Role of Nutrition in Periodontal Health - A Review

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Abstract: The relation between the systemic and oral health is now well established. Researches have shown that many systemic diseases either cause or exaggerate the course of the certain periodontal conditions. Nutrition plays an important role in growth, development as well as the maintenance of the hard and soft tissue and also has a significant influence on the host immune response. Nutritional deficiencies therefore adversely affect periodontal health, causing as well as affecting the course and severity of periodontal diseases. The purpose of this article is thus to elaborate in detail the interrelation between the nutrition, periodontal health and diseases.

Keywords: Nutrition, Periodontal diseases, Nutrient deficiencies.

INTRODUCTION

The health of oral tissues, as that of all other tissues depends upon the adequate supply of nutritive materials and the proper metabolic processes necessary to utilize them. The adequacy of a diet relates to its consistency as well as to its nutritive valve. The superimposition of local etiologic factors upon effects of nutritional deficiencies complicates the situation, making the study of human subjects a most difficult method of determining the precise effects of nutritional deficiency upon periodontal tissues.

NUTRITION

Nutrition- A Definition

"In the simplest terms Nutrition may be defined as the process by which organism utilizes food. This process is complicated one, involving digestion, absorption, transport, storage, metabolism and elimination of the many nutrients that are to be found in the very varied diets which we call our food. All of this has its purpose the maintenance of life, growth, reproduction and normal functioning of organs and production of energy." [1].

Nutriture, or nutritional status, is the state of the body produced by the process & is determined by the balance between the supply of nutrients on the one hand and the expenditure by the organism on the other.

"Nutrition", "Diet", and "Food": Are they different??

Since the words nutrition, diet and food are often misunderstood, the following definitions are included:

NUTRITION is defined as "The sum of the processes concerned in the growth, maintenance and repair of the living body as a whole or of its constituent parts."

Diet is referred to as "food and drink regularly consumed".

Food is taken to mean "any substance which when taken into body of an organism may be used either to supply energy or to build tissue." [2].

Nutrients "Types And Classification"

It is convenient to divide the nutrients into two categories, micro and macro- nutrients [1]:

Macro Nutrients

- Carbohydrates
- Proteins
- Fats

Micro Nutrients

Vitamins
- Water-soluble: Vit. B Complex, Vit. C
- Fat Soluble: Vit. A, D, E, K.

Essential >0.005%

Mineral
- (Calcium, Sodium, Potassium, Phosphorus, Magnesium)
- Trace <0.005% (Chlorine & sulfur). (Iron, Iodine, Copper, Zinc, Cobalt, Fluorine, Vanadium, Silicon)

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MALNUTRITION

Malnutrition is disordered nutrition of any kind and may be categorized in a number of ways:

Classification of Malnutrition

1	Course:	Primary (Exogenous) Secondary (Endogenous)
2	Type:	Excess, Toxicity (Over nutrition) Deficiency (Under nutrition)
3	Nutrient:	Vitamins, Elements, Protein, Energy Sources.
4	Degree:	(i) Mild- Moderate-Severe or alternatively. (ii) Depleted Stores- Biochemical Lesion- Functional Change- Structure Lesion
5	Duration:	Acute, Sub acute, Chronic
6	Outcome:	Reversible, Irreversible.

NUTRITION IN PERIODONTAL DISEASE

The Controversy

Problem in Experimental Design and Data Interpretation

The primary reason for the controversy concerning the importance of nutrition in the etiology of inflammatory periodontal disease is the extreme difficulty encountered in the design and analysis of studies of this problem. There are six basic problems which one must confront in the study of nutrition in periodontal disease:

- The multifactor aspects of periodontal disease.
- Common pitfall in nutrition research.
- Inadequate Experimental parameters
- Animal model systems
- Duration of the study
- Data interpretation.

Multifactorial Aspects of Periodontal Disease

There's probably no disease, including dental caries, which is believed to be modulated by more factors than human inflammatory periodontal disease. Local factors such as calculus, faulty restorations, poor tooth alignment and traumatic occlusion, and systemic factors including age, immunological, nutritional, psychosomatic, and endocrinologic status of the host are all believed to modulate the progress of periodontal disease.

It's clear that complex etiology of periodontal disease usually prevents the study of one component of pathogenesis of the disease.

