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Review Article

All About Vitamins and Its Role in Oral Health

Ahmad Naeem*, Tiwari Arun Kumar, Tiwari Aanshika***, Srivastav Ankita***, Nadeem Ahmad****, Rubeena Bano*****

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Abstract

A vitamin is defined as an organic compound that is required in diet in small amounts for the maintenance of normal metabolic integrity. As new vitamins were discovered, successive letters of the alphabet were assigned to them. However, in some cases a letter was used because it was the first letter of a word describing the principle characteristic of the vitamin. For example, Vitamin K, which is concerned with the coagulation of blood, was derived from the Scadinavian word koagulation. Deficiency causes a specific disease which is cured or prevented by restoring the vitamin in the diet. However, Vitamin D, which can be made in the skin after exposure to sunlight, and niacin which can be formed from the essential amino acid tryptophan, do not strictly conform to this definition. In the present article, a systematic review is presented on multivitamins and its role in oral health care.

Keywords: Vitamins; Diet, Compounds; Energy, Health.

Introduction

Vitamins are a class of organic compounds categorized as essential nutrients. Vitamins are calorie-free molecules and are required by the body in very small amounts. They fall in the category of micronutrients. 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. A common misconception is that "vitamins give you energy." The truth is, vitamins help with the metabolic reaction that releases energy within the food molecules, making them the directors of cell processes. But if the body has an adequate supply of vitamins to help with the creation of energy, taking more will not make one more energetic. Minerals are inorganic substances that play an important role in a variety of metabolic reactions, as cofactors. They form one of the essential components of the diet. They are essentially divided into two major groups: macronutrients which are required in large amounts such as calcium, magnesium, sulphur, sodium, potassium and chloride; and micronutrients or trace elements which are required in very small quantities in the diet [1].

Author's Affiliation: *Senior Lecturer, **Post-graduate Student, Dept. of Prosthodontics, ***Post-graduate Student, Department of Oral Medicine and Radiology, Career Post Graduate Institute of Dental Sciences, Lucknow. ****Professor & HOD, Community Medicine, *****Associate. Professor, Physiology, Integral Institute of Medical sciences & Research, IIMSR, Lucknow

Reprint Request: Naeem Ahmad, Flat No. T1-(A), IMT Estate Apartment, New Hyderabad Colony, Lucknow, Uttar Pradesh-226007.
E-mail: naeem_bds@yahoo.co.in

Material and Methods

For this review, we identified studies through Science Direct, Google Scholar and Medline search engines. Search terms included the following, individually and in combination: "vitamins, diet, compounds, energy, health". We retrieved all papers discussing role of vitamins in general and oral health. We reviewed the findings of each article, and reviewed the references of each paper for additional papers that had been missed in the initial search and that might include findings relevant to the scope of our review. Ultimately, six manuscripts were felt relevant

and representative for inclusion among those referenced in this paper.

Literature Review

Frederick G. Hopkins, a biochemist at Cambridge University, England, and a Nobel Prize winner, reported in 1906 that there was an unknown something in food essential for life and health. In 1912, he had experimental data that supported this statement. Casimir Funk, a Polish chemist, obtained an antiberiberi substance from rice polishings. He believed that the active factor that he found was a protein, i.e., an "amino". Since he considered this amino to be the vital element in the food, he called it a "vitamine." It was found that very few of these "vitamines" were truly protein so the final 'e' was dropped. Vitamin discovery, both accidental and deliberate, was spread out over 40 years. By 1940, all 13 currently recognized vitamins had been discovered and given a sequential letter of the alphabet [2].

Vitamins can be Divided into 2 Main Groups

Fat-Soluble Vitamins

Vitamin A, D, E and K. These are

- Stored in the liver
- Not absorbed or stored easily
- Sometimes toxic in excess (particularly A and D)

Water-Soluble Vitamins

Vitamin B-complex and C. These are;

- Not stored extensively
- Required regularly in the diet
- Generally non-toxic in excess (within reason)
- All B vitamins are coenzymes in metabolic pathways.

Digestion and Absorption

Vitamins are released from food during the digestive process but are not digested [3]. Water-soluble vitamins (vitamin B-complex and C) that are utilized by the body for metabolic processes are absorbed through the small intestine. Any excess that is not used right away is excreted by the kidneys in the urine. Fat-soluble vitamins (vitamins A, D, E, and K) are absorbed with dietary fats through the small intestine. After absorption, they are sent to the liver and fat depots and circulate through the blood with the help of chylomicrons and lipoproteins. (Leevy et al 1968) Patient with liver disease may exhibit reduced storage, reduced hepatic intake, and diminished consumption of vitamins.

