

Impact of Nutrition on Periodontal Health: The victuals and periodontium

Dr. Sonali Sarkar¹, Dr. Dhirendra Kumar Singh², Dr. Md. Jalaluddin³

¹*PGT, Department of Periodontics & Oral Implantology, Kalinga Institute of Dental Sciences, KIIT
Deemed to University, Bhubaneswar*

²*Reader, Department of Periodontics & Oral Implantology, Kalinga Institute of Dental Sciences, KIIT
Deemed to be University, Bhubaneswar*

³*Professor, Department of Periodontics & Oral Implantology, Kalinga Institute of Dental Sciences, KIIT
Deemed to be University, Bhubaneswar*

Abstract - Periodontal health is influenced by a number of factors such as oral hygiene, genetic and epigenetic factors, systemic health, and nutrition. A balanced diet is crucial for the maintenance of periodontal health as suggested by various number studies. Along with that, the impact of nutritional supplements and dietary components have been known to bring a positive effect in the healing of periodontium post surgeries. Studies have attempted to find a correlation between tooth loss, periodontal health, and nutrition. Moreover, bone formation and periodontal regeneration are also affected by numerous vitamins, minerals, and trace elements. The focus of this review is to critically estimate the currently available data on diet and maintenance of periodontal conditions in disease and health. The effects of nutritional intervention studies to improve the quality of life and well-being of patients with periodontal disease have been discussed.

Index Terms - Nutrition, Periodontal Health, Vitamins, Minerals

INTRODUCTION

WHO describes ‘nutrition’ as the process and system by which any living organism can live and convert extraneous solid and liquid substances, which may be necessary for day-to-day maintenance of life, growth, and normal functions of various organs, apart from energy production.

Nutrition refers to the absorption and assimilation of nutrients, whereas diet means the amount and type of food and drink consumed.

A balanced diet is one containing proportional amounts of proteins, minerals, fatty acids, and vitamins. Carbohydrate is utilized for the production of energy. It is unusual for a single constituent to be

deficient in the diet of humans and the clinical descriptions of nutritional deficiency may be due to a group of essential nutrients.

The vitality of the periodontal tissues, in both health and disease, depends highly upon an adequate source of essential nutrients being available to the host. The epithelium of the dento-gingival junction and the underlying connective tissue are the most dynamic tissues of the body. So, the integrity and maintenance of these tissues depend upon an adequate supply of a balanced diet.¹

EFFECT OF NUTRITION ON ORAL AND PERIODONTAL TISSUES

1. There are nutritional imbalances that produce changes in the oral cavity e.g., alterations of lips, oral mucosa, bone, and periodontal tissues. These changes are considered to be periodontal or oral manifestations of periodontal disease.
2. There are no nutritional disorders that by themselves can cause gingivitis or periodontal pockets. It is more likely, however, that a state of malnutrition will predispose a subject to the onset of a periodontal infection or will have an effect on the rate of progression of established disease.²

The exact mechanisms by which nutritional deficiencies alter periodontal destruction have not been precisely defined. has suggested that any of the basic periodontal defense factors could be affected.³ These include the protein and urea content of both saliva and GCF, the integrity of dentogingival junction, chemotaxis of PMNL, and activation of

lymphocytes, and the production of immunoglobulin in the immune response.

Nutritional deficiency can be primary or secondary, primary being due to the deficient dietary supply and secondary due to the inability to utilize the dietary nutrients.

The multifactorial etiology of periodontal disease makes the study of a specific predisposing factor very difficult. Although animal experiments have shown that nutritional deficiency can affect periodontal health, the evidence for similar periodontal changes in humans is less convincing. When nutritional problems are detected in humans they are often associated with predisposing conditions such as alcoholism, mental retardation, medical problems, or inadequately functioning dentures.⁴

THE CONSISTENCY OF DIET

From the viewpoint of periodontal health, it is stated that a firm and fibrous diet is more beneficial than an intake of softer, more loosely textured foods. Softer diets tend to produce greater deposits of plaque.⁵ and this trend is even greater when the soft diets contain high proportions of sucrose. It may be because of sucrose being the main substrate for the plaque-forming Streptococci.

Fibrous foods are considered advantageous in their ability to impart a natural cleansing action to the teeth and periodontium. However, Sreebny & Birkeland^{6, 7} found that fibrous food in humans does not have an effect on plaque formation, GCF production, or the severity of gingival inflammation.

