39% vs. 33.33%, $P = .02$); in parallel, among controls, we found a higher rate of T_0 vD insufficiency with respect to the one recorded for BPPV group (50% vs. 23.08%, $P = .04$). Rate differences were statistically significant, confirming that vD insufficiency is a common condition of the general population and probably sufficient to otoconial metabolism, namely, enough to avoid BPPV onset (OR = 0.30); on the other hand, vD deficiency could be a critical factor in detaching of otoconial debris and, for that, in promoting BPPV (OR = 3.78).

This suggests that an alteration of calcium metabolism can be crucial for the development of BPPV; therefore, a lower vD serum level can be associated with the development of labyrintolithiasis. Actually, a vD deficiency can cause the down-regulation of both calcium-binding proteins[9] and epithelial calcium transport system, normally expressed also into the membranous labyrinth[8]; this alteration would have 2 consequences: an impaired otoconial production and repairing, with a consequent detachment of otoconial debris, and an increase of free calcium concentration in the rest of the endolymph, resulting in a reduced capacity of dissolving detached otoconia.

The second purpose of our study was to verify if hypovitaminosis D correction leads to a reduced number of relapses.

Out of 23 patients presenting BPPV and vD deficiency (17 cases) or insufficiency (6 cases), and supplemented with cholecalciferol, only 16 came back for the second vD serum dosage at T_1. None of the 16 patients had hypercalcemia or some adverse effects caused by vD supplementation, neither of minor entity nor such as to discontinue therapy. The latter finding is consistent with what was reported by the literature[13]: the use of cholecalciferol is safe and manageable and its molecule maintains the proper 25(OH) vD levels for a long period.[20]

Among the supplemented patients, 13 passed from vD deficiency to normal values (6 cases) or an insufficiency condition (7 cases); 2 of the latter 7, however, had vD blood levels very close to normality (29.4 ng/mL and 28.1 ng/mL). Therefore, we obtained a correction of vD deficiency in 81.25% of cases (13/16), which was complete in 6 cases (46.15%), and partial in 7 (53.85%).

Conversely, 3 subjects, starting from vD insufficiency, even showing vD levels increased after supplementation, remained in an insufficiency condition (being 2 of them almost normal: 29 ng/mL and 29.4 ng/mL).

On the whole, 62.50% of cases (10/16) had vD values normal or almost normal after supplementation. Therefore, vD supplementation was effective in correcting the deficiency state.

In patients whose vD levels were not completely corrected, it could be hypothesized that they were poorly compliant with therapy, adjusting it according to their own, or that they followed the indication of their attending physicians, who set vD dosage as used for other pathological conditions; moreover, a poor absorption and a deficient diet as well as a little sun exposure could be taken into account.

All the 16 patients supplemented had a recurrent BPPV before treatment, with an average number of recurrences per patient of 9.31 ± 8.80. After vD supplementation, only 5/16 patients (31.25%) had 1 relapse each (average recurrence per patient: 0.31 ± 0.48); 3 out of these 5 patients suffered from osteoporosis. Considering only the group of the 5 relapsing patients, they initially had an average number of recurrences per patient of 5.70 ± 3.49, that decreased to 1.00 ± 0 after treatment ($P = .0168$).

Based on the latter data, it is evident that hypovitaminosis correction reduces both the number of relapsing patients and the number of relapses per patient ($P = .0003$), in accordance with data reported by Rhim,[21] who identified low vD concentrations as a factor that affects recurrence of BPPV, independent of age, gender, follow-up period, and type of BPPV.

Even if we sum to the 16 supplemented (and controlled) patients and the 7 who dropped out of the study, the percentage of relapsing patients after treatment decreases from 95.65% (22/23 patients) to 21.74% (5/23 patients). Similarly, the average number of recurrences per patient decreases from 8.48 ± 8.42 to 0.23 ± 0.43; $P = 0$).

To give validity to our observations, we had to ensure that patients did not relapse because of therapy and not of a limited follow-up.

In order to verify this assumption, we first calculated the average number of recurrences per month occurring in the 16 controlled patients, before and after vD supplementation; this value was 0.24 ± 0.24 before and 0.04 ± 0.07 after therapy, showing a statistically significant difference ($P = .0042$).

Afterward, we counted the number of relapses expected over the available follow-up, considering the frequency of pre-treatment recurrences (Figure 5).