Toxicity / Lmbalance

Eating too many fortified foods or taking a megadose of vitamin supplements—more than the recommended daily intake can produce toxic effects. Fat-soluble vitamins are more toxic than water-soluble vitamins because they can be stored and accumulate in adipose tissues. There is less danger of water-soluble vitamins exerting a toxic effect on the body because storage is limited and usually the excess is excreted on a daily basis.

Vitamin D is the most toxic of all vitamins because of its ultimate effect on the human body. Vitamin D enhances the absorption of calcium, and so an excess of Vitamin D causes an excess of calcium circulating in the blood, which is detrimental to the heart. Two water-soluble vitamins can be somewhat toxic: vitamin B-6 and niacin. Both can have detrimental effects if the amount ingested is more than the kidney can handle and excrete.

Vitamin A: Night blindness was cured in ancient Greece either by ingesting cooked liver or by applying topically to the eyes the juice squeezed from cooked liver. McCollum and Davis called it fat-soluble A.

Clinical Manifestation of Deficiency of Vitamin A

Vitamin A deficiency may be caused by a decreased dietary intake; however, this is usually seen in very severe malnutrition. It may also occur secondary to fat malabsorption. In the eye symptoms are progressive. The earliest sign of deficiency is a loss of sensitivity to green light, followed by impairment to adaptation to dim light, followed by night blindness. This is reversible. More prolonged deficiency leads to xerophthalmia: keratinization of the cornea and conjunctiva and skin and blindness. If untreated, keratomalacia develops, causing corneal ulceration and formation of opaque scar tissue (cataracts); this causes irreversible blindness. Vitamin A is intimately involved with differentiation of epithelium and experimentally produced deficiencies have retarded epithelization, closure of wounds, the rate of collagen synthesis and the cross-linking of newly formed collagen.

Thaimine (Vitamin B_1)

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

Clinical Diagnosis of Deficiency

Severe thiamin deficiency is called beriberi. It affects principally the cardiovascular, muscular, and nervous systems. Beriberi may manifest in one of the three forms. In the dry beriberi, there is loss of appetite, tingling and numbness in hands and legs and a dropping of the feet. In wet beriberi, there is dropsy, palpitation and breathlessness and weakness of heart muscle leading to heart failure and edema of the legs. Infantile beriberi, caused by inadequate thiamine in breast milk, is characterized by dyspnoea, cyanosis and cardiac failure. In the alcoholic, a severe thiamin deficiency may lead to Wernicke's syndrome, which is characterized by confusion, paralysis of eye muscles, and loss of memory. Peculiar gait and foot and wrist drop are seen in advanced cases. The possible oral manifestations of a deficiency of this vitamin are hyper sensitivity of the oral mucosa, glossitis and loss or diminution of taste[1], minute vesicles (simulating herpes) on the buccal mucosa, under the tongue or on the palate; and erosion of oral mucosa [4].

Therapy: A good diet and administration of 5 to 10 mg of thiamin hydrochloride three times a day will help the usual cases of nutritional polyneuropathy (a disease involving several nerves). Alcoholics or others who may develop beriberi should also have some multivitamin B complex tablet supplement in a therapeutic dose (which is two or three times the supportive dose), brewer's yeast, liver extract, or wheat germ. Mechanism of action of thiamin. Thiamin pyrophosphate (TPP), the active form of thiamin, acts as coenzyme for pyruvate dehydrogenase and α-ketoglutarate dehydrogenase reactions in the TCA cycle and for transketolase in the pentose phosphate pathway.

Riboflavin (Vitamin B_2)

Chemical research on riboflavin was begun in 1879 by an English food chemist, Winter Blyth, who observed some pale yellow material in the whey of milk, which he called lactoflavin. A yellow enzyme essential for cell respiration was isolated from yeast by Warburg and Christian in 1932. But it was Kuhn and his co-workers who reported in 1935 on the synthesis of riboflavin and the relation of its activity to green fluorescence. The latter group established that lactoflavin and the vitamin are the same. This was the first evidence that a vitamin functions as a coenzyme [5].

Clinical Diagnosis of Deficiency

The basis for a riboflavin deficiency is an inadequate dietary intake, most often seen in alcoholics and the economically deprived. Also, a severe gastrointestinal disease that causes vomiting and hypermotility of the gastrointestinal tract can produce this deficiency, as can polyuria resulting from uncontrolled diabetes. Clinically, riboflavin deficiency may be found in conjunction with a lack of other B vitamins such as pyridoxine, niacin, and folic acid. The reason for the multiplicity of vitamin deficits is that dietary deficiencies tend to be multiple because the food sources are similar.

