



The Role of Vitamins in Periodontal Health

Dr. Dhaval J. Thakkar	Post Graduate student, Department of Periodontics K.M. Shah Dental College & Hospital Sumandeep University
Dr. Neeraj Deshpande	Professor, Department of Periodontics K.M. Shah Dental College & Hospital Sumandeep University
Dr. Prasad Nadig	Senior lecturer, Department of Periodontics K.M. Shah Dental College & Hospital Sumandeep university,
Dr. Ruchita Shah	MPH Candidate University of texas health science center

ABSTRACT

Periodontitis is an infection which is caused by plaque and bacteria accumulating below the gums. If left untreated, it can destroy gums and bone, resulting in loss of teeth. While poor dental hygiene plays a role in the development of periodontal disease, poor nutrition also can be a factor. As dental professionals become increasingly aware of the association between systemic and oral health, the importance of addressing systemic nutrition issue takes on a new urgency. Nutrition is one of the modifiable factors that impact host's immune response and the integrity of hard and soft tissues of oral cavity.

KEYWORDS

Vitamins, Nutrition, Periodontitis

INTRODUCTION:

Periodontitis is a destructive inflammatory disease of the tissues that surround and support the teeth, and can be successfully treated by scaling, root planing and periodontal surgery. The onset and progression of disease depend upon a delicate equilibrium between the microbial challenge and the host response. In this respect, nutrition may be of great importance since it has been implicated in a number of inflammatory diseases and conditions, including cardiovascular diseases, type 2 diabetes, rheumatoid arthritis and inflammatory bowel disease, all of which have been associated with periodontitis.¹

Vitamins can be divided into 2 main groups

1. Fat-soluble vitamins

Vitamin A, D, E and K.

2. Water-soluble vitamins

Vitamin B-complex and C.

VITAMIN A

In 1913 two teams of chemists McCollum and Davis, and Osborne and Mendel extracted an accessory food factor from butterfat by the use of ether, thus proving that it was fat soluble. McCollum and Davis called it fat-soluble A. C.E. Bloch, a Danish physician, noted that xerophthalmia in children could be cured by the addition of a milk product to their diet. In 1920 the term fat soluble was dropped, and the factor was simply called vitamin A.²

Effects of vitamin A deficiency on oral structures:

Animal studies have shown vitamin A to be indispensable to the proper growth and development of periodontium, teeth, salivary gland, and oral epithelium. In humans, the teeth are less sensitive to vitamin A deficiency, although some studies suggest that it can exacerbate existing periodontitis.³ Several epidemiologic studies have failed to demonstrate any relation between this vitamin and periodontal disease in humans.

VITAMIN B₁

It is also known as 'Anti beri-beri' or 'Antineuritic' vitamin.

A Dutch physician, Dr. Christian Eijkman, made a observation

that chickens fed milled white rice contracted beriberi, whereas those living on unhusked brown rice did not. When the ailing chickens were placed on a diet of brown rice, they were cured. This experiment demonstrated the cause and cure of beriberi. The first pure preparation of thiamin from rice polishings was made by Jansen. After ten years, Williams and Cline synthesized thiamin and determined its chemical structure.

Effects of vitamin B₁ deficiency on oral structures:

The possible oral manifestations of a deficiency of this vitamin are hyper sensitivity of the oral mucosa, glossitis and loss or diminution of taste, minute vesicles (simulating herpes) on the buccal mucosa, under the tongue or on the palate and erosion of oral mucosa.⁴

VITAMIN B₂

Chemical research on riboflavin was begun in 1879 by Winter Blyth, who observed some pale yellow material in the whey of milk, which he called lactoflavin. Kuhn and his co-workers who reported in 1935 on the synthesis of riboflavin and the relation of its activity to green fluorescence. The later established that lactoflavin and the vitamin are the same. This was the first evidence that a vitamin functions as a coenzyme.

Effects of vitamin B₂ deficiency on oral structures:

The symptoms of riboflavin deficiency (ariboflavinosis) include glossitis, angular cheilitis, seborrheic dermatitis, and a superficial vascularizing keratitis. The glossitis is characterized by a magenta discoloration and atrophy of the papillae. In mild to moderate cases, the dorsum exhibits a patchy atrophy of the lingual papillae and engorged fungiform papillae, which project as pebble-like elevations. In severe deficiency, the entire dorsum is flat, with a dry and often fissured surface. Angular cheilitis begins as an inflammation of the commissure of the lips, followed by erosion and ulceration.