Common Pitfalls in Nutrition Research

A common problem in the design of nutritional studies is the selection of an appropriate diet. Chemically synthesized defined diets are usually the most useful because levels of the nutrient in question and all other nutrients are known. Inadequately analyzed natural diets often supply additional nutrients and other factors that may complicate the analysis of the study. In addition, the stability and palatability of a diet should be considered when evaluating nutritional studies. Many nutrients degenerate rapidly; diets which are stored improperly or for prolonged time periods become progressively unpalatable and deficient in a number of nutrients and invalidate the experiment. Finally, the amounts of nutrients which are fed and any potential nutrient interactions should be considered.

Inadequate Experimental Parameters

Unfortunately, numerous investigators who have studied the nutrition periodontal disease relationship have not recognized the importance of using standardized periodontal indices. Therefore, their studies, based on individually developed indices or subjective criteria, are usually difficult and sometimes impossible.

Animal Model System

Problems occur in selection of an animal model because of differences in tooth shape, occlusion, interdentally spacing attachment apparatus and immune responses.

Duration of the Study

Periodontal problems are usually slowly progressive and nutritional deficiencies in man are often relative or marginal deficiencies. Theses marginal deficiencies require long periods of time for deleterious effects to become manifest. Because the majority of studies on nutrition in periodontics have used short term acute nutrient deficiencies, it's conceivable that the importance of nutrition is often underestimated.

Data Interpretation

The statistical analysis of nutritional studies is frequently quite complex. The many contributing factors in studies necessitate the consideration of several

interactions and numerous contrasts of the data are required.

PRIMARY NUTRIENT DEFICIENCIES

Two primary nutrient deficiencies appear to play some role in the etiologic history of periodontal diseases. **Protein Calorie Malnutrition** is a common problem in developing countries which promotes the development of acute periodontal lesions in children. In contrast a **relative deficiency in calcium compared to phosphorus** in the diet may play a role in development of periodontal disease in adults of industrialized countries.

PROTEIN- CALORIE MALNUTRITION

Protein-Calorie Malnutrition (Kwashiorkor) is by far the most widespread nutritional disorder in underdeveloped countries. It's primarily a disease of infants and young children with peak age incidence of 1 to 3 years.

PERIODONTAL CHANGES

- Significant generalized osteoporosis has been demonstrated, and there's evidence of alveolar bone loss.
- Epidemiological studies reveal that children suffering from kwashiorkor show significant differences in their oral hygiene index (OHI) scores and demonstrate more periodontal pathologic conditions when compared with children of similar ages drawn from a higher socioeconomic level.
- Necrotizing Ulcerative gingivitis (NUG) is rarely seen in children in developed countries; on the other hand more than half the cases seen in India are reported under the age of 10 years.

POSSIBLE MECHANISMS OF PROTEIN – CALORIE MALNUTRITION ON PERIODONTIUM

Regeneration of the gingival epithelium takes place from the basal layer; mitosis is a prerequisite. A 20% to 30% decrease in food supply in mice results in a 35% decrease in mitotic activity. Lesions in organs with rapid cell renewal and high protein turn are characteristic of protein- calorie malnutrition. In view of the ubiquitous presence of local etiologic factors in periodontal diseases, the periodontium is believed to exist in a state of continuous wound healing and repair. The healing wound presents a picture of rapid turnover of cells, collagen & mucopolysaccharides. Not surprisingly, therefore, periodontium is susceptible to deficiency of these essential nutrients.

There is also evidence that protein- synthesizing capacity of lysine deficient fibroblasts is reduced, resulting in diminished collagen production.

DIETARY CALCIUM AND PHOSPHORUS INTAKE

The suggestion has been made that periodontal disease is a metabolic bone disease resulting from a deficiency of dietary calcium of an excess of phosphorus [3].

A nutritional secondary hyperparathyroidism (NSH) occurs to maintain serum calcium levels in the presence of altered calcium to phosphorus ratio [4]. Calcium is supplied to serum from bone stores. Alveolar bone is reported to be one of the most labile sources of calcium [3]. Alveolar bone resorption, mediated by parathyroid hormone release in NSH, occurs by process of "Osteocystic- Osteolysis". This type of bone resorption differs from classical osteolytic process ("Osteoclastic resorption") as bone mineral is removed by deep seated osteocytes rather than by surface osteoclasts. "Internal resorption- cavities" are formed by fusion of the enlarged bone lacunae and a marked store. Alveolar bone is reported to be one of the most loss of bone structure is noticed as evidenced by micro- radiographic changes [5].

