

Nutritional Requirements of the Periodontal Patient

Rosaiah K¹, Aruna K², Nandini TN³

Abstract

The course and severity of most infections are exaggerated in malnutrition. Tissues with a rapid rate of cell renewal such as the periodontium depend on the ready availability of essential nutrients for the maintenance of their activities. They are therefore susceptible to the effects of malnutrition.

The objectives of this discussion are two fold:

1. To update the clinician about the effects of specific nutritional deficiencies on the periodontal tissues.
2. To provide a practical method for implementing this information by using an orderly, easily understood nutritional counselling service that will appeal to the patient.

Key words: Malnutrition, periodontium, nutritional counselling, essential nutrients.

Introduction

Nutrition may influence the growth, development and metabolic activities of the periodontium. It is generally acknowledged that gingivitis and periodontitis are the result of an accumulation of supra and subgingival plaque or calculus or both. However, the extent and the intensity of the gingival inflammatory process are directly affected by both the number and the virulence of dental plaque bacteria around the supra and subgingival margins of the teeth. It is indirectly affected by the relative innate resistance of the periodontal tissues to infection which depends on the nutritional status of the individual.

The dental health professional therefore has a responsibility not only to remove the local gingival plaque and calculus by scaling, polishing, root planning and curettage accompanied by a meticulous daily home plaque-control program (tooth brushing and flossing) but also to help the patient increase the systemic resistance of periodontal tissues by nutritional counselling.

Nutritional deficiency may not initiate periodontal disease but perpetuates it. Stahl Periodontitis is associated with low serum/plasma micronutrient levels, which may result from dietary and/or life-style factors. Early evidence suggests beneficial outcomes from nutritional interventions supporting the contention that daily intake of certain nutrients should be at the higher end of recommended daily allowances. For prevention and treatment of periodontitis daily nutrition should include sufficient antioxidants, vitamin D and calcium.^{1,2} Inadequate antioxidant levels may be managed by higher intake of vegetables, berries, and fruits, or by phytonutrient supplementation. Low zinc and calcium intake was associated with oral mucosal pathology and lesser number of teeth.^{3,4}

A look at the type of nutritional deficiencies:

Primary Nutrient Deficiencies

Protein-calorie malnutrition is a common problem in developing countries. It promotes the development of acute periodontal lesions in children and adults. Kwashiorkar is complicated by concurrent deficiencies of other essential nutrients. It is primarily a disease of infants and

young children with a peak age incidence of 1-3 years.

It is characterized by:

- Lesions of buccal mucosa.
- Significant generalized osteoporosis.
- Alveolar bone loss.
- Raised oral hygiene index scores with more periodontal pathologic conditions.
- Degeneration of connective tissue fibres in the gingiva and periodontal ligament.
- Osteoporosis of alveolar bone, retardation in deposition of cementum.

Protein calorie malnourished children have higher incidence of necrotizing ulcerative gingivitis and periodontitis which may extend to the adjacent tissue causing necrosis and destruction of orofacial tissue called 'noma' or 'cancrum oris'.⁵

Conditioned marginal nutrient deficiencies^{6,7}

Marginal nutrient deficiencies may be conditioned by a number of factors and social habits such as increased use of drugs, learned taste aversions, alcoholism and food faddism. For example:

- Contraceptive steroids may condition marginal nutritional deficiencies by increasing requirements for folate, ascorbate and pyridoxine.
- Aluminium containing antacids can cause calcium and magnesium deficiencies.
- Alcoholics who take 2000 or more calories from ethanol have less opportunity to consume a balanced diet. Altered liver and digestive functions can result in deficiencies of thiamine and folate.
- Taste aversion to citrus fruits can lead to ascorbate deficiency.

The repair and defense mechanisms of the patient may be jeopardized not only by the marginal nutritional deficiencies in proteins but also by deficiencies in vitamins such as ascorbic acid, folic acid and vitamin A or in minerals such as iron, zinc and calcium.

