

Vitamins and Their Role in Dental Tissue: A Review from Dentist Aspect

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ABSTRACT

Oral hygiene is the practice of keeping the mouth and teeth clean to prevent dental problems, most commonly, dental cavities, gingivitis, periodontal diseases and bad breath. There are also oral pathologic conditions in which good oral hygiene is required for healing and regeneration of the oral tissues. These conditions include gingivitis, periodontitis, and dental trauma. A complete oral health home program includes sound nutritional habits. Many of the foods that help your body build strong muscles and bones also help build strong, healthy teeth and gums. Vitamins are calorie free, organic, essential molecules needed by the body in minute amounts. Vitamins belong in two groups named as water soluble and fat soluble. Vitamins C and B are water soluble and dissolve in water, while excesses are excreted by the body. Fat-soluble vitamins, A, D, E, and K dissolve in fat and excesses are stored in our fat cells. Dairy products provide calcium and vitamin D for strengthening teeth and bones. Sometimes known as the “sunshine vitamin”, vitamin D can help lessen inflammation associated with periodontal disease. Research shows that foods fortified with vitamin D such as milk, eggs, sardines, and tuna fish, as well as moderate exposure to sunlight, can provide you with the amount of vitamin D required to stay healthy. Breads and cereals supply B vitamins for growth and iron for healthy blood, which in turn contributes to healthy gum tissue. Fruits and vegetables containing vitamin C are essential to maintaining healthy gums. This review describes the importance and role of vitamins in prevention of oral diseases particularly from Pedodontics aspect.

Keywords: vitamins, oral health, pediatric dentistry, water soluble, fat soluble.

INTRODUCTION

A vitamin is usually defined as an organic substance not made by body, which is soluble in either fat or water and is ordinarily needed in only minute quantities to act as co factor in variety of metabolic reactions. The word vitamin is reference to the fact that the substance it designates is essential to life. The term therefore, is functional and not chemically descriptive. Some of vitamins occur in physiologically inactive form. They become active only after conversion within the animals. These are called provitamins such as vitamin A exists in plants as carotene, which is activated in liver. The vitamins are classified based upon the fat and water solubility. The fat soluble vitamins are vitamin A, D, E and K whereas water soluble includes vitamin B complex and vitamin C [1].

Vitamin A

Vitamin A is a fat-soluble vitamin that is essential to our health. It helps us see normally in the dark, promotes normal growth and health of body cells and keeps skin healthy. There are animal sources (retinol) and vegetable sources (carotenoids) of vitamin A in foods. Only a few of the carotenoids in foods are converted to vitamin A in the body. Beta-carotene is the most familiar carotenoids. Beta-carotene, like several other carotenoids, acts as an antioxidant that slow or prevent cell damage. By protecting cells from damage, antioxidants may reduce risk for certain cancers and heart disease.

It is well established that vitamin A is concerned primarily with process of differentiation of epithelial cells. In vitamin A deficiency the epithelial cells fail to differentiate. This means that the cells in the basal layer lose their specificity and tend to form stratified squamous epithelium with keratin production, independent of type of cells previously formed by the basal

cells. Thus one of the basic changes is the keratinizing metaplasia of epithelial cells that occurs throughout the body, including the mucous membrane of the trachea, conjunctiva, ureter and salivary and other glands [2].

Oral manifestation

1. The classic work of Wolbach and Howe [3] on dental changes in vitamin A deficiency of the rat and guinea pig has shown that in deficiency of vitamin A, the odontogenic epithelium fails to undergo normal histodifferentiation and morphodifferentiation and the result is an increased rate of proliferation. Therefore epithelial invasion of pulpal tissue is characteristic of vitamin A deficiency. Since the enamel forming cells are disturbed, enamel matrix is arrested and poorly defined so that calcification is disturbed and enamel hypoplasia results. The dentin too is atypical in structure lacking the normal tubular arrangement and containing cellular and vascular inclusions.
2. The eruption rate is retarded and ceases completely in prolonged deficiency.
3. The rate of alveolar bone formation decreases.
4. The gingival epithelium becomes hyperplastic and its prolonged deficiency shows keratinization. This tissue is invaded by bacteria that may cause periodontal destruction and microabscess formation.
5. The major and minor salivary glands undergo typical keratinizing metaplasia.

