ORIGINAL RESEARCH

The Sunshine Vitamin: An Orthodontic Perspective

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Abstract

Background: Vitamin-D insufficiency is almost universally prevalent, with south Indians having the highest prevalence of the condition. Based on the normal range, the deficiency in literature is categorized as vitamin-D sufficiency, insufficiency, and deficiency. Worldwide, it is believed that one billion people of all ages and ethnicities are vitamin D deficient (VDD). The main factors limiting exposure to sunshine, which is necessary for ultraviolet-B (UVB)-induced vitamin-D synthesis in the skin, are lifestyle (such as a drop in outdoor activities) and environmental (such as air pollution) factors. It is very important for public health that vitamin-D insufficiency is not widespread because it is so common and is a standalone risk factor for overall mortality in the general population, vitamin-D insufficiency is a significant public health issue. The significance of this hormone for general health and its application in orthodontic treatment is at the forefront of the study as the number of patients with VDD keeps rising. The prevalence of VDD varies by age-group. As few foods really contain vitamin D, standards call for daily consumption and an acceptable upper limit. Serum 25-hydroxyvitamin-D-level measurement is often advised as a preliminary diagnostic procedure for people receiving orthodontic care. Deficient patients are encouraged to obtain either vitamin-D2 or vitamin-D3 therapy with the doctor's clearance prior to starting orthodontic treatment.

Keywords: Orthodontics, Rate of orthodontic tooth movement, Vitamin D, Vitamin-D deficiency.

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Introduction

Orthodontics has been in the limelight in the field of dentistry for its promise of addressing the esthetic concern of patients.[1] With esthetics in play, one of the most important factors during fixed orthodontic therapy is extraction space closure. A delay in space closure might deliberately discourage the patient from continuing the therapy and might increase the treatment duration. Addressing this concern, literature provides numerous data on accelerating tooth movement.[2] Vitamin D, the oldest hormone, functions by maintaining serum calcium and phosphorus concentrations, thereby regulating bone mineralization. 25-OHD (25 hydroxyvitamin D) and 1,25-OHD are the two primary forms of vitamin D. Vitamin D is delivered to the liver after absorption as a pre-vitamin to be hydroxylated into 25-OHD form, which is thought to be the major circulating form of the vitamin with a half-life of 2–4 weeks.[1,2] 25-OHD is converted by the body to 1,25-OHD having two hydroxylation sites. Although 1,25-OHD, the active hormonal form of vitamin D, has a half-life of only 4–6 hours, it is not a reliable measure of the amount of vitamin D in the blood. In orthodontics, bone remodeling follows orthodontic force. The application involves resorptive and depository phases at the level of the alveolar process. This happens through the production of type-I collagen, alkaline phosphatase, and osteocalcin. A positive correlation is already evident between vitamin-D receptor polymorphisms and bone metabolism.[3] Though several shreds of evidence are provided as animal and clinical studies in the field of accelerating orthodontics, little is known regarding the relationship between the actual level of vitamin D in systemic circulation and the speed of orthodontic tooth movement.[3] Therefore, additional research, including clinical trials, is needed to better understand the rate of orthodontic tooth movement and the impact of serum vitamin D3.

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Vitamin D

Vitamin D, the oldest hormone, and the sunshine vitamin comes in two forms: vitamin D2 and D3, wherein the former is attained from the irradiated incentive that is naturally available in mushrooms, while the latter is attained from UVB light directly from sun exposure striking the skin, leading to the formation of natural vitamin D evident in human beings. Vitamin-D-25 hydroxylase first converts the physiologically inactive form of vitamin D to 25-hydroxyvitamin D in the liver.[3,4]

1,25(OH)2D increases calcium absorption from the gut. The absorption of calcium and phosphorus is boosted by enough vitamin D by 30–40% and 80%, respectively.[4]

Vitamin-D Deficiency: Prevalence

Vitamin-D deficiency should historically be defined as a 25(OH)D of less than 0.8 IU, according to a recent Institute of Medicine (IOM) recommendation. A 25(OH)D level of 21–29 ng/mL has been deemed to indicate vitamin-D deficiency.[1,5] Anywhere in the world, there is a high risk of VDD and insufficiency in children, young

adults, and middle-aged adults. Australia, the Middle East, India, Africa, and South America all have high rates of VDD.[1,4,5] A prenatal vitamin, a calcium supplement containing vitamin D, increases the incidence of VDD in pregnant women.

Why does vitamin-D deficiency occur? The main source of vitamin D for both adults and toddlers is exposure to the sun. Thus, inadequate sun exposure is a major cause of VDD. Vitamin-D synthesis in the skin is decreased by more than 95% while wearing sunscreen with a UV protection factor of 30. Someone with naturally dark skin must spend three to five times as much time in the sun as someone with a white complexion in order to create the same amount of vitamin D.[6,7]