Coarse and granular diets can predispose to direct traumatic injury to supporting tissues, most likely in proximal regions where food impaction occurs.

The consistency of diet may be more important than its nutritional content in contributing to gingival irritation and subsequent inflammation.

Along with natural cleansing action, another advantage of coarse and fibrous diet is their ability to stimulate the oral lining tissues and enhance keratinization. Weinmann & Alfano^{8, 2} points out that the crevicular epithelium, which is under constant insult from bacterial plaque, is non-keratinized and dietary stimulation is not likely to induce keratinization. Therefore, keratinization promoting effect of fibrous diet cannot be substantiated.

PROTEINS

Protein deprivation causes the following changes in the periodontium of experimental animals: degeneration of the CT of gingiva and PDL, osteoporosis of periosteum and alveolar bone, a decrease in cementum formation, delayed wound healing, and atrophy of tongue epithelium. In rats fed protein-deficient diets, there is a reduction in PDL collagen fibers, particularly the transeptal component as observed by Chawla & Glickman 1951⁹. The epithelial attachment remained largely unaffected. Further, when protein deprivation was combined with a diet of soft consistency, the pathological changes occurred extremely rapidly with marked degeneration of periodontal support.¹⁰

Studies conducted on humans indicated that a high protein and low carbohydrate diet had a significant effect in reducing sulcus depth and clinical tooth mobility and in improving gingival health.

Protein deficiency accentuates the destructive effects of local irritants and occlusal trauma. Type I diabetes complicated by chronic renal failure reduces the synthesis of Vitamin D. This causes decreased serum Ca level and secondary hyperparathyroidism can contribute to alveolar bone loss. Under these circumstances, a low protein diet can slow the progression of renal failure by lowering serum phosphate. Further work is needed to establish in humans that a low protein diet has a favorable effect in reducing alveolar bone formation.

CARBOHYDRATES

Holloway¹¹ suggested that refined carbohydrates in the diet influence the severity of chronic inflammatory PDD in humans.

Syrian hamsters fed on a soft high carbohydrate diet showed increased periodontal breakdown and substitution of fat in the diet reduced the breakdown. Studies in rodents found that high content of sucrose in the diet increased the bacterial growth which invaded the sulcus and contacted the JE thereby separating the epithelium from the tooth surface. However, others report no correlation between PDD and carbohydrates.

VITAMINS

Vitamins are essential and biologically active constituents of a diet, which cannot be synthesized within the body and are acquired in only small amounts. The deficiency of certain vitamins has been implicated as being a primary etiological factor in the pathogenesis of PDDs. Vitamins are classified into fat-soluble (Vitamin A, D, E, K) and water-soluble (Vitamin B complex and C).

VITAMIN A

Vitamin A is essential for the normal function of the retina; for growth, differentiation, and maintenance of epithelial tissues; and for bone growth and embryonic development.

The following periodontal changes have been reported in vitamin A-deficient rats, hyperplasia and hyperkeratinization of gingival epithelium with the proliferation of the JE, and retardation of gingival wound healing. It is unlikely that a deficiency of Vitamin A alone will cause gingivitis.

In the presence of local irritation, Vitamin A-deficient rats develop periodontal pockets that are deeper than those in non-Vitamin A-deficient animals and exhibit associated epithelial hyperkeratosis.

The reported observations regarding alveolar bone changes have been contradictory and include the replacement of bone trabeculae with fibrous CT reduced bone formation, and increased thickness of bone, and greater deposition on the labial aspect of the cortical plates.¹² Frandsen suggested that the main effect of Vitamin A deficiency on bone is to suppress resorption by inhibiting osteoclast function. Osteoblast function may also be reduced, although if the magnitude of suppression is greater in favor of the resorbing cells, then bone deposition will continue, albeit at a slower rate.

Studies in humans have been unable to determine any significant correlation between Vitamin A deficiency state and PDD (Vitamin E is known to inhibit the oxidation of Vitamin A).

VITAMIN B COMPLEX

Vitamin B complex includes thiamine (B1) riboflavin (B2), niacin, pantothenic acid, pyridoxine (B6), biotin, folic acid, and cobalamin (B12). Oral disease is rarely due to a deficiency in just one component of the B complex group, the deficiency is generally multiple. Oral changes common to B complex deficiencies are gingivitis, glossodynia, angular cheilitis, and

inflammation of the entire oral mucosa. The gingivitis in Vitamin B deficiencies is nonspecific, as it is caused by bacterial plaque rather than by the deficiency, but it is subject to the modifying effect of the latter.

