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RESEARCH ARTICLE

IMPACT OF VITAMIN D DEFICIENCY ON ORAL HEALTH

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ABSTRACT

Approximately Normally, the body can produce vitamin D at sun exposure. Vitamin D plays a key role in bone and tooth mineralization, and when levels are unregulated it can lead to the "rachitic tooth", which is a defective and hypomineralized organ highly susceptible to fracture and decay. The conditions like recurrent aphthous stomatitis, atrophic glossitis or a painful, burning tongue which is characterized by inflammation and defoliation of the tongue, are possibly caused by nutritional deficiencies such as vitamin B and iron deficiencies. The deficiency has been linked to a variety of diseases, including in the oral cavity. Inadequate sun exposure may accelerate the onset of these diseases since the synthesis of vitamin D is interrupted. Vitamin D not only benefits the overall health of oral cavity but is also associated with tooth mineralization. It can help to fight against inflammation and stimulate the production of antimicrobial peptides for immune response. Thus, in this paper, we will briefly discuss the origin of various oral diseases caused by insufficient level of vitamin D in the body and shed light on the potential benefits of safe sun exposure to maintain both oral and body health. Lack of vitamin D can lead to dental caries, and weak or brittle teeth that easily break, chip, and crack. Their inadequacy leads to absorption impairment, increased bleeding tendency, bone resorption, looseness, and premature tooth loss. Inadequacy of those essential minerals is associated with delayed tooth eruption and with enamel or dentin hypoplasia.

KEYWORDS: Oral Health, Vitamin D Deficiency, Periodontal Disease, Periodontitis, Dental Caries.

INTRODUCTION

Studies have found that people of darker skin color with vitamin D deficiency, overweight or obesity have a higher risk of diabetes or oral diseases (periodontal disease, periodontitis and dental caries). Vitamin D is essential in maintaining the calcium homeostasis, even though it also plays an important role in immunity, cardiovascular system, diabetes, cancer, and chronic illness [1]. The primary sources of vitamin D are dietary intake and sunlight exposure in the form of vitamin D_2 and D_3, which are metabolized to 25-hydroxyvitamin D [25 (OH)D] in the liver. The compound is activated in the kidneys in the form of 1,25-dihydroxyvitamin D. Periodontitis is characterized by alveolar bone loss induced by the host's immune response to bacterial assault. For its crucial role in the maintenance of bone structure and immunity, it is only logical to suspect its deficiency will negatively affect the periodontium in the oral cavity. Vitamin D deficiency is diagnosed by the level of serum 25(OH)D, in which the normal value should fall between 20 to 75ng/mL. Although there is no accepted standard value to declare deficiency, most scholars have agreed to numbers from 20 to 30 ng/mL as mild deficiency and severe case if the value falls below 20 ng/mL. low vitamin D and calcium levels are associated with gingivitis and periodontitis. Not only inflammatory conditions, but it also affects the glycaemic status of individuals. Thus, the patients diagnosed with both periodontal disease and type 2 diabetes mellitus (T2DM) can be vitamin D and calcium deficient. Vitamin D and calcium levels are inversely correlated with random blood sugar and glycosylated haemoglobin and also probing pocket depth and clinical attachment loss, thus may contribute towards increase in the severity of periodontal disease activity.

ORAL MANIFESTATION OF VITAMIN D DEFICIENCY

Vitamin D also exhibits anti-inflammatory activities and thus, its deficiency may well relate to some immune diseases, such as insulin-deficiency diabetes, rheumatoid arthritis, etc. The COVID-19 epidemic may also be associated with vitamin D insufficiency [2-5]. Teeth are considered mineralized tissues, surrounded by alveolar bones, which are constituted of three different hard tissues, namely enamel, dentin, and cementum. The process of mineralization occurs simultaneously in the teeth and the bone structure. Any interruption to the process will exhibit similar symptoms of osteogenesis failure. Vitamin D is a key player in this process and low concentration will result in an increase in dental caries [5-7]. Also, the process is subjected to congenital gene defects that may also result in dental caries or corrosion (demineralization) if the teeth were not sufficiently developed. Other studies have pointed out that vitamin D can protect the oral cavity and reduce oral infection, as these bacteria do not only cause inflammation in the gums and periodontium but also can lead to tooth loss. Without knowing the baseline concentration of 25(OH)D, it would be impossible to determine the role of vitamin D in periodontal disease. Vitamin D reduces the risk of gum inflammation, mainly through protecting the periodontium. Thus, it is speculated that by preventing inflammation and modulating the immune response in periodontium, vitamin D can affect the pathogenesis in the oral cavity. Plus, vitamin D functions to increase bone mineral density (the mandible), reduce alveolar bone resorption, and inhibit the inflammatory response associated with periodontal disease[8,9]. The correlation between vitamin D and the disease is dependent of the dosage, especially when we already know that the concentration of vitamin D will affect bone stability and growth, such as in tooth. In addition to the direct impact on bone metabolism, it also indirectly helps by inhibiting pathogens in the periodontium and healing from gingivitis [10-12]. Vitamin D is also known to inhibit cytokines for inflammatory response that leads to the destruction of periodontium. Therefore, immune response is highly dependent of nutritional status of the host. Vitamin D is not only important for the prevention of periodontal disease, but also gingivitis. In fact, it can reverse the state of destruction even when the periodontal disease has already damaged the alveolar bone tissues. Vitamin D is needed to maintain healthy teeth and immune system to prevent periodontal disease, implying that malnutrition will delay the repair process of gingival damage and increase the dental space in oral cavity to permit bacterial invasion into the teeth and the marrow. The biologically active compound, 1,25(OH)2D and the vitamin D receptor will interact to maintain healthy functioning oral epithelial cells and immunity. Although the deficiency of vitamin D is rarely the sole reason to cause cancer, the risk of oral squamous cell carcinoma is dramatically increased if synergistically combined with other genetic or environmental factors. The binding of 1,25(OH)2D/VDR is most likely to maintain the metabolic balance of oral mucosal tissues and provide a protective barrier against diseases like periodontitis and dental plaques [13-15].

