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Role of Vitamins in Oral Health & Disease: an Overview

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ABSTRACT *Vitamins in diet play an important and equivocal role in general and oral health. Vitamins (both fat soluble and water soluble) are essential for development, maturation and functioning of oral tissues at almost every decade of life. The objective of this article is to highlight the importance of vitamins in oral health and disease.*

Introduction

Vitamins are catalysts for all metabolic reactions using proteins, fats and carbohydrates for energy, growth and cell maintenance. Because only small amounts of these chemical substances obtained from food facilitate millions of processes, they may be regarded as miracle workers.[1] Eating fats, carbohydrates and proteins without enough vitamins means the energy from these nutrients cannot be utilized. The opposite is also true. Vitamins do not provide energy and they cannot be used without an adequate supply of fats, carbohydrates, proteins and even minerals.[2] Fat soluble vitamins (Vitamin A,C,D,E,K) are different from water soluble vitamins (B,C), mainly because larger amounts can be stored in the body. Vitamin A and vitamin D are stored for long periods of time; hence minor shortages may not be identified until drastic depletion has occurred.[1] Vitamin B complex and vitamin C are all water soluble and also organic in nature. In contrast to vitamin C and other fat soluble vitamins, B complex vitamins also contain nitrogen. Water soluble vitamins have vital roles as co-enzymes, which are necessary for almost every cellular reaction in the body. Most water soluble vitamins are readily absorbed in the jejunum. As a rule, high concentrations of these vitamins result in decreased absorption efficiency.[2]

Vitamin A (Retinol)

Vitamin A is an essential nutrient needed in small amounts by humans for the normal functioning of the visual system; growth, development and maintenance of epithelial cellular integrity; immune function and reproduction. These dietary needs for vitamin A are normally provided for as preformed retinol (mainly as retinyl ester) and pro-vitamin A carotenoids.[3] Vitamin A also has an important role in the development of teeth, especially in the formation of ameloblasts (in enamel) and odontoblasts (in dentin). Vitamin A deficiency during pre-eruptive stages of tooth development leads to enamel hypoplasia and defective dentin formation. Severe vitamin A deficiency may result in enamel hypoplasia and defective dentin formation in developing teeth. Enamel hypoplasia involves defects in the enamel matrix and incomplete calcification of the enamel and dentin. Odontoblasts lose their ability to arrange themselves in normal parallel linear formation, resulting in degeneration and atrophy of ameloblasts. The normal deposition of dentin is thus altered. Vitamin A is also involved with normal teeth spacing and promotes osteoblast function of the alveolar bone.[4]

Vitamin D

Although vitamin D has been called a vitamin, it is more appropriately classified as a hormone (a compound that is secreted by one type of cell that acts to control the function of another type of cell). Vitamin D may also be involved in the functioning of cells involved in the formation of red blood cells, skin, cardiovascular function, and immune response.[5] Deficiency of vitamin D causes rickets in children and osteomalacia in adults. In rickets, the alveolar bone is affected just like other bones in the body. The trabeculae of the alveolar bone will also be affected. Delayed dentition and small molars are also observed in vitamin D deficiency. In osteomalacia, the oral manifestations include loss of the lamina dura around the roots of the teeth. A small number of patients with evidence of rickets develop enamel hypoplasia as a result of vitamin D deficiency. Usually these changes are visible only with the aid of a microscope or during clinical assessment with careful exploration of the tooth surface using a sharp explorer. The enamel does not appear to be weakened, but the rough surface may facilitate adherence of dental plaque and food residue.[4]

Vitamin E

Eight different compounds are collectively called vitamin E; four tocopherol and four tocotrienols. Biological activity of

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each form varies; alpha tocopherol is the most active form and is better utilized by the body.[6]