Thiamine (B1)

It is an important factor in the energy metabolism, formation of RNA, DNA, etc. The human manifestations of thiamine deficiency called beriberi are characterized by paralysis, cardiovascular symptoms including edema, and loss of appetite. In the oral cavity, it may manifest as hypersensitivity of the oral mucosa, minute vesicles simulating herpes on the buccal mucosa, under the tongue, or on the palate, and erosion of oral mucosa.

Riboflavin (B2)

Riboflavin is essential for ATP synthesis and the metabolism of carbohydrates, proteins, and fats. The symptoms of riboflavin deficiency (riboflavinosis) include glossitis, angular cheilitis, seborrheic dermatitis, and superficial vascularizing keratitis.

The glossitis is characterized by a magenta discoloration and atrophy of the papillae.

Angular cheilitis begins as an inflammation of the commissure of the lips, followed by erosion, ulceration, and fissuring. Candidiasis may develop in the commissures of debilitated persons; this lesion has been termed perleche.

In animal's riboflavin deficiency produced severe lesions of the gingivae, periodontal tissues, and oral mucosa including noma.

Niacin

This acts as a coenzyme for tissue respiration and glycolysis. Niacin deficiency results in pellagra, which is characterized by dermatitis, diarrhea and dementia, glossitis, gingivitis, and generalized stomatitis.

The gingiva may be involved in niacin deficiency with or without tongue changes. The most frequent finding is ANUG, usually in areas of local irritation.

Oral manifestations of Vitamin B complex and Niacin deficiency in experimental animals include the black tongue, and gingival inflammation with destruction of the gingiva, PDL, and alveolar bone. Necrosis of the gingiva and other oral tissues and leucopenia are terminal features of niacin deficiency in experimental animals.

Pantothenic acid

It is an important cofactor in the metabolism of carbohydrates, proteins, and fats. Its deficiency usually causes fatigue, nausea, malaise, etc. Mice with pantothenic acid deficiency showed wider PDL with epithelial rest proliferation and an increase in the size of BV on the bone side of PDL.

Biotin

It is important in DNA and RNA synthesis. Its deficiency causes dermatitis, glossitis, loss of appetite, nausea, muscle pains, etc.

Pyridoxine

This vitamin is essential for protein synthesis, Hb production, and regeneration of RBC. Its deficiency in humans causes angular cheilitis, glossitis, stomatitis, etc.

Folic acid

It is essential for the normal formation and development of RBC and DNA synthesis.

Folate deficiency results in macrocytic anemia with megaloblastic erythropoiesis, with oral changes and gastrointestinal lesions, diarrhea, and intestinal malabsorption.

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Folate deficient animals demonstrate necrosis of the gingiva, PDL, and alveolar bone without inflammation. The absence of inflammation is the result of deficiency-induced granulocytopenia. In human's folate deficiency results in generalized stomatitis, which may be accompanied by ulcerated glossitis and cheilitis.

In humans, a significant reduction of gingival inflammation was observed after systemic or local use of folate with no change in plaque accumulation. Some authors have postulated that the gingival changes associated with pregnancy, oral contraceptives, and phenytoin may be partly related to a suboptimal level of folate in the gingiva. Folate deficiency is common in pregnant women.

Cyanocobalamin (Vitamin B12)

This vitamin is essential for DNA synthesis. Its deficiency is generally due to a lack of intrinsic factors which results in pernicious anemia.

VITAMIN C

Vitamin C is the anti-scorbutic vitamin, present in fresh fruits and vegetables and human milk. Man, non-human primates, and guinea pigs are dependent totally upon exogenous sources for Vitamin C. Severe deficiency of vitamin C in humans result in scurvy, a disease characterized by hemorrhagic diathesis and retardation of wound healing. Alcoholism may predispose an individual to scurvy. A minimum daily intake of 10mg ascorbic acid is protective against scurvy. It is generally considered that a daily intake of 30 mg will satisfy all metabolic requirements.