Nutrition and the epithelial barrier

The rapid rate of the turnover of epithelial cells of the

¹Professor & Head, Dept. of Periodontics, ²Sr. Lecturer, Dept. of Conservative Dentistry, Peoples Dental Academy, Bhopal, ³Senior Lecturer, Conservative Dentistry & Endodontics, Bapuji Dental College & Hospital, Davangere India.

gingival sulcus indicates the need for continuous synthesis of DNA, RNA and tissue proteins which means that the sulcular epithelium has a high requirement for such nutrients as folic acid and protein, which are involved in cell formation. Further, if malnutrition does occur, the gingival sulcular tissue may be among the first to be affected adversely, followed by involvement of oral mucosal membranes and perioral tissues.⁸

To maintain the integrity of this epithelium, Vitamin A is also needed. At the base of the sulcular epithelium is a narrow basement membrane, made up of collagen. This basement membrane acts as a barrier against the entrance of toxic materials into the underlying connective tissue. Since collagen is the major biochemical component of the basement membrane, adequate amounts of ascorbic acid, iron and zinc are important for collagen synthesis and ultimately for wound healing.

Role of vitamins^{8,9}

A study was conducted to correlate how the water-soluble vitamins B2, B3, B6, B12, C, and folic acid, fat-soluble vitamins (A, D, and E), and minerals (calcium, fluoride, iron, and zinc) can affect the oral mucosa which concluded that oral manifestations of nutritional deficiencies can affect the mucous membranes, teeth, periodontal tissue, salivary glands, and perioral skin.

Thiamin B1: The affected oral tissues due to B1 deficiency show slight edema of mucous membrane, have a satiny appearance and show hyperesthesia. Thiamin affects both central and peripheral nervous system and is associated with a typical neuralgia.

Riboflavin B2: The tissues affected due to B2 deficiency have a typical purplish magenta color. Marginal gingiva and oral mucosa have a purplish color and are edematous. Itching and burning of oral mucosa, ulceration of marginal gingiva and interdental papillae, marginal gingivitis and periodontitis may be seen. Deficiency also causes glossitis and angular cheilitis along with epithelial atrophy.

Niacin: Deficiency causes pellagra characterized by painful, fiery red lesions of oral mucosa and tongue with intense burning. The gingivitis is extremely painful with wedge-shaped punched out ulcers involving the interdental papillae and marginal gingiva. The lesions are necrotic, exudative and foul smelling.

Vitamin C: Dietary deprivation of Vitamin C for 3 months proceeds marked depletion of the body stores. A dietary intake of 10 mg/day is necessary to prevent scurvy in healthy adults. It is characterized by ocular hemorrhage, xerostomia, femoral neuropathy, impaired vascular reactivity, psychologic disturbance, ascorbutic arthritis and gingivitis.

Oral manifestations of scurvy

Fiery red, glazed and swollen gingiva with loss of stippling is the characteristic feature of vitamin C deficiency. Periodontal ligament is widened by resorption of surrounding bone and breakdown of collagen fibres in the periodontium. Vitamin C deficiency affects fibroblasts, osteoblasts and odontoblasts. The cells fail to produce normal collagen, osteoid and dentin and the ability of cells to form epithelial and vascular basement membranes is also restricted. Severe Vitamin C deficiency is characterized by hemorrhagic tendencies, impaired wound healing and osteoporosis.

Folic acid: In folic acid deficiency, there is impairment of keratinization with increased susceptibility to ulceration and secondary infections.

Vitamin A: Deficiency of Vitamin A produces marked retardation in growth, alterations in epithelial and nervous tissue, cartilage and bone, severe interference with vision and reproduction. Hyperplasia and keratinization of mucous membranes are characteristic features.

Vitamin D: Vitamin D deficiency causes rickets in the young and osteomalacia in the old. The lesion shows defective mineralization of osteoid.

Vitamin E: Deficiency results in increased tendency for hemolysis. It affects cross linking of collagen.

Vitamin K: Vitamin K is necessary for prevention of hemorrhagic condition associated with insufficient ability of blood to coagulate.

