International Journal of Academic Health and Medical Research (IJAHMR)
ISSN: 2643-9824
Vol. 5 Issue 1, January - 2021, Pages: 34-37

Functions of Vitamin C

Ermatov Jalol Nurali ugli and Oltiboyeva E`tiborxon Baxrom qizi

Samarkand State Medical Institute,
Uzbekistan, student
mailjaloliddin0088@gmail.com

Abstract*: Vitamin C is an important component of diet. It has been found to help in healing of wounds, ulcers, trauma and burns, in allergic conditions, common cold and during labor. Deficiency of vitamin C has an important impact on gingival and periodontal health. The aim is to review the concepts of effects of vitamin C deficiency and current methods available for the detection of vitamin C deficiency. Analysis of literature indicates that the microscopic signs of vitamin C deficiency are quite different from those that occur in plaque induced periodontal disease in humans.*

Keywords: Ascorbic acid, gingivitis, periodontitis, polymorphonuclear neurophils.

1. INTRODUCTION

It is water soluble and is easily destroyed by heat, alkali and storage. In the process of cooking 70% of the vitamin c is lost. Some lower mammals like rats can synthesise the vitamin from glucose by the uronic acid pathway. Man, monkey and guinea pig lack the enzymes necessary for the synthesis. They cannot convert ketogluconolactone to ascorbic acid. Hence the entire human requirement must consequently be supplied by diet. It is absorbed readily from the small intestine, peritoneum and subcutaneous tissues. It is widely distributed throughout the body. It is excreted as diketogluconic acid and oxalic acid in urine. Since vitamin C is a strong reducing agent Benedicts test will be positive in the urine sample. Ascorbic acid is partly excreted unchanged and partly as oxalic acid. Most of the oxalates in urine are derived from ascorbic acid. The ascorbic acid level varies between 0.7 to 1.2mg/100ml of plasma and 25mg/100 cc of WBC. A low level in blood is noted in women taking contraceptives and also in chronic alcoholics. Under normal dietary intake of 75 to 100mg, 50-70% are converted to inactive compounds.25-50% is excreted in urine as such. It is secreted in milk. It is widely distributed in plants and animal tissues. In animal tissues usually it is not stored. But the highest concentration in metabolically high active organs eg: adrenal cortex, corpus leuteum, liver etc. Dietary sources: These are chiefly vegetable sources .Good sources are citrus – orange, lemon, lime etc. other frits like papaya, pineapple, banana, strawberry. Amongst vegetables, leafy vegetables like cabbage and cauliflower, germinating seeds, green peas and beans, potatoes and tomatoes. Amla is the richest source.

The outstanding property of this vitamin is its capacity for oxidation-reduction between ascorbic acid and dehydroascorbic acid. Most of the physiological properties of the vitamin could be explained by this redox system. Ascorbic acid is necessary for the normal production of supporting tissues of mesenchymal origin such osteoid, dentine, collagen and intercellular cement substance of capillaries. Ascorbic acid promotes collagen formation through its own action on post-translational hydroxylation of proline and lysine residues. The collagen is synthesized by fibroblasts intracellularly as a large precursor, called procollagen. It is then secreted. The extracellular procollagen is cleaved by specific peptidases to form tropocollagen.

2. DEFICIENCY MANIFESTATIONS

Gross deficiency of Vitamin C leads to Scurvy. The disease is likely to occur in infants 6-12 months age, the period in which weaning from breast milk is started. The eponym of infantile scurvy is Barlow's disease. In scurvy, the main defect is failure to deposit intercellular cement substance. Capillaries are fragile, tendency to haemorrhages [Petechial, subcutaneous, subperiosteal and even haemorrhages occur under minor pressure. Collagen is abnormal and intercellular cement substance is brittle. Haemorrhage may occur in the conjunctiva and retina. Internal bleeding may be seen as epistaxis, hematuria or malena. In severe cases of scurvy the gingiva is swollen, painful and spongy. In bones, the osteoblasts fail to form osteoid. Without the normal ground substance, the deposition of bone is arrested. The resulting scorbutic bone is weak and fractures easily. There may be subperiosteal bleeding as well as hemorrhages into the joint cavities. Painful swelling of joints may prevent locomotion of the patient. Microcytic hypochromic anemia is also seen. Poikilocytosis and anisocytosis are also seen. Anaemia is due to

- Loss of blood by hemorrhage.
- Decreased iron absorption.