VITAMIN B₃

Pellagra was described more than 200 years ago by Casal, a court physician to King Philip V of Spain, soon after the introduction of corn (maize) into Europe. An Italian physician, Frappoli, named the disease pelleagra.

Effects of vitamin B₃ deficiency on oral structures:

Pellagra is a niacin deficiency disease caused by a primary inadequate dietary intake. Pellagra is characterized by dermatitis, diarrhea, dementia, glossitis, gingivitis, and generalized stomatitis³. Oral manifestations of vitamin B-complex and niacin deficiency in experimental animals include black tongue and gingival inflammation with destruction of the gingiva, periodontal ligament, and alveolar bone.

VITAMIN B₉

During the 1930s and 1940s the classic studies of Dr. Lucy Wills, described an antianemic factor for the treatment of tropical macrocytic anemia in pregnant women. It was effective in the treatment of megaloblastic anemia of pregnancy and of tropical sprue. This factor was present in green leaves. The word 'folic' is derived from the Latin word 'folium', meaning leaf. Therefore, the preparation was called folic acid. Folic acid (folate or folacin) was finally synthesized in 1946 by a team of industrial chemists.

Effects Folic acid deficiency on oral structures : Folic acid deficiency is probably the most common vitamin deficiency in humans because body's stores of folate are relatively low which can last for upto 4 months only. Folic acid deficiency may cause Megaloblastic anemia. Folic acid-deficient animals demonstrate necrosis of the gingiva, periodontal ligament, and alveolar bone without inflammation. Angularcheilosis and gingivitis are also present. Folic acid deficient animals have shown to be vulnerable to ulcerations and infections of lips, tongue, gingiva, periodontium and oropharynx. Folic acid supplementation may increase resistance to the development of periodontal inflammation in humans.

VITAMIN B₁₂

Pernicious anemia leading inevitably to death in 2 to 5 year was first described by Thomas Addison of London in 1849. In 1929 W.B. Castle demonstrated that ingesting a combination of beef muscle and gastric juice was effective in the treatment of pernicious anemia. Castle called the gastric juice intrinsic factor and the beef muscle extrinsic factor.

Effect of vitamin B₁₂ Deficiency on oral structures : Oral structures have a bright red, smooth, sore, and burning tongue resulting from an atrophic glossitis.

VITAMIN C

In 1932 C.G. King isolated vitamin C from lemons and identified it as the 'antiscorbutic' vitamin, and at the same time Szent-Gyorgyi was making the same discovery using sweet red peppers. As this vitamin was the third to be discovered, it was called vitamin C. It is also called ascorbic acid (a shortened form of antiscorbutic) because it is an acid with antiscorbutic properties.²

There are a variety of factors that influence vitamin C requirement. Cigarette smokers require as much as 50% more vitamin C than nonsmokers. Women taking birth control pills and elderly persons may have lower blood levels of ascorbic acid than desirable. Also, it has been shown that work in hot climates may increase vitamin C requirements.⁵

Effect of Vitamin C deficiency on oral structures: Severe vitamin C deficiency results in scurvy, characterized by hemorrhagic diathesis and retardation of wound healing. Bleeding, swollen gingiva and mobile teeth are also common features of scurvy³.

It also leads to defective formation and maintenance of collagen, retardation or cessation of osteoid formation, and impaired osteoblastic function. It is also characterized by increased capillary permeability, susceptibility to traumatic hemorrhages, hyporeactivity of the contractile elements of the peripheral blood vessels, and sluggishness of blood flow.⁵

Gingival changes include redness, swelling, tendency towards bleeding upon minimal stimulation and an alteration towards

spongy consistency.⁶

VITAMIN D

In 1918 Sir Edward Mellanby demonstrated that rickets in puppies was a nutritional deficiency disease that was curable by the administration of cod liver oil.² In 1922 researchers found that heated and aerated cod liver oil would not cure xerophthalmia in experimental animals (because the vitamin A in the oil had been destroyed by the heating process).