Obesity is linked to VDD, and there is an inverse relationship between serum 25(OH)D and a body mass index (BMI) of more than 30 kg/m^2. Patients with nephrotic syndrome excrete 25(OH) D bound to the vitamin-D-binding protein in their urine. The fat-soluble vitamin D is often not absorbed by overweight people or those with one of the fat malabsorption syndromes.[8] Those who are taking a variety of medications are at risk because anticonvulsants and drugs used to treat AIDS and HIV speed up the breakdown of 25(OH)D and 1,25(OH)2D. Those who have primary hyperparathyroidism, certain lymphomas, and persistent granuloma-forming illnesses like sarcoidosis, tuberculosis, and chronic fungal infections are also at significant risk of developing VDD.[7,8] A lack of vitamin D has negative effects. Vitamin-D deficiency causes problems in the metabolism of calcium, phosphorus, and bone. PTH levels rise as a result of VDD's reduced ability to absorb dietary calcium and phosphorus.[4] The causes of osteopenia and osteoporosis include the general loss in bone mineral density (BMD) and the localized foci of bone thinning brought on by the PTH-mediated increase in osteoclastic activity. An insufficient calcium–phosphorus supplement causes a flaw in the way bones mineralize.[4,8] This deficiency causes a number of skeletal abnormalities that are typically referred to as rickets in young children whose skeletons contain little minerals. The elderly, who have deteriorating balance and more frequent falls, are more at risk for fractures due to VDD, which also results in muscle weakness. Children that are affected have difficulty standing and walking.[6,8]

Vitamin D and Orthodontics

At the alveolar process, bone remodeling caused by orthodontic forces involves the resorptive and bone-formation phases. Periodontitis, bone metabolism, and vitamin-D receptor polymorphisms have all been linked.[7] Calcium and phosphorus levels are regulated by calcitonin, vitamin D, and parathyroid hormone, according to research.[2] In some investigations, vitamin D induced osteoclast development from its progenitors and elevated osteoclast activity, which induced bone resorption.[8]

Boyce and Weisbrode performed one of the first attempts when they looked at how rat's bone development was altered by calcium-rich diets and vitamin-D metabolite injections. On day 1, treated rats had more osteoclasts than untreated animals. The researchers noticed a decline in osteoclast activity on days 3 and 4. Days 6, 8, and 10 saw the continuation of this sequela. In the meantime, over the same trial period, treated rats had much more osteoblasts than control rats did. The levels of calcium and phosphorus increased as expected. Weisbrode and Boyce found that the experimental group's bone production had increased overall. By increasing the number of osteoclasts and subsequently the rate of bone resorption, Collins and Sinclair showed that intraligamentary injections of vitamin D metabolites improve tooth movement during canine retraction. Kale et al. in 2004 evaluated the effects of prostaglandin and 1,25-dihydroxycholecalciferol (1,25-DHCC) therapy on tooth movement. When compared with controls, both were shown to dramatically increase the amount of tooth movement. On the pressure side, Howship lacunae and capillaries were dense in the experimental group. Also, when 1,25-DHCC was administered as opposed to prostaglandin administration, there were more osteoblasts on the alveolar bone's exterior surface. Consequently, the scientists described how 1,25-DHCC controls the processes of bone deposition and resorption to facilitate tooth mobility.[9]

Researchers have proposed that localized vitamin-D therapy enhances tooth position stability in addition to accelerating tooth movement. By boosting osteoblastic activity, calcitriol, according to Kawakami and Takano-Yamamoto's hypothesis, may enhance bone production and periodontal tissue remodeling, which would increase the stability of the teeth's position after orthodontic movement. Sixteen Wistar rats were used as the study's sample, and the investigators separated the animals into experimental and control groups. Bilateral orthodontic elastics were placed around the upper molars in the experimental group. The right-side upper teeth were palatally and locally injected with calcitriol every 3 days. Orthodontic elastic was not used; however, calcitriol was locally administered in the control group.

The rats that experienced tooth movement were given an orthodontic force and calcitriol injections in the submucosal palatal area, the researchers observed an increase in the mineral appositional rate on the alveolar bone. On day 7, there were noticeably more osteoblasts and osteoclasts at the mesial side of the interradicular septum, and only osteoblasts increased on day 14. By doing this, they demonstrated the powerful influence calcitriol had on bone growth.

The authors got to the conclusion that calcitriol administration may make it easier to restore tissue-supporting teeth after receiving orthodontic treatment. After 2 days of calcitriol treatment, Boyce and Weisbrode observed a brief rise in bone resorption, This, after 14 days, was followed by an ongoing rise in bone production.[9] According to this laboratory research, patients under orthodontic treatment who lack vitamin D may have delayed tooth repositioning.[2,9] Osteoclastic activity increased significantly at first, then osteoblastic activity followed.

According to these studies, orthodontic treatment could be made simpler by vitamin D and its metabolites. Further research is required to establish the appropriate amount and area of administration for vitamin-D therapy in orthodontic patients, as well as its safety.[2,9,10]

Conclusion

For optimal bone hemostasis, 3000–5000 IU of vitamin D daily is required. Throughout the day, the body ought to keep its vitamin-D levels at or above 30 ng/mL. Sun exposure and consumption of comparable health-promoting items, such as adipose fish, eggs, and fortified meals, can meet this need. Muscle compression, whim-wham conduction, and normal bone mineralization will all suffer from vitamin-D deficiency's negative consequences. Many laboratory-based tests have shown that a lack of vitamin D may slow down the movement of teeth in orthodontics. More investigation is required to establish the vitamin-D therapy safety,

as well as the ideal dosage and administration site in orthodontic patients. Researchers also need to investigate the therapeutic applications of these results, such as the implicit use of vitamin-D metabolites to speed up tooth movement during orthodontic treatment, considering the widespread prevalence of vitamin D insufficiency.

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