- Decreased availability of tetrahydrofolate.
- Accumulation of methaemoglobin inside the erythrocytes.

Bachelor scurvy: Seen in elderly bachelors and widowers, who may prepare their own foods are particularly prone to Vitamin C deficiency.

Oral manifestations: Bleeding, swollen gingiva and loosened teeth. Gingival enlargement is marginal. Gingiva is bluish red soft and friable and has a smooth, shiny surface. Hemorrhage occurring either spontaneously or on slight provocation and surface necrosis with pseudomembrane formation are common features.

Detection of deficiency in man:

- Prompt improvement following administration of Vitamin C.

- Determination of concentration of ascorbic acid in blood.

- **Urine ascorbic acid saturation test**: By administration a test dose of 5mg/lb body wt. If 50% or more is excreted in 24hrs, the individual has no deficiency of the vitamin.

- **Intradermal test**: Consists of intra dermal injection of 2, 6-dichlorophenol indophenol and determination of the time required for decolorisation that is reduction of the dye

- **Torniquet test**: [Capillary resistance or fragility test] A sphygmomanometer cuff is applied around the arm and inflated so that it compresses the venous flow. In a short time, appearance of several Petechial haemorrhages on the fore arm skin indicates deficiency.

Requirement. Daily intake of 100mg/day is adequate in normal adults:

- Adults 75 mg/day;

- Infants 30mg/day;

- Adolescence 80mg/day;

- Pregnant women 100mg/day;

- Lactating women 150mg/day.

Emperical uses of Vitamin C:

-Apart from treatment of scurvy, Vitamin C has been used emperically in many other conditions viz in control and treatment of infectious diseases;

-Has been found to help in healing of wounds, ulcers, trauma and burns, in allergic conditions, common cold and coryza, during labor Vitamin C given in doses of 150-200mg produces oxytocic action. -In methhemoglobinaemia it may be used for its reducing property.

Histopathology. Gingiva has a chronic inflammatory cellular infiltration with a superficial acute response. There are scattered areas of hemorrhage, with engorged capillaries. Marked diffuse edema, collagen degradation, and scarcity of collagen fibrils or fibroblasts are striking findings.

3. EPIDEMIOLOGIC STUDIES

Several studies in large populations have analyzed the relationship between gingival or periodontal status and ascorbic acid levels. These studies used different methods for biochemical analysis of vitamin C and various indices for assessment of periodontal changes. They were made in different persons of different socioeconomic status, races and various ages. All the epidemiologic studies failed to establish a causal relationship between the two. Mega doses of vitamin C have also been found to be unrelated to better periodontal health.

Gingivitis with enlarged, hemorrhagic bluish-red gingiva is described as one of the classic signs of deficiency of vitamin C. But gingivitis is not caused by vitamin C deficiency per se. Acute vitamin C deficiency does not cause or increase the incidence of gingival inflammation but it does "increase its severity." Gingivitis in vitamin C deficient patients is caused by bacterial plaque. Vitamin C deficiency may aggravate the gingival response to plaque and worsen the edema, enlargement and bleeding. Correcting the deficiency may reduce the severity, but gingivitis will remain as long as bacterial factors are present.