Effect of Vitamin D deficiency: Eruption of permanent teeth may be delayed, and there may be enamel defects ranging from pitting to complete absence of enamel⁸. Vitamin D and calcium deficiency have been found to result in generalized jaw bone resorption and loss of PDL³

Enamel hypoplasia is a common sign associated with vitamin D deficiency during mineralization and maturation of tooth enamel prior to eruption. Such hypoplastic teeth have increased susceptibility to dental caries². The first dental change seen when vitamin D deficiency occurs during tooth development and calcification is hypoplasia (incomplete development of the enamel and dentin). As a result of a lack of vitamin D, the enamel calcifies poorly and may in some areas fail to form. In the dentin, spaces that represent uncalcified dentin matrix occur. The appearance of a calcitoxic line in the dentin is the earliest sign of an acute deficiency of vitamin D⁹

More recent studies showed significant associations between periodontal health and intake of vitamin D and calcium and that dietary supplementation with calcium and vitamin D may improve periodontal health, increase bone mineral density in the mandible and inhibit alveolar bone resorption.⁹

Garcia et al.¹⁰ reported that calcium and vitamin D supplementation may reduce the severity of periodontal disease if used at doses higher than 800-1,000 IU daily. It was also observed that vitamin D, in addition to its role in bone and calcium homeostasis, acts as an anti-inflammatory agent because it inhibits immune cell cytokine expression and causes monocyte/macrophages to secrete molecules that have a strong anti-inflammatory effect.

VITAMIN E

In 1936 the first group isolated an alcohol having vitamin E activity from wheat germ oil and called it alphatocopherol from the Greek words meaning childbirth.²

Deficiency: Vitamin E deficiency is rare in human beings. No relationship has been demonstrated between deficiencies in vitamin E and oral disease, but systemic vitamin E appears to accelerate gingival wound healing in the rat.

Conclusion:

Vitamins do not yield energy but enable the body to use other nutrients. Since the body is generally unable to synthesize them (at least in sufficient amounts) they must be provided by food. A well balanced diet supplies in most instances the vitamin needs of a healthy person.

Vitamins play an essential role for constant regenerative processes for coping with oxidative stress and to maintain adequate immune response. In oral health, deficiency of certain vitamins can lead to defects of hard tissue, oral mucosa and periodontium. It can be assumed that an additional intake of nutritional supplements will not cause any side effects.

REFERENCES

- 1) Van der Velden U, Kuzmanova D, Chapple L. Micronutritional approaches to periodontal therapy. *J Clin Periodontol*. 2011;38(11):142–58. | 2) Rosenfeld L. Vitamine—vitamin. The early years of discovery. *Clin Chem* 1997;43(4):680-85. | 3) Dorsky R. Nutrition and oral health. *Gen. Dent* 2001;49(6):576-82. | 4) Newman M, Takei H, Klokkevold P, Carranza F. Influence of systemic conditions on the periodontium. In: Newman M, Takei H, Klokkevold P, Carranza F, 11th ed. *Carranza Clinical periodontology* South Asia edition. India: Elsevier, 2011: 284-312. | 5) Nishida M, Grossi S, Dunford R, Trevisan M, Genco R. Dietary vitamin C and the risk for periodontal disease. *J Periodontol* 2000;71(8):1215-23. | 6) Hodge RE, Hood J, Canham JE. Clinical manifestation of ascorbic acid deficiency in man. *Am J Clin Nutr* 1971;24(4):432-38. | 7) Woolfe SN, Hume WR, Kenny EB. Ascorbic acid and periodontal disease. *J West Soc Periodontol Periodontol Abstr* 1980;28(2):44-56. | 8) Miley D, Garcia M, Hildebolt C, Shannon W. Cross-Sectional Study of Vitamin D and Calcium Supplementation Effects on Chronic Periodontitis. *J Periodontol* 2009;80(9):1433-39. | 9) Anand N, Chandrasekaran S, Rajput N. Vitamin D and periodontal health: Current concepts. *J Indian Soc Periodontol* 2013;17(3):302-8. | 10) Garcia MN, Hildebolt CF, Cvitelli R. One year effect of vitamin D and calcium supplementation on chronic periodontitis. *J Periodontol* 2011;82(1):25-32.