SOURCES OF VITAMIN D

Vitamin D is a fat-soluble compound in the class of steroids. 90% of vitamin D can be synthesized naturally by exposing to ultraviolet B rays through the skin. Skin color, age, and duration and area of exposure will all affect the yield [2,16-19]. The most important function of vitamin D is to maintain the balance of calcium and phosphorus ions in the growth and reconstruction of bones. It is also involved in regulating the functions of bones, muscles, nerves and immunity, also including the cardiovascular and endocrine systems, as well as playing a role in cell cycle. Vitamin D requires activation from substrates in the liver and kidneys to become biologically active. These active metabolites of vitamin D include Cholecalciferol (vitamin D3), Calcidiol (25-hydroxyvitamin D3), Calcitriol (1,25-dihydroxyvitamin D3), Ergocalciferol (vitamin D2) and others. The most important of these metabolites are vitamin D2 and vitamin D3 (calciferol).

ORAL WOUND TREATMENT AND HYGIENE

Vitamin D regulates calcium and immune function. Although the correlation between vitamin D deficiency and periodontal inflammation has been well studied, its impact on the healing of oral wound from periodontal surgery is not clear. When compared with sufficient level of vitamin D, the deficiency showed less regression of subosseous defects [16,20-22]. Animal studies have also suggested that vitamin D is essential in anabolic bone formation in the mandible and may have a positive effect on the healing of fracture. Given its ability to heal bones in the oral cavity, the supplement of vitamin D at the time or after the surgery may benefit. However, vitamin D

deficiency is very common in the population that it is usually recommended to supplement enough vitamin D before the periodontal surgery to achieve a good prognosis. Healthy people are capable of producing vitamin D in the body after exposing to sunlight. Its deficiency has been linked to a variety of diseases, including oral diseases. And inadequate exposure to sunlight may accelerate the onset of these diseases, possibly because of insufficient vitamin D synthesis. Vitamin D is not only limited to the mineralization of teeth but also can help the body fight against inflammation and infection by stimulating the production of antimicrobial peptides. This paper would briefly discuss the origin of various oral diseases caused by the insufficient level of vitamin D in the body and shed light on the potential benefits of safe sun exposure for maintaining oral and body health. Since vitamin D deficiency is highly prevalent, it may be advisable to keep the level within the normal range in advance of periodontal surgery to get the best prognosis of treatment [17,23]. Good nutrition is needed for sustaining healthy tissues, maintaining the immune system, and protecting the body against periodontal disease. Malnutrition will delay the repair process in the gingival space and increase the cell permeability, making it easier for bacteria to enter the tissues [24,25]. Apolipoprotein E4 (ApoE4), one of the isoforms coded by a polymorphic APOE gene, has been widely recognized as a risk factor for cardiovascular diseases and as an immunoinflammatory factor, less is known regarding how ApoE4 affects atherosclerosis in periodontitis patients.

PERIODONTAL DISEASE AND THE RISK FOR ATHEROSCLEROSIS

Periodontal disease (PD) is a multifactorial, chronic, inflammatory disease, associated with bacterial plaque, gingival bleeding, edema and increased crevicular fluid formation that results from endotoxin bacteria-driven host immune responses. PD is characterized by the inflammation and destruction of tooth-supporting connective tissues in response to subgingival infection by various periodontal pathogens. The direct causality between atherosclerosis and periodontal diseases is challenging to dissect, due to common etiological roots, such as aging, life-style (diet, sedentarism), and immune-inflammatory and genetic factors. Apolipoprotein E4 (APOE4) is an attractive gene because it has been extensively associated with increased risks and worse outcomes for cardiovascular diseases. The complications associated with atherosclerosis and metabolic syndrome are recognized as worrisome public health problems in developing countries and may especially affect populations with nutritional and immunoinflammatory disorders, with the likelihood of increased fatalities. The systemic inflammatory processes (even those that are low-grade) induced by chronic periodontal disease may be related to peripheral arterial (and endothelial) inflammatory conditions, which may further elevate the risks of atherosclerosis, coronary disease, myocardial infarction, and death. Cardiovascular (CVD) and periodontal diseases share genetic bases of susceptibility and important behavioral components, such as diet, plaque control and smoking-related habits. Studies had improved that both conditions increase with age, lower socioeconomic strata and poor educational background, especially for males, the diabetic population, and individuals undergoing psychological stress or who have a significant genetic predisposition.

CONCLUSION

Periodontitis pathophysiology is associated with a myriad of pro-inflammatory cytokines, skewed towards Th1/Th17 responses and induced osteolysis, the latter of which has been found to be reduced by simvastatin (a cholesterol-lowering drug) treatment. Statistics have shown that the average level of serum 25(OH)D appears to be declining over the past decades because of changes in BMI, dietary habit, and sun exposure. The participants under medication of Teriparatide showed linear bone growth. The deficiency of vitamin D, when compared with those with sufficient vitamin D, showed lower resolution in radiograph, which indicated periodontal infrabony defects. Animal studies suggested that vitamin D plays a crucial role in the anabolic bone formation of the mandible and may even positively affect the healing of fracture. Moreover, vitamin D deficiency was shown to compromise the osseous healing in the oral cavity, as evidenced in the pre-clinical study of bisphosphonate-associated osteonecrosis of the jaw, further supporting the fact that vitamin D assists in the healing of bones in the oral cavity.

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