Periodontitis. Changes in the supporting periodontal tissues and gingiva in vitamin C deficiency have been documented extensively in experimental studies. Acute vitamin C deficiency results in edema and hemorrhage in periodontal ligament, osteoporosis of alveolar bone and tooth mobility. Hemorrhage, edema and degradation of collagen fibres occurs in gingiva. Vitamin C deficiency also retards gingival healing. Periodontal ligament fibres least affected are those below the junctional epithelium and above alveolar crest. Vitamin C deficiency does not cause periodontal pockets; local bacterial factors are required for pocket formation. However acute vitamin C deficiency accentuates the destructive effect of the gingival inflammation on the underlying periodontal and alveolar bone. The exaggerated destruction results partly from an inability to marshal a defensive delimiting connective tissue barrier reaction to inflammation and partly from destructive tendencies caused by the deficiency itself, including inhibition of fibroblast, osteoblast formation and differentiation as well as impaired formation of collagen and mucopolysaccharides ground substance. Experimental studies conducted in humans failed to show the dramatic clinical changes that have traditionally been described in scurvy. A case report by Charbeneaue and Hurt, (1983) showed worsening of pre-existing moderate periodontitis with development of scurvy. Retrospective analysis of 12,419 adults studied in found there was a weak but statistically significant dose-response relationship between the levels of dietary vitamin C intake and periodontal disease in current and former smokers as measured by clinical attachment. This suggests that vitamin C deficiency has its greatest impact on periodontal disease when pre-existing disease and other codestructive factors are present.

Gingival enlargement in Vitamin C deficiency. It is essentially a conditioned response to bacterial plaque. Acute vitamin C deficiency does not by itself cause gingival inflammation, but it does cause hemorrhage, collagen degradation, and edema of gingival connective tissue. These changes modify the response of gingiva to plaque to the extent that the normal defensive delimiting reaction is inhibited and the extent of inflammation is exaggerated. The combined effect of acute vitamin C deficiency and inflammation produces the massive gingival enlargement as seen in scurvy.

4. RESULTS AND DISCUSSIONS

Evidence for role of vitamin c in periodontal disease. Animal studies

The early experimental studies of ascorbic acid deficiency on the periodontal tissues in both monkeys and guinea pigs demonstrated that periodontal pathology could be induced by omitting the vitamin from the diet. Boyle (1937) believed that the deficiency produced atrophic changes in the gingiva and underlying bone in guinea pigs. Others have described changes similar, but not identical to those of plaque induced periodontitis in humans. They include bleeding and flabby gingiva, osteoporosis and resorption of alveolar bone, rupture of periodontal ligament fibres, widening of periodontal ligament space and increased tooth mobility (Glickmann, 1948; Turseky and Glickmann, 1954; Hunt, 1941). Driezen and Stone (1961), pointed out that plaque and calculus need to be present before pocket formation. This finding, when considered with the results of later research, is particularly significant. Alvares et al., (1981) failed to induce spontaneous gingivitis or periodontitis after 23 wks feeding with an ascorbate deficient diet. However when plaque-associated lesions were induced, the pocket depths were significantly greater in animals with subclinical ascorbate deficiency than in pair fed controls. This would indicate that indicate ascorbic acid deficiency is unlikely to be an initiating factor in inflammatory periodontal disease although it may enhance a plaque induced lesion that is already present (Woolfe et al., 1980).

Human studies

The findings of numerous studies of vitamin C and human periodontal disease have failed to produce clear scientific evidence of a relationship between these variables. One group of proponents (Barahal and priestman, 1942; Kyhos et al., 1944) concluded that when the plasma levels of vitamin C are lowered then some degree of gingivitis or periodontal disease will ensue. Crandon et al., (1940) did perhaps the classical and perhaps most widely quoted study. He consumed a vitamin C deficient diet for six months and only after the fifth month was a slightly boggy gingival appearance found. Irregularities of the lamina dura appeared at this time, although the oral changes occurred almost 2 months after skin lesion were detected. These observations have been contradicted by the findings of both extensive epidemiological and longitudinal clinical trials which did not find a significant correlation between ascorbic acid status and either gingival health or periodontal destruction (Barros et al.,1963; Russell et al., 1963; Buzina et al., 1985; Hodges et al., 1971). Nevertheless, although low plasma levels of ascorbic acid may not alone initiate periodontal lesions and patients with severe scurvy can have healthy gingiva, it is possible that a deficiency of vitamin C will exacerbate an existing gingivitis (Hodges et al., 1969). There are also conflicting results from studies in which supplementation