Role of Saliva^{10,11}

The significance of the buffering, rinsing, antibacterial and remineralization properties of saliva in the prevention of dental caries is well established. However, it is apparent that the protective aspects of saliva are less affective for the periodontal tissues than they are for the tooth.

Nevertheless, certain salivary proteins including lactoferrin, lactoperoxidase, lysozyme and secretory IgA may be useful in either clearing periodontal pathogens from the oral cavity or minimize the ability of pathogens to adhere to and colonize periodontal tissues.

Gingival fluid¹⁰

Although gingival fluid contains numerous antimicrobial agents including antibodies, lysozyme, complement phagocytes and transferrin, it also represents one of the primary nutrient source for subgingival bacteria. Nevertheless, gingival fluid has been shown to generate considerable bactericidal activity in situ and dental plaque does not initially form near the gingival sulcus. It is therefore probable that early gingival fluid flow has a net protective effect on the periodontium.

Role of minerals^{12,13}

Maintenance of life and optimal health requires the availability of several inorganic elements of which some (calcium, phosphorus, magnesium) are present in the body in micro-quantities and others (iron, copper, cobalt, iodine, sulphur, manganese, zinc, fluorine, sodium, potassium and chlorine) are required in trace amounts. Abnormalities of the oral cavity such as angular stomatitis and atrophic changes on the dorsum of tongue and buccal mucosa have been noted in iron deficiency. Zinc seems to have the property of accelerating wound healing which may be due to its anti-infective action. Vitamin D, magnesium, calcium and phosphorus are important nutrients for promoting the density of alveolar bone, which surrounds the roots of the teeth.

Continuous critical period¹²

Gingival sulcular epithelium has one of the fastest turnover rates in the body, and this tissue is probably completely removed in 3-6 days. This rapid renewal, in combination with the facts that epithelial cells may either adsorb or phagocytize microbial irritants, may constitute an important mechanism of periodontal defense. To maintain this short turnover time, the tissue must be in a state of continuous rapid DNA, RNA and protein synthesis. Thus the tissue is particularly sensitive to nutrient restrictions.

In an effort to emphasize this nutritional susceptibility we have suggested that the sulcular epithelium is in a 'continuous critical period' analogous to the critical periods which occur during organogenesis in the growth and development of an organism. Nutritional stress during the continuous critical period will impair the renewal of the sulcular epithelium and possibly compromise its protective qualities.

Immunity^{5,6}

Malnutrition has significant effects on the inflammatory process as well as cellular and humoral immune mechanisms. Nutritional deficiencies result in inadequate mobilization of leucocytes with an altered phagocytic capacity and decreased anti-bacterial potential. Although inflammation, cellular and humoral immunity are usually considered to be mechanisms of host defense, the potent biological activity generated by these processes in periodontal disease may actually contribute to the progress of tissue destruction. One can argue that the constant migration of polymorphonuclear leukocytes through the junctional epithelium and into the gingival crevice serves to phagocytize and destroy bacterial cells, thereby protecting the host. Conversely, the release of potent lysosomal enzymes associated with the activity of these host cells may be more damaging to the periodontal tissues than the bacteria themselves.

Therefore, the impairment of phagocytic activity and cellular or humoral immunity associated with malnutrition may contribute to the initiation and progression of periodontal disease.

Repair¹²

The progression of inflammatory periodontal disease is often considered to depend upon the balance between active destruction, mediated perhaps by both host and bacterial factors and the capacity for repair of periodontal tissues. This wound-healing interaction is dependent upon many factors including the nutritional status of the host. The ability of the host to maintain an effective healing capacity is dependent upon an adequate supply of nutrients to the healing tissues either from diet or from systemic nutrient stores.

Clinical applications of nutrition in periodontal patients

The periodontist and the general dentist who treats periodontal patients probably have more experience in evaluating the host response to chronic irritation than any other health professional. This experience provides the periodontist with a unique tool for diagnosing latent systemic problems - "The Oral Bioassay". This assay is based on the subjective integration by the clinician of the amount of local irritants (plaque, calculus, overhanging restorations etc.) present in the oral cavity with the degree of periodontal destruction present. When the amount of periodontal destruction is excessively greater, then based on the amount of local irritants present, the clinician's suspicion of systemic involvement should increase.