Authors studied vitamin A deficiency in humans and found that young ones whose mothers are deficient in vitamin A for five months preceding their birth, changes are more severe, resulting in distortion of the shape of both the incisors and molars.

Vitamin D

Vitamin D is known as sunshine vitamin and antirachitic vitamin. It has always been described as vitamin; however it is probably best thought as hormone. Nutritionally important forms are vitamin D₂ (Calciferol), vitamin D₃ (Cholecalciferol) and vitamin D₃ is formed from 7-dehydrocholesterol, which is an intermediate compound in synthesis of cholesterol. 7-dehydrocholesterol is ultimately formed from acetyl coA which is never in short supply. Vitamin D exerts its major influence by combining with non-histone protein in the nuclei of intestinal epithelial cells. This combination, in turn, exposes a portion of the genetic material for transcription of a specific protein, calcium binding protein. In its role as an activator to calcium binding protein, vitamin D has a protean manifestation in parathyroid function, which subsequently affects calcium and phosphorus levels in body [4].

Vitamin D deficient Rickets

Vitamin D deficient rickets is developed in urban areas that are deprived of sunlight. When air pollution filters out the ultraviolet portion of the spectrum, cholecalciferol formation is blocked.

Infants rapidly develop the characteristic bony deformities. Identical lesions are seen in sun-rich areas where the diet is high in phytate, which binds the available dietary calcium. Social customs, such as the use of the pardha, may also result in rickets. The age of onset of deficiency is important in the eventual morbid, with premature infants being at highest risk.

Clinical features

The effect of rickets is reflected only in bones and teeth. The changes in the bones are found in the epiphyseal plate, the metaphysis and the shaft. The degree of change depends on the rate of growth of the bones at the time of the deficiency, young children are more severely affected than older individuals. In young children, placed on rachitogenic diets the first change seen is the cessation of calcification of their epiphyseal disks. Since the intercellular ground substance does not become calcified, the cartilage cells are not denied nutrition. Therefore they do not die, and their continued growth and multiplication lead to an increase in the width of the disks. The disks thicken irregularly because some focal areas usually calcify. Since unmineralized bone is not capable of supporting weight as normal bone, children with rickets show bowing of legs.

Oral manifestation

Mellanby [5] was the first to report the effects of rickets on teeth, which include developmental abnormalities of dentin and enamel, delayed eruption and misalignment of teeth in the jaw. The usual dental findings in rickets are large pulp chambers and pulp horns, loss of lamina dura, interglobular dentin, and premature loss of primary teeth. In human rachitic teeth there is an abnormally wide predentin zone and much interglobular dentin. The eruption rate of deciduous and permanent teeth is retarded in rickets.

Osteomalasia (Adults Rickets)

Osteomalasia in adults is equivalent to juvenile rickets. Unlike the juvenile rickets only the flat bones and the diaphysis of long bones are affected. The disease is more common in post menopausal females with history of low dietary calcium

intake and little exposure to sunlight. This disorder is endemic in certain areas of India, China and Japan. The clinical features include the remodeling of bone in the absence of adequate calcium, which results in softening and distortion of the skeleton and an increased tendency towards fracture. Periodontitis is more prevalent in vitamin D deficiency cases.

Vitamin D resistant rickets

The characteristic features include hypophosphatemia and hyperphosphaturia associated with decreased renal tubular reabsorption of inorganic phosphates, familial occurrence, being inherited as an X-linked dominant trait, rickets or osteomalacia which does not respond to usual doses of vitamin, diminished growth with short stature, normal vitamin D metabolism and the absence of other related abnormalities. The children affected by this form of resistant rickets are usually recognized when child begins to walk. The X rays reveals abnormalities such as skull deformities, retardation and eruption of teeth and sitting deformities of legs [6].