Ascorbic acid and CT metabolism

Vitamin C contributes to the formation of collagen, bone matrix (glycosaminoglycan), and the intercellular cement substance of the endothelial compartment in the vascular tree.¹²

Vitamin C is required in 3 steps of collagen formation:

1. Activation of prolyl and lysyl hydroxylase from inactivate precursors.
2. Hydroxylation of proline and lysine residues in the already synthesized collagen polypeptides.
3. Aggregation of the polypeptide into the triple helix before its secretion from the cell.

In case of deficiency of vitamin C, there is a failure of hydroxylation, incomplete intra, and intermolecular cross-linkage and hence the collagen fibrils formed lack tensile strength, have increased solubility, and are more vulnerable to enzymatic degradation.

The highest collagen content is present in tunica adventitia, tunica media, and basal lamina of BV.

Vitamin C may also be associated with alkaline phosphatase, which is present in high concentration in the vicinity of collagen formation¹³. Alkaline phosphatase activity is reduced significantly in vitamin C deficient guinea pigs.

Formed collagen is unaffected by vitamin C deficiency and so alteration in the structure of the fibers of PDL will result from the inability of the host to synthesize and repair.¹⁴ Vitamin C does not appear to reduce the number of fibroblasts present in CT.

Vitamin C and PMNL

Vitamin C may be important in maintaining the function of WBC e.g., phagocytes and chemotaxis. In vitamin C deficiency chemotactic and phagocytic abilities of WBC are reduced increasing the susceptibility of the dento-gingival area to bacterial attack. But the evidence for this is not substantial.

Clinical features:

Signs and symptoms of scurvy are associated with the impaired production of collagen and intercellular ground substance and with the resulting weakness of capillary walls. Common signs include hemorrhagic lesions in the muscles of extremities, the joints, and sometimes the nail beds; petechial hemorrhages, often around hair follicles, and increased susceptibility to infections. Anemia may result from the loss of blood. Wound healing is impaired, particularly in the deeper aspects of wounds that rely upon capillary growth and the production of collagen fibers for successful organization.

Periodontal features of scurvy

The oral symptoms associated with scurvy can be very similar to those of chronic edematous gingivitis. The oral inflammation, which is exacerbated by poor oral hygiene, can involve the free gingiva, attached gingiva, and alveolar mucosa. Gingivitis with enlarged hemorrhagic, bluish-red gingiva is one of the classic signs of vitamin C deficiency. In severe cases, the gingiva becomes red, tender, and grossly swollen. The spongy tissues are extremely hyperemic and bleed spontaneously or on gentle stimulation. In long-standing cases, the tissues attain a darkish blue or purple hue and can suggest conditions like leukemia. Ulcerations may develop and lead to secondary infections.

Alveolar bone resorption with increased tooth mobility has been described.³ True loss of attachment and pocket formation do not occur as a result of vitamin C deficiency alone.¹⁵

Possible etiologic relationships between Vitamin C and PDD

1. Low levels of vitamin C influence the metabolism of the periodontium, thereby affecting the ability of the tissue to regenerate and repair itself. There is no evidence to support this view.
2. Vitamin C deficiency interferes with bone formation, leading to loss of periodontal bone.

Bone changes occur as a result of the failure of the osteoblasts to form osteoid and take place very late in a deficiency state.

3. Vitamin C deficiency increases the permeability of the oral mucosa to tritiated endotoxin and tritiated insulin and of the normal human crevicular epithelium to tritiated dextran. It suggests that vitamin C maintains the epithelium's barrier function to bacterial products.
4. Increased levels of vitamin C enhance both the chemotactic and the migratory action of leucocytes without influencing their phagocytic ability.
5. Megadoses of vitamin C seem to impair the bactericidal activity of leukocytes. Excessive intake of vitamin C may precipitate problems such as renal calculi, diarrhea and can also interfere with the action of certain drugs including warfarin and aspirin (increased calcium oxalate, decreased vitamin B12 synthesis, mental toxicity).
6. Vitamin C is required to maintain the integrity of periodontal microvasculature and vascular response to bacterial irritation and wound healing.
7. Vitamin C deficiency interferes with the ecologic equilibrium of bacteria in plaque thus increasing its pathogenicity but there is no evidence to demonstrate it.

Gingivitis

Gingivitis is not caused by vitamin C deficiency per se. Gingivitis in vitamin C deficient patients is caused by bacterial plaque. Vitamin C deficiency may aggravate the gingival response to plaque. Acute vitamin C deficiency does not cause or increase the incidence of gingival inflammation.