Therapy: In the treatment of riboflavin deficiency, a good diet of liver, meat, eggs, and enriched cereals plus a special emphasis on a quart of milk daily is a first consideration. Also, a therapeutic multivitamin capsule containing at least 5 mg of riboflavin should be taken two or three times a day.

Niacin: Niacin should be used as the generic description for derivatives exhibiting qualitatively the biological activity of nicotinamide. 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. Shortly thereafter niacin was shown to be effective in the prevention and treatment of pellagra.

Clinical Diagnosis of Deficiency

Pellagra is a niacin deficiency disease caused by a primary inadequate dietary intake or by a secondary conditioning (systemic) factor such as alcoholism, gastrointestinal disease, hyperthyroidism, or infection that inhibits absorption and use of nutrients. Pellagra is characterized by dermatitis, diarrhea, dementia, glossitis, gingivitis, and generalized stomatitis. Early symptoms of pellagra are similar to those produced in other B complex deficiency states: weakness, persistent fatigability, irritability, headache, and depression. Soreness and inflammation of the tongue (glossitis) and mouth (stomatitis) aggravated by highly seasoned foods is also a frequent complaint. The tongue is sore, swollen, scarlet in color and smooth.

Therapy: In treatment of mild niacin deficiency, administration of 50 mg of nicotinamide orally three times a day is recommended. For the treatment of pellagra, 300 to 500mg of nicotinamide daily in divided doses, as well as supplements of other nutrients that are frequently deficient, is prescribed.

Pantothenic Acid

The discovery of pantothenic acid stemmed from investigation of yeast growth factor and a liver filtrate

factor. It was named pantothenic acid by R.J. Williams in 1938 because of its distribution in many foods.

Clinical Diagnosis of Deficiency

In humans, a deficiency is rare and can be induced experimentally. The symptoms are fatigue, sleep disturbances, headaches, malaise, nausea, and abdominal stress. Burning, prickling sensation (paresthesia) of the hands and feet, cramping of the leg muscles, and impaired coordination are additional findings.

Therapy: No definite therapeutic regimen for dealing with pantothenic acid deficiency has been presented. Multivitamin B complex preparations usually contain pantothenic acid.

Pyridoxine (Vitamin B_6)

Vitamin B_6 is not a single vitamin but rather a group of metabolically and functionally interrelated pyridines namely, pyridoxine, pyridoxamine (the amine of pyridoxine), and pyridoxal. Each of these substances is widely distributed in foods and is present in both free and bound forms.

Clinical Diagnosis of Deficiency

A frank deficiency of Vitamin B_6 in humans is rare because of its widespread distribution in natural foodstuffs. An experimental deficiency produced cheilosis, glossitis and stomatitis, and an itching and burning dermatitis with redness in the nasolabial folds. Subjective symptoms accompanying these signs were loss of appetite, nausea, drowsiness, and peripheral neuropathy. These signs and symptoms resemble those seen in deficiencies of riboflavin, niacin, and thiamin, which attests to the close metabolic relationship of the B complex vitamins.

Therapy: When primary deficiency of this vitamin is suspected in an adult, a daily dosage of 10 mg is given. In iron-resistant hypochromic anemia, doses up to 100 mg/day have been given. Certain medications, such as isoniazid and penicillamine, produce a need for a supplement of vitamin B_6.

Vitamin B_{12}(Cobalamin)

Pernicious anemia leading inevitably to a patients 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. These two factors had to be given at the same time to produce an effective result. Castle concluded that the disease was not dietary in origin but was caused by a failure of the stomach to secrete the intrinsic factor for the absorption of the dietary extrinsic factor. In 1948 Shorb reported that the liver extracts used to overcome anemia also had growth-promoting properties for a Lactobacillus species. Using this microorganism, Rickes and associates were able to isolate the red crystalline cyanocobalamin (vitamin B_{12}).

Clinical Diagnosis of Deficiency

Clinically, pernicious anemia is insidious in onset and characteristically fluctuates, with remissions and relapses. The anemia results from impaired DNA synthesis due to a block in purine and thymidine biosynthesis. The usual complaints are weakness, numbness, and tingling in the extremities. Patients may have difficulty in walking and in coordination of movements; vibratory sense may be absent. These neurological signs are a major distinguishing characteristic of pernicious anemia. The patient also has a bright red, smooth, sore, and burning tongue resulting from an atrophic glossitis. There are atrophic changes in the alimentary tract, and in the central nervous system, there is myelin degeneration of the spinal cord.

Therapy: A deficiency caused by inadequate ingestion of the vitamin (i.e., strict vegetarian patients) can be effectively treated by oral ingestion of 0.001 to 0.003 mg (1 to 3 mg) of the vitamin daily.