Vitamin E is the major lipid-soluble antioxidant in the cellular defense system and is exclusively obtained from the diet. Lack of research, especially for long term use, on excessive amounts of vitamin E prevents the establishment of toxic levels; however, vitamin E is relatively safe even in amounts above the recommended daily allowance (RDA). Because vitamin E is widely distributed in foods, dietary deficiencies seldom occur if a well-balanced varied diet is consumed. Muscle and neurological problems are also a consequence of human vitamin E deficiency. Early diagnostic signs of deficiency include leakage of muscle enzymes such as creatine kinase and pyruvate kinase into plasma, increased levels of lipid peroxidation products in plasma, and increased haemolysis.[6]

Vitamin K

Vitamin K is an essential fat-soluble micronutrient. Vitamin K dependent proteins have been identified in bone, kidney and other tissues. These proteins bind calcium and may be involved in bone crystalline formation. Vitamin K functions as a catalyst for synthesis of blood clotting factors primarily in maintaining prothrombin levels, which is the first stage in forming a clot. A low prothrombin level result in impaired blood coagulation.[5] Low dietary intake of vitamin K has been linked to reduced bone mass density in women, increasing the risk of hip fractures. Newborn infants may develop hemorrhagic disease secondary to vitamin K deficiency because the gut is sterile during the first few days after birth. Newborn infants are usually given a single dose of vitamin K intramuscularly immediately after birth to prevent hemorrhage.[4]

Vitamin C

Vitamin C functions as an antioxidant in numerous bodily reactions. As a coenzyme, it also has numerous metabolic roles. It is important in the production of collagen (insoluble protein of connective tissue, cartilage, and bone), which plays a vital role in wound healing. Vitamin C strengthens tissue and promotes capillary integrity. Vitamin C facilitates development of RBCs by enhancing iron absorption and utilization. It also aids the body in utilization of folate and vitamin B12. Based on epidemiological evidence, ascorbic acid or vitamin C intake may be associated with a lower risk of gastric and esophageal cancer.[4] Scurvy, caused by vitamin C deficiency can occur in as quickly as 20 days. It is characterized by spontaneous gingival hemorrhaging, petechiae, follicular hyperkeratosis, diarrhea, fatigue, depression and cessation of bone growth. Skeletal and vascular lesions related to scurvy probably arise from a failure of osteoid formation. Inadequate amounts of vitamin C during tooth development may result in changes similar to scurvy or scorbutic changes in the teeth because of changes in the ameloblasts and odontoblasts. Atrophy of ameloblasts and odontoblasts also occurs in vitamin C deficient environment. Gingivitis, caused by ascorbic acid deficiency, also affects the periodontium, resulting in tooth mobility. The effect is probably related to weakened collagen secondary to vitamin C deficiency, which results in resorption of the alveolar bone.[1]

Supplemental amounts of vitamin C can cause gastrointestinal distress and interfere with vitamin B12 absorption. The potential toxicity of excessive doses of supplemental vitamin C relates to intraintestinal events and to the effects of metabolites in the urinary system. Intakes of 2–3 g/day of vitamin C produces unpleasant diarrhea from the osmotic effects of the unabsorbed vitamin in the intestinal lumen in most people.[1]

Vitamin B1 (Thiamin)

Thiamin functions as a coenzyme in metabolism of energy nutrients via the Krebs cycle to produce energy. This role makes it crucial for normal functioning of the brain, nerves, muscles, and heart. Thiamin is also a necessary component in the synthesis of niacin and helps regulate appetite. It is a constituent of enzymes that degrade sucrose to organic acids that can ultimately dissolve tooth enamel.[1]

Whether or not a thiamin deficiency is evident in oral tissues is controversial. Some clinicians have associated a flabby, red, and edematous tongue with thiamin deficiency. The fungiform papillae enlarge and become hyperemic. Gingival tissues sometimes present an "old rose" color. Wernicke Korsakoff syndrome is another thiamin deficient disease typically associated with alcoholism that is characterized by mental confusion, nystagmus (involuntary rapid movement of eyeball), and ataxia (a gait disorder characterized by uncoordinated muscle movements).[4]

