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Review Article

Role of vitamin D in controlling vertigo: a review

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ABSTRACT

Vertigo is a disabling symptom that has a high recurrence rate and interferes with day-to-day functioning. Vertigo is frequently caused by Meniere's diseases and benign paroxysmal positional vertigo (BPPV). The most prevalent neuro-ontological condition causing vertigo is BPPV. Vertigo attacks that last only a few seconds and are brought on by changing the head's position with respect to gravity are the hallmark of BPPV. There is a suggestion that vitamin D deficiencies and BPPV and Meniere's diseases are positively correlated. However, there is not much awareness among clinicians about association between vitamin D deficiency and certain etiologies of vertigo. After taking vitamin D supplements, vertigo attacks stop happening. Supplementing with vitamin D may be beneficial for patients with low vitamin D levels who have BPPV and Meniere's disease. This review's goal is to discuss about how vitamin D helps to treat vertigo, especially BPPV and Meniere's disease.

Keywords: Vitamin D, Vertigo, Benign paroxysmal positional vertigo, Meniere's disease

INTRODUCTION

Vertigo is a perceived movement of one's own body, such as spinning or swaying or rotation of the surrounding, or both, in the absence of physical movement.[1] The causes of vertigo are diverse and often stem from dysfunction within the peripheral or central vestibular systems. Vertigo typically arises from various vestibular disorders such as BPPV, Meniere's disease, and other conditions originating from the peripheral vestibular system.[2] Vertigo is one of the commonest clinical conditions with which patients attend to clinicians and its incidence increases with age.[3] The mechanism of BPPV is well established as free-floating otolith debris (canalolithiasis) or debris attached to the cupula (cupulolithiasis).[4] The impact of vitamin D deficiency on musculoskeletal health, and association of low vitamin D with malignancy, cardiovascular disease, diabetes mellitus and autoimmune disorders have been well established.[5] Currently, study showing the relation of hypovitaminosis of vitamin-D with generation vertigo due to BPPV and Meniere's disease.[6] This review article aims to explore the association of vitamin D deficiency and vertigo.

METHODS OF LITERATURE SEARCH

Research articles investigating the role of vitamin D in controlling vertigo were sought through various methods. Initially, an online search was conducted across databases including Scopus, PubMed, Medline, and Google Scholar. The search strategy was designed according to PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analysis) criteria to ensure systematic and comprehensive coverage of relevant literature. In addition to manually sourcing research publications from citations, our search methodology involved screening the abstracts of published works. Eligibility criteria encompassed randomized controlled trials, observational studies, comparative studies, case series, and case reports that provided sufficient assessment of the role of vitamin D in

controlling vertigo. Total 41 papers were included such as 14 case reports, 12 case series, and 15 research articles (Figure 1). The burden of vertigo, vitamin D, otoconia, effect of vitamin D on vertigo, benign paroxysmal positional vertigo, and Meniere's diseases, and the role of vitamin D in controlling vertigo are covered in this article. This review paper provides a starting point from which future prospective trials can be developed, and it could serve as a catalyst for more investigation into this clinical syndrome known as role of vitamin D in controlling vertigo, for which there are currently very few studies available.

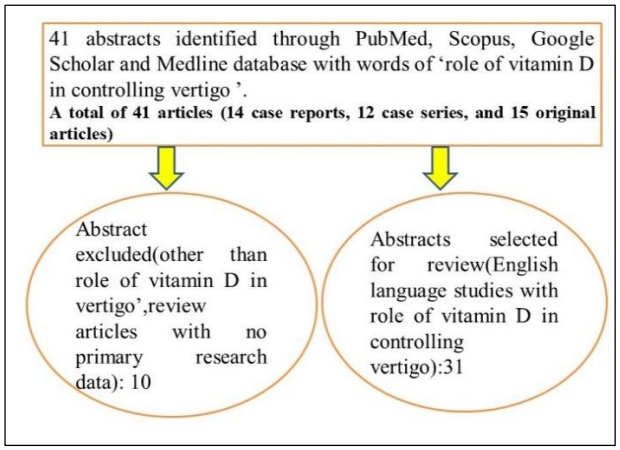

Figure 1: Method of literature search.

BURDEN OF VERTIGO

Although dizziness and vertigo are estimated to affect 20-30% of the population over a lifetime, with a 1-year prevalence estimate for vertigo at 4.9%, the healthcare burden of vertigo remains largely underestimated.[7] This is primarily due to the unpredictable nature of vertigo attacks and the complexity of the condition.[8] Although BPPV is named as a benign disorder, many patients of BPPV face recurrences and these patients show a one-year recurrence rates of around 50%.[9] Some patients of BPPV experience severe difficulties in their routine life because of frequent recurrences. BPPV recurs frequently in 15 to 56% cases.[10] Although BPPV is a benign disorder, approximately 86% of cases of BPPV suffer from interruptions in daily activities and loss of days at working place.[10] So, the direct and indirect expenses for BPPV are significant.