Periodontitis

Acute vitamin C deficiency results in edema and hemorrhage in PDL, osteoporosis of alveolar bone, tooth mobility, edema and hemorrhage, and degeneration of collagen fibers in the gingiva. The periodontal fibers that are least affected by vitamin C deficiency are those just below the JE and above the alveolar crest, which explains the infrequent apical down growth of epithelium.

Vitamin C deficiency does not cause periodontal pockets. Local bacterial factors are required for pocket

formation to occur. However, acute vitamin C deficiency accentuates the destructive effect of gingival inflammation on the underlying PDL and alveolar bone.

VITAMIN D

Vitamin D (cholecalciferol) itself is inactive but is converted to the active form (1, 25-dihydroxycholecalciferol) by 2 hydroxylation reactions that occur in the liver and kidney. This active form promotes the retention of calcium and phosphate in the body. It increases the absorption of calcium in the small intestine and mobilizes calcium from formed bone to maintain plasma levels of calcium. With advancing age vitamin D can be reduced which leads to reduced absorption of calcium leading to sec hyperparathyroidism and bone resorption.¹⁶

Deficiency of Vitamin D results in rickets in children and osteomalacia in adults. Both conditions are characterized by defective mineralization of the organic matrix.

Oliver 1969 and his coworkers observed no significant changes in the periodontal tissues of rats deficient in Vitamin D alone.¹⁷ However, in calcium-deficient rats and animals suffering from a deficiency of both calcium and vitamin D, there was a reduction of alveolar bone mass and greater areas of unmineralized osteoid. In the PDL of rats, the number and diameter of dentoalveolar fibers were reduced when both the factors were deficient.

In dogs with hypervitaminosis D, periodontal changes observed were increased osteoblastic activity, pathological calcification of periodontal membrane and gingiva, osteosclerosis of the alveolar bone, and marked hypercementosis.

VITAMIN E (TOCOPHEROL)

It acts as a lipid antioxidant and it has an important role in maintaining the stability of cell membrane and protecting RBC against hemolysis. Vitamin E deficiency can cause spontaneous abortion and impaired spermatogenesis.

The possible role of vitamin E in PDD is based on its ability to interfere with the production of PGs, which themselves are important in the development of inflammation. But there is no evidence to demonstrate it.

A specific correlation between vitamin E deficiency and PDD will be difficult to determine. In rats,

systemic vitamin E appears to accelerate gingival wound healing.

VITAMIN K

Vitamin K is essential for blood clotting. Its deficiency does not lead to any direct periodontal changes. However, there may be increased gingival bleeding owing to the failure of the clotting mechanism.

MINERALS

Minerals may be divided into 2 groups –

1. Macrominerals, which are required in amounts > 100mg/day.
2. Microminerals, or trace elements that are required in amounts less than 100mg/day.

Calcium and phosphate

There is a great deal of controversy in the role of calcium and Po4-2 in the initiation and progression of periodontal diseases.

Calcium deficiency may result from dietary deficiency or from deficiency of vitamin D. It can be induced by renal disease or dysfunction of PTH. Calcium metabolism is regulated by a series of interactions and alteration of any one of these may produce changes. Therefore, it is difficult to separate primary from secondary actions.

In the theory, hypocalcemia and hyperphosphatasemia that result from dietary imbalance will produce sec HPT, which initiates alveolar bone resorption. Henrik son (1968)¹⁷ believed that these dietary factors are of greater importance than a dental plaque in the pathogenesis of PDD. Later experiments on rats and dogs failed to substantiate this theory. G Svaberg et al 1973¹⁸ suggest that a hypocalcemic diet can produce inter-radicular osteoporosis and thinning of individual trabeculae, but it will not initiate inflammation, migration of epithelial attachment, loss of periodontal fibers, or resorption of alveolar margin.

Magnesium

Widening of PDL has been observed with Mg deficiency. In rats, Mg deficiency caused a delayed eruption of incisors. These effects may be related to the role of Mg++ ions in several enzyme systems¹⁹.

Fluoride

Fluoride in drinking water prevents tooth decay with no health hazards. In populations using fluorinated

water supplies, fluoride appeared not to have any effect on the periodontium. But findings in experimental animals vary, with some investigators reporting that fluoride increases PDD and others noting that it decreases or protects against PDD. Fluoride appears to reduce the destructive effects of excessive orthodontic movement in rats.²⁰ It was observed that there were fewer areas of PDL hyalinization in fluoride-treated animals than in controls.