Oral manifestations

Characteristically, there is histological evidence of widespread formation of globular, hypocalcified dentin, with clefts and tubular defects occurring in the region of pulp horn. The pulp horns are elongated and reach till dentinoenamel junction. Because of these defects, there is commonly invasion of pulp by microorganisms without demonstrable destruction of tubular matrix. There is often periapical involvement of grossly normal appearing deciduous or permanent teeth, followed by development of multiple gingival fistulas. Abnormal cementum and lamina dura around the teeth is also reported. Alveolar pattern is also found to be abnormal.

Vitamin-E

Authors noted that a fat soluble vitamin prevented the fetal resorption in animals. This factor was named vitamin E and given the name tocopherol, which means the alcohol which brings the forth spring. The main function of vitamin E is to prevent peroxidation of unsaturated fatty acid. Vitamin E consist of 8 naturally occurring tocopherols of which alpha tocopherol is most active. Vitamin E deficiency in experimented animals resulted in multisystem disorders, including decreased male fertility, impaired fetal –maternal vascular relationship, nutritional muscular dystrophy, encephalomalacia and hemolysis.

Vitamin K

The various authors noticed that a peculiar hemorrhagic diathesis in chicks fed with a fat extracted diet. This clotting defect was not due to deficiency of vitamin A, E or D which had previously discovered. This new substance was named vitamin K or coagulation vitamin. Studies later provide evidence that vitamin K is involved in both extrinsic and intrinsic system of coagulation, particularly with prothrombin (factor II) synthesis. Vitamin K is necessary for the post-translational carboxylation of glutamic acid necessary for calcium binding to gammacarboxylated proteins such as prothrombin, factors VII, IX, X, protein C, protein S, and proteins found in the bone.

There are two natural forms vitamin K, namely vitamin K₁, also known as phyloquinone, derived from vegetable and animal sources and vitamin K₂ or menaquinone, synthesized by bacterial flora and found in hepatic tissue. Vitamin K₃ or menadione is a chemically synthesized provitamin and is water-soluble. This is converted into menaquinone by the liver. For this reason vitamin K is discussed under water-soluble vitamins [7].

Oral manifestations

The most common oral manifestation of vitamin K deficiency is gingival bleeding. Prothrombin levels below 35 per cent will result in bleeding after toothbrushing; however, when prothrombin levels fall below 20 per cent, spontaneous gingival hemorrhages will occur.

Vitamin C

Vitamin C has been the object of intensive research for many years. Scurvy, which results from vitamin C deficiency, has been known since the time of the Ebers Papyrus in Egypt (1500 BC).

The effect on history, through the occurrence of scurvy in military troops, is notable. British sailors in the 19th century were referred to as 'limeys' because of their consumption of citrus fruits to prevent scurvy while on long voyages.

The characteristic change in the teeth of scorbutic guinea pigs is the atrophy and disorganization of the odontoblasts, resulting irregularly laid down dentin with few, irregularly arranged tubules. Eventually dentin formation ceases, and the predentin becomes hypercalcified, producing a heavy, basophilic staining line between dentin and pulp. The odontoblasts finally become indistinguishable from other pulpal cells [8].

Clinical Features of Scurvy:

1. The interdental and marginal gingiva is bright red with a swollen, smooth, shiny surface.
2. In fully developed scurvy the gingival becomes boggy, ulcerates and bleeds.
3. The color changes to a violaceous red. In infants the enlarged tissue may cover the clinical crowns of the teeth.
4. The patients have the typical foul breath of persons with fusospirochetal stomatitis.
5. Severe chronic cases of scurvy, hemorrhages it and swelling of the periodontal membranes occur, followed by loss of bone and loosening of the teeth, which eventually exfoliate

Vitamin B Complex

The oral signs of deficiencies of the B vitamins occur primarily in the oral soft tissues; the tongue, mucous membranes, gingiva and lips. At present the vitamin B group contains 11 well-characterized vitamins; thiamin, riboflavin, niacin, pyridoxine, pantothenic acid, biotin, folic acid, vitamin B12, inositol, para-aminobenzoic acid and choline [9].