The expense was estimated at about $2,000 for getting the diagnosis and treatment of BPPV in USA.[11] It was also documented that costs of healthcare for diagnosis of BPPV alone around $ 2 billion per year in USA.[12] Currently, there are no established methods to prevent further attacks of vertigo in this highly recurrent disorder. Posterior canal BPPV is the most commonly encountered type of BPPV and it is responsible for 90% of all BPPVs.[13] Lateral canal BPPV is another type of BPPV which accounts for around 10% of all BPPVs.[13] Anterior canal BPPV is very uncommon type of BPPV. Vitamin D levels are significantly lower in BPPV than in the control groups.[14] Female in the BPPV and control group have lower vitamin levels than male, and the sunlight exposure is less in female than male due to their traditional dressing style. 15,16 Although BPPV is considered as a benign disease, several patients of BPPV suffer recurrences. BPPV patients show one-year recurrence rates of approximately 20%- and five-year recurrence rates of approximately 50%. 17

VITAMIN D

Vitamin D is usually formed in the skin and is converted into 25-hydroxyvitamin D in the liver and into 1,25-dihydroxy vitamin D in the kidney to act on different parts of the human body.[15] Serum calcium and serum vitamin D3 levels play an important role for improving the bone mineral density and decrease the chance of bone fractures. Low levels of serum vitamin D3 are associated with low bone mass.[16] Vitamin D3 and serum calcium in the body play an important role for enhancing the bone mineral density and decrease the chance of bone fractures.[14] Low serum vitamin D3 are associated with low bone mass. Vitamin D3 supplementation reduces the chance of bone fractures in elders. Vitamin D receptor deficiency is associated with balance impairment in mice. Similarly, Deficiency of vitamin D may predispose human being for disturbance of balance or posture controls. Vitamin D is needed for absorption of calcium and bone hemostasis, so there is an association between biomarkers of bone turn over and benign paroxysmal positional vertigo (BPPV).[16]

OTOCONIA AND BONE

The otoconia consists of calcium carbonate as calcite crystals and an organic core consisting mainly of glycoproteins. Currently, one report showed higher prevalence of osteopenia/osteoporosis among male and female with idiopathic BPPV than in controls.[15] The saccule joins to the cochlear endolymphatic duct through the ductus reuniens. Studies have shown that the natural calcium levels in the cochlear endolymph are exceedingly low, at 20 µM. Elevated calcium concentrations have been observed to impede the mechanoelectrical transduction process in the stereocilia of cochlear hair cells.[18] This explains why the dissolution of dislodged saccular otoconia in the endolymph, resulting in increased calcium concentration, leads to sudden hearing loss and vertigo.[19]

The utricle and saccule serve as gravity receptors housing otoconia, which are calcium carbonate ($CaCO_3$) bio-crystals embedded in proteins. Otoconia, along with their surrounding membranous structure called the otoconial membrane, are situated above the kinocilia and stereocilia of hair cells within the macula, the sensory epithelium of the otolithic organs.[19] Otoconia related balance diseases are prevalent among people. Abnormalities in otoconia can have several causes such as genetic mutation, head injury, and ototoxic drugs. There are common features between the bone and otoconia biomineralization. The organization of the matrix is similar between the bone and otoconia, and

most of the protein constituents are seen in both tissues. The crystals of otoconia are formed by active calcium metabolic process of the vestibular component of the inner ear. Similar to bone and teeth, biomineralization in otoconia involves compact regulation of the organic matrix formation at specific locations and deposition of mineral crystallites in an ordered manner.[20] In osteoporosis, this process becomes impaired.[21] It is widely recognized that BPPV occurs due to the displacement of otoconia, which detach from the utricular macula and migrate into the semicircular canals, rendering them sensitive to gravity. Interestingly, a positive therapeutic effect has been observed in both BPPV and osteoporosis patients, particularly females, when treated with bisphosphonates.[22]

EFFECT OF VITAMIN D

One study suggested a link between BPPV, osteoporosis and osteopenia.[21] The role of vitamin D is already established in the literature.[23] Vitamin D and calcium play an important role for enhancing the mineral density and decrease the risk of fractures.[23] It is also known that supplementation of vitamin D reduces the chances of falls and fractures in old age persons.[24] These may increase the balance of a person with vitamin D by improving the neuromuscular function. The calcium channel proteins associated with vitamin D in the vestibular epithelium play a crucial role in calcium metabolism within the vestibular apparatus. The established interaction between calcium-related diseases and BPPV underscores the importance of understanding how disruptions in calcium metabolism can contribute to the development or recurrence of BPPV.