Iron

Its deficiency may cause a decrease in thickness of the oral epithelium, decrease in size of progenitor cells, and delayed maturation of the epithelial barrier. Severe iron deficiency has been related to periodontal destruction in dogs. In humans, however, there does not seem to be a clear correlation between iron deficiency anemia and chronic inflammatory PDD.

Cobalt

It retarded the eruption of rat incisor.²¹ there were no reports concerning its effects on the periodontium.

Copper

A positive and significant correlation has been demonstrated between serum copper and the severity of PDD.²² the inflammatory process itself is known to elevate serum copper and this may be related to a leukocyte factor and mobilizes ceruloplasmin from the liver. Copper is also essential for the development and maturation of CT.

Zinc

Serum zinc levels were found to decrease with an increase in alveolar bone resorption. Zinc can inhibit several functions of PMNL ad it can also stabilize cell membrane and inhibit the release of lysosome enzymes. The reduction in serum zinc in PDD therefore may stimulate both leukocyte function and the release of potent enzymes that will enhance the inflammatory process and leads to loss of periodontal collagen. It is essential for wound healing as it is involved in collagen and DNA synthesis. Zinc is essential for cross-linking collagen.

Nutritional in Aging

Aged persons select foods requiring less chewing effort when masticatory efficiency is impaired. Avitaminosis is relatively common. An adequate intake of vitamins, calcium, iron, and potassium is important. A diet high in fiber, vitamins, and proteins and comparatively low in fat may be beneficial.

Nutrition and wound healing

A well-balanced diet is essential for normal wound healing. In case of deficiencies in the healing process may take a longer time and the scar formed may be lacking in tensile strength. Concerning oral wound healing, the nutrients of importance are vitamin A due to its role in epithelization, vitamin C due to its role in collagen formation and maturation, vitamin D, Ca, and P as they are required for osteogenesis.²³ During healing there is an increased demand for all the nutrients as there is increased metabolic activity.

DAILY REQUIREMENTS

Name of vitamin	For Infants	For Adolescents	For Adults	Pregnancy/Lactation
Vitamin A	0-6 months – 400 micrograms 6-12 months – 300 micrograms	13 to 15 & 16 to 19 – 750 micrograms	750 micrograms	25,000 IU weekly as an oral liquid
Vitamin D	5.0 mcg (200 IU)	5.0 mcg (200 IU)	2.5 mcg (100 IU)	10.00 mcg (400 IU) (IU of vitamin D = 0.025 ugs)
Vitamin E	2.8mg/kg per day	3.5 mg/day	10 mg / day	22-30 mg/day, prevents the damaging effect of ethanol and diabetes on the fetus
Vitamin K	2.5 micrograms/day	66 mcg	Adult 0.03 mg / kg-everyday	10 mg daily
Thiamine (vitamin B ₁)	0.3 mg	1.2 mg	0.5 mg per 1000 k cal of energy	1.4 milligrams 1 daily.
Riboflavin (B ₂)	0.4 mg	1.0 mg	1.3 mg/day	1.4 milligrams
Niacin	4 mg/day	12 mg/day	14 mg/day	18 mg/day

Vitamin B ₆ (pyridoxine)	0.1 mg/day	1.0 mg/day	2mg/day	2.5mg/day
Pantothenic acid	3 mg/day	4-5 mg/day	5 mg/day	6mg/day
Folate	65 mcg	100 mcg	100 mcg	300 mcg/150 mcg
Vitamin B ₁₂	0.2 mcg	0.2 mcg	1 mcg	1.5 mcg/1.5 mcg
Vitamin C	40 mg	50 mg	40 to 60 mg / day	80-115mg/day
Calcium	260 mg/day	1,300 mg/day	1200 mg/day	1,000 mg/day
Phosphorous	275 mg	1250mg	1,250 mg	700 mg

CONCLUSION

Although nutrition is considered a secondary factor in the etiology of PDD, its mediating influence on the periodontal tissues should not be overlooked. Clinical manifestations are suggestive, but definite diagnosis of nutritional deficiencies and their nature requires the combined information revealed by the history, clinical and laboratory findings, and therapeutic trial.

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