Thiamin (Vitamin B1)

Thiamin is a colorless basic organic compound composed of a sulfated pyrimidine ring. The main sources of thiamin are yeast, pork, legumes, whole grains, and nuts.

Clinical Features of Thiamin Deficiency.

In man thiamin deficiency leads to beriberi, which is generally insidious in onset, chronic in course and sudden death may occur. Alcoholic patients with chronic thiamin deficiency are having CNS manifestations known as Wernicke's encephalopathy, which consists of horizontal nystagmus, ophthalmoplegia, cerebral ataxia, and mental impairment [10].

Riboflavin (Vitamin B2)

Riboflavin is a fully dialyzable, intensely yellow water-soluble pigment which is decomposed by light. It is readily absorbed from the intestinal tract and is phosphorylated in the walls of the intestine as well as in other tissues of the body. Riboflavin is a constituent of two different groups of coenzymes, riboflavin 5'-phosphate and flavin adenine dinucleotide. These coenzymes are essential to the oxidative enzyme systems utilizing the electron transport system. It is essential for carbohydrate, fat, and protein metabolism reflecting its role as respiratory coenzyme and electron donor [11].

Clinical Features of Riboflavin deficiency

1. Riboflavin deficiency is particularly common among children who do not drink milk.
2. In the mild deficiency state there is a glossitis which begins with soreness of the tip and the lateral margins of the tongue
3. The filiform papillae become atrophic, while the fungiform papillae remain normal or become engorged and mushroom shaped, giving the tongue surface a reddened, coarsely granular appearance.
4. In severe cases the tongue may become glazed and smooth, owing to complete atrophy of all papillae.
5. In many cases the tongue has a magenta color which can be easily distinguished from cyanosis.
6. Paleness of the lips, especially at the angles of the mouth, but not involving the moist areas of the buccal mucosa, is the earliest sign of the deficiency disease.
7. The pallor which usually continues for days, is followed by cheilosis, which is evidenced by maceration and fissuring at the angles of the mouth.
8. The fissures may be single or multiple.
9. Later the macerated lesions develop a dry yellow crust which can be removed without causing bleeding.
10. The lips become unusually red and shiny because of a desquamation of the epithelium. As the disease progresses, the angular cheilosis spreads to the cheek.
11. The fissures become deeper, bleed easily and are painful when secondarily infected with oral and skin microorganisms. Deep lesions leave scars on healing. The gingival tissues are not involved.
12. Riboflavin deficiency also affects the nasolabial folds and the alae nasi, which exhibit a scaly, greasy dermatitis.
13. A fine scaly dermatitis may also occur on the hands, vulva, anus, and perineum.
14. Ocular changes, consisting of corneal vascularization, photophobia and a superficial and interstitial keratitis, have also been described.

Niacin

In the living organism, ingested niacin is transformed into nicotinic acid amide, which is utilized to form coenzyme I (nicotinamide-adenine dinucleotide, or NAD) and coenzyme II (nicotinamide-adenine dinucleotide phosphate, or NADP). A deficiency of this vitamin leads to the basic symptoms of pellagra in human beings and to black tongue in dogs.

Pyridoxal-5-phosphate is required for the conversion of tryptophan into nicotinic acid in the tissues. The accepted conversion ratio is 60mg tryptophan to 1mg nicotinic acid. It is important in pentose, steroid, and fatty acid biosynthesis, glycolysis, protein metabolism and oxidation of lactate, pyruvate, and alcohol [12].