BENIGN PAROXYSMAL POSITIONAL VERTIGO

BPPV stands as the most prevalent peripheral vestibular disorder, marked by brief episodes of rotational vertigo triggered by specific head movements. These episodes typically endure only for a few seconds.[25] The majority of BPPV cases are considered idiopathic, where the cause is unknown. Secondary BPPV, which is attributable to other underlying conditions, constitutes approximately 10% of cases.[26]

BPPV can stem from either canalithiasis or cupulolithiasis and may affect any of the three semicircular canals. However, the posterior semicircular canal is most commonly affected in BPPV cases. This condition arises when otoconia become dislodged from the macula of the utricle or saccule. These displaced otoconia can then enter the semicircular canal or adhere to the cupula. Comprising calcium carbonate ($CaCO_3$) and glycoprotein crystals, otoconia are connected to hair cells through protein fibers.[27] The central nucleus of otoconia crystals primarily consists of organic glycoproteins with relatively low levels of calcium. A recent study revealed a higher prevalence of vitamin D deficiency or insufficiency in patients with BPPV compared to individuals without the condition. This association suggests a potential link between reduced bone mineral density and BPPV.[28] These crystals are covered by inorganic peripheral areas with minerals that contains mainly CaCO3 with high calcium levels.[29] One study demonstrated lower levels of vitamin D than control groups, and some cases have shown severe deficiency of vitamin D in patients who chronically suffer from BPPV recurrence.[30] Another study compared 14 patients of BPPV who had no relapse of vertigo with four patients of BPPV who had a relapse pf vertigo.[31] In patients experiencing a relapse of BPPV, the documented serum vitamin D level was 14 ng/ml, notably lower than the serum vitamin D level of 27 ng/ml observed in patients who did not experience a relapse of BPPV. Following an 8-month period of vitamin D supplementation, there was no evidence of BPPV recurrence. Additionally, a study conducted by Talaat et al. demonstrated that treating severe vitamin D deficiency resulted in an improved recurrence rate of BPPV among patients with low serum vitamin D levels, through oral supplementation of vitamin D.[32]

MENIERE'S DISEASES

Meniere's disease is a persistent clinical condition marked by recurring bouts of vertigo lasting from minutes to hours, along with varying degrees of sensorineural hearing loss, ringing in the ears (tinnitus), and a sensation of fullness in the affected ear.[33] Numerous factors have been suggested to contribute to the development of endolymphatic hydrops. These factors encompass excessive production of endolymph, decreased absorption of endolymph by the endolymphatic sac, imbalance in ionic concentrations, genetic anomalies, viral infections, dysfunction of the autonomic nervous system, dietary influences, autoimmune responses, disturbances in vascular function, allergic reactions, and various other potential contributors.[34] Meniere's disease is recognized for its unpredictable clinical trajectory, with many patients experiencing spontaneous improvements over time. In cases where the disease exhibits fluctuating symptoms, supplementation with vitamin D has been found beneficial, particularly for patients with a deficiency in vitamin D.[35]

Before considering intratympanic gentamicin therapy, which is typically reserved for patients with severe symptoms, supplementation with vitamin D was administered. There appears to be a potential connection between vitamin D levels and otolin-1, a protein relevant to inner ear function. Additionally, vitamin D plays a role in regulating the expression of pro-inflammatory mediators such as cyclooxygenase and 5-lipoxygenase, as well as interfering with transcription factors like NF-kappaB and MAP kinase, which are involved in inflammatory responses.[36] In Meniere's disease NF-kappaB is an important factor for mediating the inflammatory responses in MD.[36] Elevated levels of otolin-1, comparable to those found in patients with benign paroxysmal positional vertigo (BPPV), have been noted in the blood of individuals diagnosed with Meniere's disease. This observation aligns with the possibility of an otolithic

hypothesis for Meniere's disease, which could be exacerbated by a deficiency in vitamin D.[37] Thus, a deficiency in vitamin D might exacerbate the detachment of otoconia, and the presence of loose saccular otoconia could contribute to the manifestation of Meniere's disease. However, it's worth noting that the blockage of the ductus reuniens, believed to be a potential factor in Meniere's disease, might not solely account for the condition. Research indicates that the size of the ductus reuniens is likely larger than what would be obstructed by otoconia ranging in length between 10-20 microns (µm). Additionally, studies have revealed that the blockage observed in the affected ear of individuals with Meniere's disease does not significantly differ from that observed in a normal ear.[38]

The blockage or collapse of the ductus reuniens can indeed lead to cochlear hydrops. Trauma to the ductus reuniens provides a plausible explanation for the frequent occurrence of cochlear hydrops and collapse of the saccule observed after cochlear implantation procedures.[39] Vitamin D deficiency is believed to exacerbate symptoms among patients with Meniere's disease. Meniere's diseases have a fluctuant clinical course and many patients improve spontaneously. So, supplementation of vitamin D in newly diagnosed patients of Meniere's disease may be helpful to reduce the clinical morbidity. The supplementation of Vitamin D consists of daily 8000IU cholecalciferol for two weeks, and daily 4000 IU cholecalciferol for next two weeks, then a weekly dose of 8000 IU.[40]

CONCLUSION

The clinical condition of forming abnormal otoconia as result of deranged calcium metabolism may lead to certain inner ear diseases that result in vertigo. Vitamin D is very effective for controlling vertigo of patients with BPPV and Meniere's diseases. Vitamin D is helpful to prevent recurrence of vertigo episodes in patients independent of age, gender among BPPV. Vitamin D deficiency may be considered as a risk factor for BPPV and Meniere's disease. Vitamin D prophylaxis may be helpful to prevent the disabling vertigo. More number of large randomized control trials or large case control studies are required to strongly establish the relationship between deficiency of vitamin D and vertigo.

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