The oral bioassay should be conducted at three different stages during periodontal therapy:

- The primary evaluation should be conducted prior to any form of therapy, when the patient first presents himself for treatment. This analysis provides useful

baseline data in terms of both oral hygiene and the response of the periodontal tissues to the existing irritants.

- The response of the periodontal tissues should be evaluated a second time after initial preparation when the local irritants have been removed. Poor tissue reactivity at this stage suggests that an inadequate healing potential exists which may compromise the effectiveness of any anticipated surgical procedures.
- It is useful to include a third assay for tissue response after the first surgical procedure. As the surgical periodontal patient has potential nutritional and dietary requirements which may surpass those of the average dental patient, the clinician should analyze these requirements, counsel the patients and modify or supplement the diet as the needs of the patient dictate.¹²

Nutritional counselling for a patient with chronic periodontal disease^{8,9,12,14,15}

The following is a step by step procedure for giving personalized nutritional guidance to a patient with chronic periodontitis.

Step 1: Evaluate the diet and if necessary, demonstrate the method for keeping a food intake diary.

A food intake evaluation chart is one in which all foods are classified into 4 food groups.

1. Vegetable fruit group.
2. Bread cereal group.
3. Milk cheese group.
4. Meat, poultry, fish and beans group.

Step 2: Explain the nutrition periodontal relationship:

Patients tend to be more co-operative if the nature of the problem and the rationale for making some improvement in their dietary pattern is explained. Thus the advice will be-

1. Reducing sugar intake.
2. Replacing sweets and other empty calorie foods with nutritious, firm and fibrous foods that will stimulate and strengthen the periodontal tissues.
3. Selecting a well balanced, varied, adequate diet to provide all the essential nutrients and to support overall health in general and the health of tooth supporting structures in particular.

Step 3: Assess nutritional status by taking a detailed medical history.

Social and Diet histories: In order to prescribe a diet that the patient will be able to fulfill with ease, it is necessary to ascertain his/her daily routine, food likes and dislikes, food purchases, preparations and eating habits.

Examine for clinical signs of malnutrition: Observe whether there are any clinical or oral manifestations of malnutrition such as pallor, dry skin, over weight, skin petechiae, under weight, cheilosis or glossitis. Record the findings under clinical nutritional finding.

Diagnosis: From the complaint, medical and dietary histories and clinical findings, a nutritional diagnosis that includes the primary and secondary nutritional factors can be made.

Step 4: Prescribe a balanced diet.

Step 5: Follow up

Just as home care oral hygiene procedure must be constantly reinforced and checked for proficiency, so

should the patient's prescribed diet be periodically reviewed.

Diet before periodontal surgery

When prescribing a diet before periodontal surgery, the goal is to enable the patient to meet the stress of surgery. Furthermore, a well nourished state is optimal for wound healing. It also increases resistance to infection and hastens convalescence and recovery.¹³ If the periodontal patient is malnourished, a diet high in protein and enough carbohydrates and fat to provide about 2,500 K cal should be prescribed for 7-14 days before surgery. If the periodontal surgery is elective, it may be best to delay it until the patient's nutritional status is optimal. It may be necessary to prescribe a multivitamin capsule to be certain that adequate amounts of ascorbic acid are ingested.

Postoperative dietary management of hospitalized periodontal surgery patient

During periodontal surgery under general anesthesia, intravenous levels of solution of 0.45% saline with 5% dextrose in water and 38.5 milliequivalents of sodium is given. The intravenous infusion is terminated in the recovery room if the patient is in good health. However, if the patient seems dehydrated, it may be wise to continue the infusion until the patient is fully rehydrated and can take fluids by mouth. When fully recovered from the anesthesia, the patient should be given clear fluid as tolerated. In addition to water, beverages such as cola drinks, ginger ale, apple juice and orange juice in addition to clear broths or clear tea or black coffee with sugar and flavored gelatin are usually well tolerated.

