



Vitamin C and Oral Health: A Review

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Ascorbic acid, Periodontitis, Scurvy, Vitamin C.

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ABSTRACT Vitamin C is an essential dietary nutrient required as a co-factor for many enzymes. Ascorbic acid is a reduced form of this vitamin, which is an effective antioxidant owing to its high electron-donating power and readily converts back to the active reduced form. In body tissues and fluids, the concentrations of the vitamin C are maintained by interplay between absorption from intestine, cellular transport and excretion. Very small amount of vitamin C is needed to prevent scurvy. Evidence has shown that oxidative damage is a root cause of or at least associated with many diseases, the clinical role of vitamin C becomes very important. Although clinical trials have not confirmed this, but still vitamin C may prove to be effective in people with certain diseases or conditions. However, the evidence that ascorbic acid acts as an important antioxidant in many body tissues is convincing. In healthy individuals, amounts taken greater than the RDA are not helpful.

Introduction

Vitamin C or L-ascorbic acid, or simply ascorbate (the anion of ascorbic acid), is an essential nutrient of the diet for humans and some other animal species. Vitamin C consists of a number of vitamers that have vitamin C activity in animals including ascorbic acid and its salts and many oxidized forms of the molecule like dehydro-ascorbic acid. Ascorbate and ascorbic acid, both are present naturally in the body when any of these is introduced into cells as the forms interconvert according to pH. In at least eight enzymatic reactions, Vitamin C is a cofactor including various collagen synthesis reactions. When these synthetic reactions become dysfunctional, they cause the most severe symptoms of scurvy¹.

Ascorbate may also act as an antioxidant agent. The enantiomer D-Ascorbate, which is not found in nature, has identical antioxidant activity to L-Ascorbate but very less vitamin activity. Ascorbic acid is a weak sugar acid which is structurally related to glucose. It can be found only at low pH in biological systems, but in neutral solutions having pH above 5 it is predominantly found in its ionized form i.e. ascorbate.

Biological significance

The biological role of ascorbate is to act as a reducing agent by donating electrons to various enzymatic and a few non-enzymatic reactions. The one and two-electron oxidized forms of vitamin C (semi-dehydro-ascorbic acid and dehydro-ascorbic acid respectively) can be reduced in the body by glutathione and NADPH- dependent enzymatic mechanisms. Thus, the presence of glutathione in cells and extracellular fluids helps maintain ascorbate in a reduced state.

Vitamin C and oral health

Vitamin C has diverse functions in the body at a chemical and structural level. For example, a major function of ascorbic acid is its involvement in the synthesis of collagen fibres from proline via hydroxyproline. Other metabolic reactions for which vitamin C is required are the hydroxylation of lysine into hydroxylysine in collagen, the conversion of folic acid to the active form of folic acid in-vivo, the formation of steroids by the adrenal gland, the oxidation of lysine in muscle proteins, the microsomal metabolism of drugs and the pro-

tective action on enzymes such as p-hydroxyphenylpyruvic acid oxidase.²

At the tissue structural level vitamin C is involved with the synthesis of intercellular substances and the collagen fibres of various forms of the connective tissues in which collagen forms a part, for example organ capsular/trabecular, tendinous and fascial tissue, the matrix of calcified tissues such as bone and teeth and the endothelial cells of the entire vascular system, including capillaries.

In a normal adult man of 70 kg mass and with a vitamin C reserve of 1500 mg, general scurvy will manifest when the vitamin C reserve is reduced to 300 mg. This can be caused by a reduction of 45 mg vitamin C per day for 60 days on a vitamin C-free diet. Scurvy is an avitaminosis resulting from lack of vitamin C, because without this vitamin, the synthesized collagen is very unstable to perform its normal function.³ Features of Scurvy include the formation of brown spots on the skin, spongy & swollen gums and bleeding from all mucous membranes. The spots are more abundant on thighs and legs; the person looks pale, feels depressed and is partially immobilized. In advanced stages of scurvy, there are open, suppurating wounds, loss of teeth and eventually, death.

In scurvy, administration of L-ascorbic acid (in a dose of about 1 g/d for adults) will cause rapid disappearance of subcutaneous hemorrhages and nearly complete reversal of symptoms. Gingivitis is not caused by vitamin C deficiency per se, but if the gingivitis exists in a scorbutic patient it seems to be caused by local irritants. The legendary association of severe gingival disease with scurvy led to the incorrect presumption that vitamin C deficiency is a primary factor in the causation of gingivitis and periodontal disease. It is now believed that blood ascorbic acid levels bear no relationship to the incidence or severity of gingivitis or periodontal disease in non-scorbutic patients. Furthermore, vitamin C deficiency does not cause periodontal pockets; local irritating factors are required for pocket formation to occur.⁴ It has been shown that smokers who have diets poor in vitamin C are at a higher risk of lung-borne diseases than those smokers who have higher concentrations of vitamin C in the blood.

Avitaminosis C is associated mainly with defective collagen synthesis, and symptoms such as the failure of wound healing or the rupture of capillaries are due to intrinsic intercellular weakness with lack of connective tissue support of the capillary walls. This vascular fragility leads to a bleeding tendency which produces petechiae and ecchymoses and is confirmed by a positive Hess test.