Vitamin B2 (Riboflavin)

Riboflavin functions as a coenzyme in the metabolism of carbohydrate, protein, and fat to release cellular energy. Riboflavin is also essential for healthy eyes and maintenance of mucous membranes. Along with thiamin, riboflavin is necessary for synthesis of niacin.[1]

Symptoms associated with riboflavin deficiency or ariboflavinosis, include angular cheilosis, dermatitis and anemia. With consistently inadequate intake, these symptoms may be observed within 8 weeks. Along with angular cheilosis, the lips may become extremely red and smooth. Fungiform papillae become swollen and slightly flattened and mushroom shaped during early stages of riboflavin deficiency; the tongue has pebbly or granular appearance. Severe chronic deficiencies lead to progressive papillary atrophy and patchy, irregular denudation of the tongue. The tongue may become purplish red or magenta colored because of vascular proliferation and decreased circulation. In more advanced cases, the entire tongue may become atrophic and smooth. These symptoms, especially glossitis and dermatitis, may actually be secondary to vitamin B6 deficiency.[4]

Vitamin B3 (Niacin)

Niacin is crucial as a coenzyme in energy (ATP) production. It functions with riboflavin in glucose production and metabolism and is also involved in lipid and protein metabolism. Niacin is essential for growth of cariogenic oral microorganisms. It also functions in enzymes involved in the microbial degradation of sucrose to produce organic acids. Niacin (nicotinic acid) deficiency classically results in pellagra, which is a chronic wasting disease associated with a characteristic erythematous dermatitis that is bilateral and symmetrical, a dementia after mental changes including insomnia and apathy preceding an overt encephalopathy, and diarrhea resulting from inflammation of the intestinal mucous surface.[7]

Deficiency also affects mucous membrane: painful stomatitis causes diminished food intake, and lesions in the gastrointestinal tract result in diarrhea and less vitamin absorption. Pellagrous glossitis begins with swelling of the papillae at the tip and lateral borders of the tongue. The tongue becomes painful, scarlet, and edematous. Atrophic changes involve loss of filiform and fungiform papillae, and

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the tongue becomes smooth and shiny. The mucosa is also reddened. Fissures occur in the epithelium and along the sides of the tongue; these become infected rapidly. The gingiva may become inflamed, resembling that found in ulcerative gingivitis. The corners of the lips are initially pale; fanlike fissuring occurs that radiates into the perioral epithelium and may leave permanent scars.[6]

Vitamin B6 (Pyridoxine)

A deficiency of vitamin B6 alone is uncommon because it usually occurs in association with a deficit in other B-complex vitamins. Hypovitaminosis B6 may often occur with riboflavin deficiency, because riboflavin is needed for the formation of the coenzyme PLP. Infants are especially susceptible to insufficient intakes, which can lead to epileptiform convulsions. Skin changes include dermatitis with cheilosis and glossitis. A decrease in the metabolism of glutamate in the brain, which is found in vitamin B6 insufficiency, reflects a nervous system dysfunction. As is the case with other micronutrient deficiencies, vitamin B6 deficiency results in an impairment of the immune system. Pyridoxine deficiency induced glossitis is denoted by pain, edema, and papillary changes. Initially, the tongue has a scalded sensation, followed by reddening and hypertrophy of the filiform papillae at the tip, margins and dorsum.[4]

Folic Acid

The generic term folate encompasses several constituents that have nutritional properties similar to those of folic acid. Several different metabolically active forms have been identified. Folate deficiency, the most common vitamin deficiency among the B complex vitamins, may occur secondary to excessive alcohol consumption, pregnancy/lactation, kidney dialysis, liver disease, inadequate dietary intake, gastrointestinal disease or medications that interfere with folate absorption or metabolism. Glossitis is usually present in persons with folic acid deficiency. The tongue becomes fiery red and papillae are absent. Marked chronic periodontitis with loosening of teeth may occur. Periodontitis is an inflammatory disease of the periodontium. Folic acid deficiency impairs immune responses and resistance of the oral mucosa to penetration by pathogenic organisms such as candida.[4]