Specifically, for each patient, we first calculated the mean number of actual relapses in 1 month before vD supplementation (MNAR pre-vD), knowing how many months the patient had suffered from BPPV, according to the following:

MNAR pre-Vd = number of actual relapses/months of disease suffering

Subsequently, for each patient, we counted the mean number of expected relapses in the number of months available for the follow-up after vD supplementation (MNER post-vD), according to the following:

MNAR pre-vD : 1 month = MNER post-vD: follow-up months

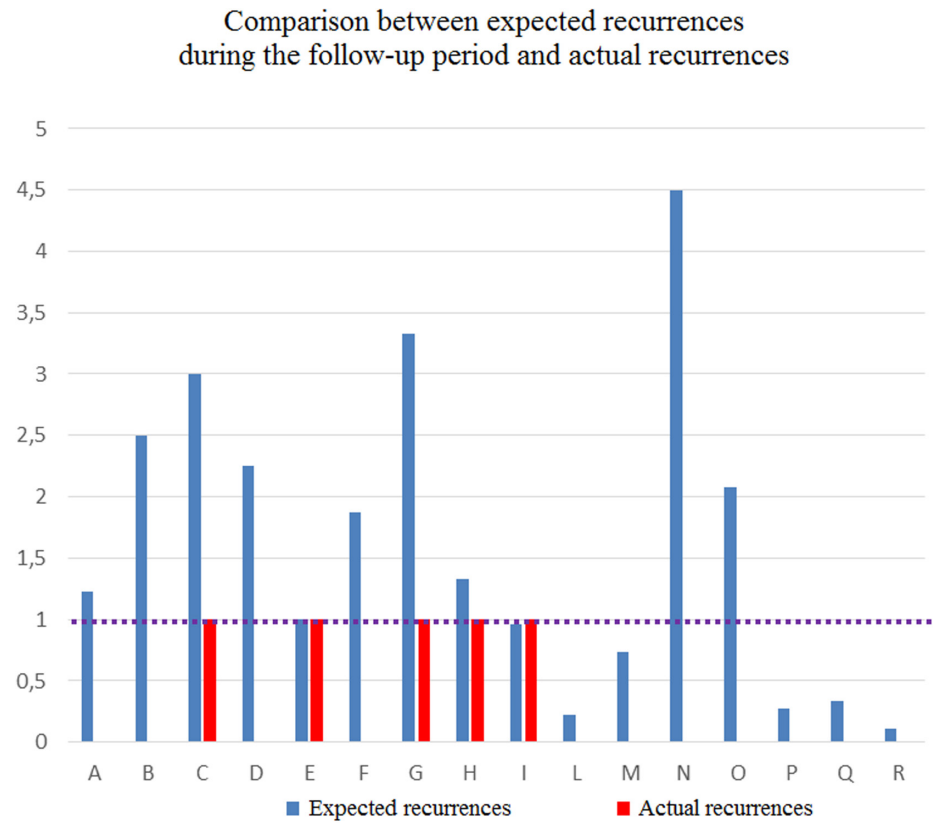

Figure 5. Comparison between expected recurrences during the follow-up period and actual recurrences.

that is,

MNER post-vD = MNAR pre-vD × follow-up months/1 month

From the analysis of these data, it emerges that the follow-up period was long enough to expect at least 1 relapse in 6 cases that actually did not recur; conversely, for the remaining 5 cases, the follow-up period was too short to have the certainty that the patient would not have had recurrences. On the other hand, if we consider the 5 subjects who had relapses, the number of expected recurrences was greater than the unique actually occurring in 3. Therefore, we observed that patients treated with vD did not have relapses or have a lesser number of recurrences than those expected in 56.25% of cases (9/16).

As regard BPPV relapses in patients with the highest levels of vD, that is 2 cases before and 1 case after cholecalciferol supplementation, we can assume that hypovitaminosis D may not be the only cause of otoconial detachment in all 3 of these patients; specifically, as for the 1 relapsing patient with the highest value of vD after cholecalciferol supplementation, we can hypothesize that it takes time for vD metabolism to be corrected, and likewise for otoconial metabolism, provided that high vD values are maintained.

The third objective of our work was to verify if hypovitaminosis correction facilitates the resolution of BPPV by reducing the number of the needed maneuvers. Considering the 5 patients who relapsed after supplementation, we found no significant difference in the mean number of maneuvers required to resolve episodes occurring before (1.37) and after (1.00) vD intake ($P = .6543$). The above data would indicate that vD correction does not influence BPPV refractoriness but the sample is not large enough to make definitive considerations.

Given our results, in the management of BPPV we suggest a diagnostic/therapeutic flow chart (Figure 6). At the time of the diagnosis, in addition to properly treating the pathology with a physical therapy, we believe a useful etiological approach as well, supplementing vD.

If the patient is on the first BPPV episode, vD serum levels determination should be performed only in selected cases that, based on age, comorbidities, and lifestyle customs, may suggest a lack of vD.

If the patient is a relapsing one, the dosage should be required anyhow.

Once the basal value is detected, cholecalciferol supplementation should be initiated as indicated.

Proposed cholecalciferol doses have proven both safe with regard to adverse side effects and effective in BPPV treatment.

By concluding, our data confirm the tight relationship between vD deficiency and BPPV development; conversely, vD insufficiency would seem a very common condition also in the general population.