Vitamin C (Ascorbic Acid)

The first accurate and scientific discussion of the cause and cure of scurvy was made in 1953 by a Scottish surgeon, James Lind, in his first edition of "A Treatise on the scurvy". He described scurvy as the disabling disease that affected the 110 men who sailed from France to Newfoundland in 1536 under the command of the French explorer Jacques Cartier. A variety of factors can influence vitamin C requirements. Cigarette smokers have been shown to 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.

Deficiency of Vitamin C

Severe vitamin C deficiency in humans results in scurvy, a dis-ease characterized by hemorrhagic

diathesis and retarda-tion of wound healing. Clinical manifestations of scurvy include hemorrhagic lesions into the muscles of the extremities, the joints, and sometimes the nail beds; petechial hemor-rhages, often around hair follicles; increased susceptibil-ity to infections; and impaired wound healing. Bleed-ing, swollen gingiva and loosened teeth are also common features of scurvy. Vitamin C deficiency (scurvy) results in defective for-mation and maintenance of collagen, retardation or ces-sation of osteoid formation and impaired osteoblastic function. Vitamin C deficiency is also characterized by increased capillary permeability, susceptibility to trau-matic hemorrhages, hyporeactivity of the contractile ele-ments of the peripheral blood vessels, and sluggishness of blood flow.

Diagnostic Laboratory Findings

Normal plasma ascorbic level is approximately 0.6 to 1.5 mg / 100ml; RBC contains twice this much, and WBC 20 to 40 times as much. The diagnosis of scurvy is made when the concentration of ascorbic acid in white cell-platelet layer of blood decreases to 2 mg / 100g or less. Scurvy is diagnosed by its symptoms, including ecchymosis of the skin, loose attachment of the periosteum, subperiosteal hematomas, bleeding into joint spaces, gingivitis, hemorrhages, opportunistic bacterial infections, & impaired wound healing (Vaxman et al 1990).

Vitamin D: In 1918 Sir Edward Mellanby demonstrated that rickets in puppies was a nutitional deficiency disease that was curable by the administration of cod liver oil. 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). However, it would cure rickets in the animals, a condition that was originally produced when they were fed diets containing abnormal calcium to phosphorus ratios. Because this was the fourth vitamin to be discovered, it was called vitamin D.

Recommended Dietary Allowance

For Indians no dietary recommendations are made since adequate vit D is obtained through exposure to sunlight.

Sources: There are three types of vitamin D, but only two; vitamin D_2 and vitamin D_3 are of nutritional importance. These compounds are fat soluble and stable to cooking, processing, storage, and acids but sensitive to light. Vitamin D_2 (ergocalciferol) is derived from the provitamin ergosterol, which is present in plants, especially in fungi and yeasts. It is a synthetic form produced by irradiating ergosterol with ultraviolet light. One milligram of vitamin D_2 is the equivalent of 40,000 IU. Vitamin D_3 (Cholecalciferol), the naturally occurring form of the vitamin in animal tissues, is produced in the skin.

Vitamin E: It has become one of the most misused, abused and controversial nutrients because of many erroneous claims for its healing powers in a variety of ailments. In 1922 a research team discovered that female rats aborted and male rats were sterile unless lettuce, wheat germ, and dried alfalfa were added to their diet of casein, cornstarch, lard butter, and yeast. In 1924 another researcher came to the same conclusion, and because the then four known vitamins were called A, B, C and D he suggested that this "fertility vitamin" be called vitamin E.

Clinical Findings of Vitamin E Deficiency

Vitamin E deficiency is rare in human beings. Vitamin E deficiency may cause impaired neuromuscular function sometimes seen in patients with disorders that interfere with absorption or transport of the vitamin.

Effect of Infection on Vitamins

Infection causes negative balances of vitamins A, C and B-complex. This decrease may be a result of a result of malabsorption, losses in diarrhea and / or increased needs to combat infection. Corticosteroids interfere with absorption of vitamin D and folic acid.

Future Considerations

Vitamin Q – A proposed Addition [6]

Recently a team of researchers at the 'Institute of Physical and Chemical Research' in Tokyo published a report claiming that they have isolated PQQ (pyrrololoquinoline quinine), with chemical properties similar to vit. B_6. They believe this discovery is the first new vitamin since 1948 and will include it with vitamins B_2 and B_3, calling it 'Vitamin Q'. Currently PQQ is not included in multivitamin / mineral supplements, but it can be consumed in the diet with parsley, green peppers, kiwi fruit, papaya, spinach, tofu, tea and certain meats.

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