Vitamin B12 (Cobalamin)

Vitamin B12 represents a complex group of compounds that contain cobalt. It is the only vitamin that contains a mineral. Vitamin B12 functions as a coenzyme in conjunction with folate metabolism in nucleic acid synthesis. It also functions in the catabolism of certain amino acids and fatty acids. Vitamin B12 is essential for making red blood cells and for myelin synthesis. Myelin is the lipid substance that insulates nerve fibers and affects transmission of nerve impulses.[4]

Neurological symptoms, such as numbness or tingling, occur as consequences of demyelination of the nerves. Deficiency symptoms are rapidly corrected with vitamin B12 injections. Initial oral symptoms of vitamin B12 deficiency present with glossopyrosis (unexplained pain of the tongue), followed by swelling and pallor with eventual disappearance of the filiform and fungiform papillae. The tongue may be completely smooth, shinny, and deeply reddened with a loss or distortion of taste. Bright red, diffuse, excruciating painful lesions may occur in the buccal and pharyngeal mucosa and undersurface of the tongue. An oral examination may reveal stomatitis or a pale or yellowish mucosa, xerostomia, cheilosis, hemorrhagic gingiva and bone loss.[4]

Pantothenic Acid

Pantothenic acid is synthesized by most microorganisms and plants. It is particularly abundant in animal foods and whole grain cereals. Bacteria in the digestive tract also produce pantothenic acid. Pantothenic acid is similar to the other B vitamins in its metabolic roles. Pantothenic acid plays a key role in carbohydrate, fat, and protein metabolism. Additionally, it is important in synthesis and degradation of triglycerides, phospholipids, and sterols, and in formation of certain hormones and nerve regulating substances. The widespread occurrence of releasable pantothenic acid in food makes a dietary deficiency unlikely. If a deficiency occurs, it is usually accompanied by deficits of other nutrients. Toxicity is not a problem with pantothenate, as no adverse effects have been observed.[4]

Biotin

Biotin functions as coenzyme in metabolism of carbohydrates, proteins, and fats. Biotin operates within four carboxylases. Three of the four biotin-dependent carboxylases are mitochondrial (pyruvate carboxylase, methylcrotonyl-CoA carboxylase, and propionyl-CoA carboxylase) whereas the fourth (acetyl- CoA carboxylase) is found in both mitochondria and the cytosol. In all these cases, biotin serves as a carrier for the transfer of active bicarbonate into a substrate to generate a carboxyl product.[5]

Biotin deficiency in humans has been clearly documented with prolonged consumption of raw egg whites, which contain biotin-binding avidin. Biotin deficiency has also been observed in cases of parenteral nutrition with solutions lacking biotin given to patients with short-gut syndrome and other causes of mal-absorption. Toxicity is not a problem because of the limited intestinal absorption of biotin. Oral signs of biotin deficiency are pallor of the tongue and patchy atrophy of the lingual papillae. Although the pattern resembles geographic tongue, it is confined to the lateral margins or is generalized to the entire dorsum.[4]

Conclusion:

Diet containing vitamins play an important role in normal health of the oral structures. Deficiencies causes oral diseases such as developmental defects, oral mucosal diseases and periodontal diseases. The commonly affected oral structures as a result of vitamin deficiency are soft tissues like tongue, gingiva and lining mucosa. However, fat soluble vitamin deficiency affects hard tissues, by impaired development of teeth and bony support. Signs and symptoms of vitamin toxicity have not been reported clearly for both water and fat soluble vitamins for oral health due to lack of research on long term and excessive use of vitamins as compared to vitamin deficiency.

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