Correcting vD levels by means of supplementation reduces both the number of relapsing patients and the average number of recurrences per patient. The same conclusion cannot be drawn in regard to the

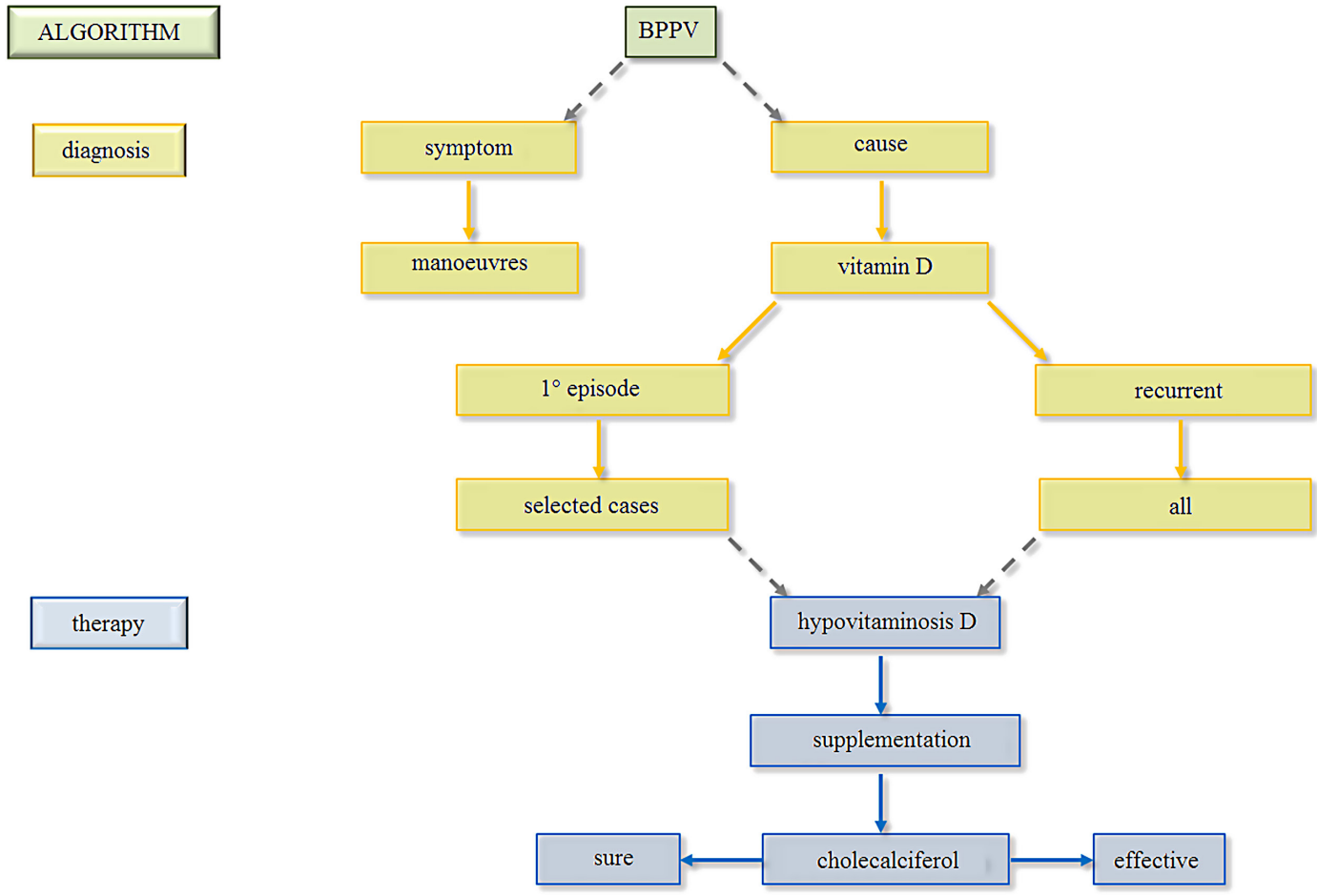

Figure 6. Proposed algorithm.

effect of vD supplementation on BPPV responsiveness to physical therapy: in our sample it was irrelevant. On the other hand, the latter finding should be investigated in a larger sample of subjects.

Benign paroxysmal positional vertigo is a very common condition in the overall population but in the elderly, this pathology can be dangerous being a potential cause of falling. Moreover, due to the very low vD serum levels, elderly subjects often suffer also from osteoporosis/osteopenia; such a condition increases the risk of fracture in case of fall.

At present, ours is an observational survey on a relatively small sample and a larger sample study could add more information and validate our findings; moreover, patients were studied along their own follow-up, without comparing the group with another one matched and not supplemented. In the future, such a comparison should be made in order to strengthen results.

Ethics Committee Approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent: Informed consent was obtained from all individual participants included in the study.

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