Haematuria, epistaxis, subperiosteal bleeding, haemarthrosis and bleeding into muscles and deeper tissues also occur. The general discoloration resulting from the bleeding and blood breakdown is called 'scurvy siderosis'. Hemorrhage under the nails with onycholysis may also occur. The hair follicles may be hyperkeratotic with vascular congestion and may show perifollicular hemorrhages. There will be general lassitude and an increased susceptibility to infections.

On exposure to certain drugs and toxins and usually also on exposure to stress, more vitamin C is needed to sustain normal plasma levels in order to meet the altered metabolic demands. Gingival manifestations of disease in scurvy are most severe when oral hygiene is poor. The gums do not become involved if the teeth are unerupted or if the patient is edentulous, although sub-epithelial hemorrhages do occur. The signs of general scurvy, such as prominence of the hair follicles because of keratin plugging, dermal perifollicular ecchymoses, aching legs, joint effusions, lethargy, vasomotor instability or pitting edema of the ankles, will appear together with gingival lesions in the dentate patient. The gingivae become hyperemic with a tendency to bleed on interdental papilla where disintegration of marginal epithelium occurs.³

Recent medical and dental research cited the prevention of free radical mediated diseases by using specific anti-oxidants, protective role of anti-oxidant supplementation in prevention of precancerous lesions.⁶ It has been observed that anti-oxidant vitamin C supplementation for six weeks was helpful in patients with various dental problems.

Deficiency of vitamin C as a risk factor in progression of periodontitis

Scurvy and periodontitis both manifest gingival bleeding but constitute separate entities. The various periodontal diseases are caused by oral micro-organisms in dental plaque, the body's reaction to which is strongly influenced by inadequate or impaired functioning of leucocytes and monocytes. Although various infections and systemic diseases cause gingival bleeding, avitaminosis C does not cause commonly encountered periodontal disease, but it will aggravate the already established periodontitis. So, Vitamin C should not be used for cure or prophylaxis of periodontitis in healthy well-nourished individuals.⁵

Studies show that patients with periodontitis are characterized by low plasma levels of vitamin C than the normal range, especially smokers. The intake of citrus fruits such as grapefruit leads to an increase in plasma levels of vitamin C and improved scores for sulcus bleeding index. However, long term studies are required to determine whether other periodontal outcomes improve with such supplementation especially in smokers.

Erosion of dental enamel

Chewable vitamin C tablets, used daily, have been reported to lead to severe erosion of dental enamel because of the acidity and abrasiveness of these products, but the practical clinical significance of this effect has not been established. The dental enamel erosion is brought about by the high acidity of ascorbic acid (pH of 2.8), therefore, if chewable tablets of vitamin C are properly formulated to a pH of approximately 4 to 5 using sodium ascorbate or another buffering agent, erosion of dental enamel should not be a problem. Chewable vitamin C should not be formulated and marketed without a buffering formulation.⁷

Conclusion

Vitamin C (ascorbic acid) is an indispensable cofactor in the hydroxylation of amino acids such as proline and lysine. It is required and essential for collagen synthesis and connective tissue integrity. It is involved in the biosynthesis of histamine, carnitine and several adrenal steroids. It promotes iron absorption and mobilization, helps in metabolism of tyrosine, folate, and xenobiotics. When vitamin C intake is below a critical level (10 mg/d) for longer periods, a clinical condition known as scurvy will ensue featuring failure of wounds to heal, petechial hemorrhages, bleeding gums, follicular hyperkeratosis and related abnormalities.

The dietary recommendation are: RDA 3000 mg Vitamin C i.e. 1000 mg three times daily. According to the Vitamin C Foundation recommendations every man, woman and child over the age of 3 years should consume at least 3 g (3000 mg) vitamin C daily in order to enjoy optimum health.

REFERENCE

1. Schleier RL, Carroll MD, Ford ES. Serum vitamin C and the prevalence of vitamin C deficiency in the United States: 2003–2004 National Health and Nutrition Examination Survey. *Am J Clin Nutrition* 2009; 90: 1252–63. | 2. Jacob RA, Sotoudeh G. Vitamin C function and status in chronic disease. *Nutr Clin Care* 2002; 5: 66–74. | 3. Hathcock JN. Vitamin and Mineral Safety. 2nd edition 2004. | 4. Touyz LZG. Vitamin C, oral scurvy and periodontal disease. *SA. Med J* 1984; 65: 838–42. | 5. Pussinen PJ, Laatikainen T, Alfthan G, Asikainen S, Jousilahti P. Periodontitis is associated with low concentration of vitamin C in plasma. *Clin Diagn Lab Immunol* 2003; 10: 897–902. | 6. O'Leary TJ, Rudd KD, Crump PP, Krause RE. The Effect of ascorbic acid supplementation on tooth mobility. SAM-TR-68-112. Tech Rep SAM-TR. 1968; 1-6. | 7. Cheraskin, E. The Vitamin C Controversy: Questions and Answers. 1st edition. Wichita, Bio-Communications Press 1988. |