On the first post operative day, in addition to the beverages mentioned, sherbets, custards and ice creams may be advised if the patient is hungry. Gruel or cereal topped with sugar and milk, as well as, egg or eggnog or strained chicken, pea or vegetable soup can be suggested. Frequent small feeds are tolerated better than a few large ones.

On the second post operative day, the patient may supplement the diet with the following:

1. Vegetable fruit group: Citrus fruits such as oranges and grapes are recommended as are tomato and other fruit and vegetable juices.
2. Bread cereal group: Strained gruels can be given eg: wheat with milk.
3. Milk group: Milk in all forms can be given such as ice creams, milk shakes and malted milks.
4. Meat group: Eggs in the form of egg-nogs may be given in a liquid diet.

A full liquid diet that furnishes 2000 calories and 80 gm of proteins per day is recommended.

Conclusion

Usually, the role of diet and nutrition in the management of gingival and periodontal disease is primarily that of prevention and maintenance. The benefits from good nutrition are important, especially with respect to increase in the capacity of periodontal tissues to:

1. Resist infection.
2. Strengthen and maintain the epithelial barrier.
3. Promote the repair of damaged periodontal tissues.

The potential benefits are great enough that judicious use of nutritional guidance should be as much a routine periodontal preventive procedure as instructing the patient on oral hygiene home care measures.

References

1. Van der Velden U, Kuzmanova D, Chapple IL. Micronutritional approaches to periodontal therapy. *J Clin Periodontol* 2011 Mar;38 Suppl 11:142-58.
2. Adegboye AR, Fiehn NE, Twetman S, Christensen LB, Heitmann BL. Low calcium intake is related to increased risk of tooth loss in men. *J Nutr* 2010 Oct;140(10):1864-68.
3. Thomas DM, Mirowski GW. Nutrition and oral mucosal diseases. *Clin Dermatol* 2010 Jul-Aug;28(4):426-31.
4. Boyd LD, Madden TE. Nutrition, infection, and periodontal disease. *Dent Clin North Am* 2003 Apr;47(2):337-54.
5. Pollack RL, Kraritz E. eds. *Nutrition in Oral Health and Disease*. Lea and Fabiger, Philadelphia. 1985;426-67.
6. Keen CL, Gershwin ME. Zinc deficiency and immune function. *Annual review of nutrition* 1990;10:415-31.
7. Prasad AS. Zinc: an overview. *Nutrition* 1995 Jan-Feb;11(1 Suppl):93.
8. Moynihan PJ. The role of diet and nutrition in the etiology and prevention of oral diseases. *Bull World Health Organ* 2005 Sep;83(9):694-99.
9. Sawant SS, Khanderker SV. Role of vitamins C and E as chemopreventive agents in the hamster cheek pouch treated with the oral carcinogen-DMBA 2000 Jul;6(4):241-47.
10. Sorsa T, Suomalainen K, Uitto VJ. The role of gingival sulcular fluid and salivary interstitial collagenases on human periodontal diseases. *Arch Oral Biol* 1990;135 (Suppl):S193-S96.
11. Närhi TO, Ainamo A, Meurman JH. Salivary yeasts, saliva and oral mucosa in the elderly. *J Dent Res* 1993 Jun;72(6):1009-14.
12. Paola DPD, Alfano MC. Triphasic nutritional analysis and dietary counseling. *Dent Clin North Am* 1976 Jul;20(3):613-33.
13. Meisel P, Schwahn C, Luedemann J, John U, Kroemer HK, Kocher T. Magnesium deficiency is associated with periodontal disease. *J Dent Res* 2005 Oct;84(10):937-41.
14. Neiva RF, Steigenga J, Al-Shammari KF, Wang HL. Effects of specific nutrients on periodontal disease onset, progression and treatment. *J Clin Periodontol* 2003 Jul;30(7):579-89.
15. Muñoz CA, Kiger RD, Stephens JA, Kim J, Wilson AC. Effects of a nutritional supplement on periodontal status. *Compend Contin Educ Dent* 2001 May;22(5):425-8, 430, 432 passim;quiz 440.