with ascorbic acid has been used, either alone or combined with local measures. Cohen, (1955) showed that in the absence of local periodontal treatment a 500 mg oral dose of ascorbic acid improved markedly the gingival condition in teenagers after 90 days. Cowan, (1976) conducted a similar trial with larger doses of ascorbic acid 1-3mg and noticed reduction in irregularities of laminadura of young adults and concluded that this is due to consolidation of the collagen at the alveolar bone / cementum interface. Aurer-kozelj et al., (1982) found daily supplements of 70mg vitamin C for 6 wks produced marked changes in gingival epithelium and connective tissue. Desmosomal junctions between the epithelial cells became longer and the contact surface between the cells increased. They suggested that the number of collagen producing fibroblasts increases with vitamin C intake as excreted collagen was identified in large numbers of bundles in the vicinity of the cells. In contrast to the studies that have shown that ascorbic acid supplements are beneficial in maintaining a healthy periodontium, a number of reports have failed to provide such a evidence (Parfitt and Hand, 1963) conducted controlled trials and concluded that neither large dose/short term nor small dose /long term regimens have proved to be effective. Flotra et al., (1969) found that topical application of ascorbic acid by rinsing has no significant action on plaque formation or gingivitis. They also found that ascorbic acid administration along with local treatment was beneficial. Linghorne et al., (1946) showed that a high dose (375mg) of vitamin C when given alone has no effect on established inflammation. When smaller doses 75mg /day were used as an adjunct to scaling and root planing, then the incidence of recurrence of inflammation was reduced (El Ashiry et al., 1964). Combination therapy has also been shown to be more effective than prophylaxis or ascorbic acid alone in reducing gingivitis. Ismail et al., (1983) advocated that treatment of ascorbutic patients with periodontal disease must be considered outdated. Local treatments such as scaling and Root planing are more effective and more predictable than dietary supplementation with ascorbic acid. Dietary supplementation is no longer considered to be associated with improved periodontal health and is certainly not fulfilling an important principle of basic medicine.-removal of cause. Briggs et al., (1973) proposed that extensive intake of ascorbic acid may precipitate problems such as renal calculi and diarrhea. Vitamin C also interferes with the action of drugs including warfarin and aspirin (Rosenthal et al., 1971; Loh and Wilson, 1975).

CONCLUSION

In summary analysis of literature indicates that the microscopic signs of vitamin C deficiency are quite different from those that occur in plaque induced periodontal disease in humans. Patients with acute or chronic vitamin C deficiency states and no plaque accumulation show minimal, if any changes in their gingival health status.

1. Alvares O, Siegel I. Permeability of gingival sulcular epithelium in the development of scorbutic gingivitis. *J oral pathology*, 1981, 10, 40-48 [PMID: 6782209]

2. Aurer-Kozelj J, Kralj-Klobucar N, Buzina R, Bacic M. The effect of ascorbic acid supplementation on periodontal tissue ultra structure in subjects with progressive periodontitis. *Int J Vitam. Nutr. Res*. 1982, 52(3), 333-41 [PMID: 6757168]

3. Barahal HS, Priestman MG. The relationship of vitamin C deficiency to dental and gingival conditons in mental patients. *Am. J. Psychiatry*, 1942, 98, 823

4. Briggs MH, Garcia-Webb P, Daviss P. Urinary oxalate and vitamin-C supplement. *Lancet*, 1973, 2, 201

5. Boyle PE. Experimental alveolar bone atrophy produced by ascorbic acid deficiency. *Proc Soc Erp Bioi Med*., 1937, 36, 733-735

6. Suboticanec-Buzina K, Buzina R, Brubacher G, Sapunar J, Christeller S. Vitamin C status and physical working capacity in adolescents. *Int J Vitam Min Res*. 1984, 54(1), 55-60

7. Carpenter KJ. The History of Scurvy and Vitamin C. Cambridge, England: Cambridge University Press, 1986

8. Chatterjea MN. Textbook of Medical Biochemistry. Jaypee Brothers Publishers, 2004 9. Crandom J, Lund CC, Dil DB. Experimental human scurvy. *New England J. Med*. 1940, 23, 353