Clinical Feature of Niacin deficiency

1. In the prodromal stage of nicotinic acid deficiency, the patient may complain of loss of appetite and vague gastrointestinal symptoms. General weakness, lassitude mental confusion, forgetfulness, and other ill defined symptoms develop.
2. The patient then usually complains of a burning sensation in the tongue, which becomes swollen and presses against the teeth, causing indentations. The tip and lateral margins of the tongue become red.
3. In the acute stages of pellagra, the entire oral mucosa becomes fiery red and painful. The mouth feels as though it had been scalded. Salivation is profuse.
4. The epithelium of the entire tongue desquamates.
5. Tenderness, pain, redness and ulcerations begin at the interdental gingival papillae and spread rapidly.
6. Superimposed necrotizing ulcerative gingivostomatitis or Vincent's infection involving the gingival, tongue and oral mucosa is a common sequel.
7. Epithelial changes followed by the characteristic skin rash particularly in the area exposed to sunlight especially in the neck region are called Casals necklace.

Vitamin B₁₂

Vitamin B₁₂ is found only in bacteria, eggs and foods of animal origin. It does not occur in vegetables and fruit. The average daily requirement for cobalamin in adults is 1–2 µg. Most cobalamin in food is bound to proteins and released when the protein is subjected to acid-peptic digestion in the stomach. The released cobalamin rapidly attaches to a cobalamin-binding protein, R-binder, present in saliva and gastric juice. The R-binder in the R-binder complex is broken down in the alkaline environment of the jejunum by pancreatic trypsin and the released cobalamin binds to intrinsic factor produced by gastric parietal cells in the duodenum and is transported to the distal ileum, where specific receptors bind the B₁₂-intrinsic factor complex resulting in B₁₂ absorption [13].

This attachment is calcium dependent, the calcium being provided by the pancreas. In the absence of intrinsic factor, cobalamin is absorbed only very inefficiently by passive diffusion. Most cobalamin is stored in the liver (about 4–5 mg). Megaloblastic anemia occurs when the body's cobalamin stores fall below 0.1 mg. Macrocytosis due to cobalamin or folate deficiency is a direct result of ineffective or dysplastic erythropoiesis. These vitamins are the most important cofactors necessary for normal maturation of all cells and cobalamin is necessary for DNA synthesis, as its deficiency prevents cell division in the marrow. When either of these factors is deficient, red blood cells (RBCs) become large erythroblasts with nuclear or cytoplasmic asynchrony (poikilocytosis), a characteristic of all megaloblastic anemias [14].

These oral changes may occur in the absence of symptomatic anemia or macrocytosis, as they may precede many systemic indicators of B₁₂ deficiency. Thus, the general dentist, who is cognizant of normal blood values and can interpret anomalies, may order specific blood tests before the patient is referred to a hematologist. However, patients must be referred to a hematologic centre for adequate treatment. A wide range of oral signs and symptoms may appear in anemic patients as a result of basic changes in the metabolism of oral epithelial cells [15].

1. These changes give rise to abnormalities in cell structure and the keratinization pattern of the oral epithelium leading to a "beefy" red and inflamed tongue with erythematous macular lesions on the dorsal and border surfaces because of marked epithelial atrophy and reduced thickness of the epithelial layer.
2. Erythematous macules occurred on the surface of the patient's cheek mucosa and tongue.
3. In addition, soreness of the tongue and generalized ulceration, as well as reduced taste sensitivity, generalized sore mouth or burning mouth are usually reported in the .
4. Although candidiasis and angular cheilitis are common oral complaints of patients with megaloblastic anemia, these problems were not observed in our patient.

The differential diagnosis of patients with these signs and symptoms includes iron deficiency, diabetes, allergy, autoimmune disease, physical and chemical injury, atrophic candidiasis and anemia of chronic disease.

CONCLUSION

Diet influences the development of the oral cavity. An early nutritional imbalance influences malformations. Moreover, the different components of the stomatognathic apparatus undergo periods of intense growth alternated with periods of relative quiescence. It is clear that a nutritional imbalance in a very active period of growth will produce greater damage. A shortage of vitamins and minerals in the phase before conception influences the development of the future embryo, influencing dental organogenesis, the growth of the maxilla, and skull/facial development. Therefore the knowledge relating the importance and role of vitamins stands vital for every parent.

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