CONDITIONED, MARGINAL NUTRIENT DEFICIEN-CIES

It is possible to demonstrate that acute deficiencies may not always have the most marked effects. Moreover, primary nutrient deficiencies are rare in industrialized societies. On the other hand, conditioned, marginal nutrient deficiencies are more common and my play a more important role in etiologic history of periodontitis [6].

ETIOLOGIC FACTORS

Marginal nutrient deficiencies may be "conditioned" by a number of factors and social habits. By definition, these marginal deficiencies do not manifest the clinical signs and symptoms associated with classical, frank deficiency syndromes. However, sub-optimal nutrient supply can certainly compromise cell metabolism and function.

FACTORS THAT CONTRIBUTE TO MARGINAL NUTRIENT DEFICIENCIES

- Use of drugs
- Learned taste aversions.
- Alcoholism
- Food faddism

Increased use of variety of drugs by apparently healthy people can contribute to nutritional problems, for example:

- Contraceptive steroids may condition marginal nutrient deficiency by increasing requirements for folate, ascorbate and pyridoxine.
- Mineral oil and magnesium salt cathartics result in impaired absorption of fat soluble vitamins and calcium.
- Antacids may lead to calcium and magnesium deficiencies.
- The alcoholic who takes in 1200 or more calories each day from ethanol has proportionally less opportunity to consume the additional nutrients required for a balanced diet. Furthermore, as alcoholism progresses, altered liver and digestive functions can result with specific deficiencies of thiamine and folate.
- Learned taste aversions occur when an individual becomes ill after consuming a type of food. The individual assumes that a causal relationship exists and will often avoid that food for the rest of his life. For instance, taste aversions to citrus fruits may contribute to ascorbate deficiency.
- Learned taste aversions are paralleled in effect by Food faddism. Some avoid all foods grains, where as others avoid poultry skin because of its cholesterol content.

LOCAL EFFECT OF FOOD ON THE INTEGRITY OF PERIODONTAL TISSUES

Gingival Circulation

Foods of firm consistency will increase the number, distribution and tone of the capillaries [7,8]. The circulation of gingival will be improved because the hard foods will mechanically improve and increase the interchange of nutrients between blood and tissues. In short, this will improve the metabolism and vitacity of gingiva.

Epithelium

The degree of keratinization of stratified squamous epithelium which affords protection against trauma or other injurious agents is affected by the frictional qualities of diet. Without this protection, chemical and bacterial irritants can make in roads into the gingival tissue and produce inflammation [9].

Periodontal- Ligament

Chewing, by its mechanical action, produces a compression and expansion of periodontal spaces around the teeth which, in turn, stimulates the removal of waste products through the venous system and lymphatics and the entry of nutrients into the periodontium via arterial system [10].

Alveolar-Bone

The maintenance of proper balance between bone resorption and new bone formation is materially aided by hard foods. Disturbances of this balance by the inadequate function induced by soft food will produce atrophic changes slower the threshold of bone activity.

SYSTEMIC EFFECT OF NUTRITION ON PERIODONTAL TISSUES

On the basis of our present knowledge, the nutrients that can condition the periodontium are: protein, carbohydrates, fats, vitamins and Minerals.

Protein/Calorie and Ca/P levels have been already discussed. The question will arise as to the effects of other factors; these are discussed in the following section.

CARBOHYDRATES AND FATS

Although studies in various experimental animals indicate the high- carbohydrate diets are conducive to the development of severe periodontal lesions, such experiments are difficult to interpret [11,12].

In humans, about 19% of total anaerobic count of organisms of the gingival crevice are streptococci mitis [13]. Carbohydrates, particularly sucrose, are absolutely essential for the proliferation and energy metabolism of these streptococci. These micro-organisms not only appear in gingival crevice but also make up to 28% of bacterial population of dental plaque. These plaque organisms can convert excess dietary carbohydrates into sticky extracellular slimes and intracellular glycogen like polysaccharides which

can produce large masses of dental plaque, producing gingival irritation. There is some indirect evidence that high carbohydrate diets adversely affect gingival health and wound healing. Rats that were fed diet with 70% starch content and low protein showed delay in connective tissue and bone repair after being wounded [14]. There's another study in which rats that were fed on diets containing no protein and 86% sucrose showed severe osteoporosis and increased rate of bone resorption [15].

Fats

Knowledge of the effect of fats on periodontium is very meager, The comparative effects of fat deleted diets, high fat diets (30%) and a normal diet (5%) on the histology of the epithelial attachment, gingival-corium, interdental-papillae, periodontal membrane, cementum, and bone were studied in the rat. It was noted that inflammatory changes occurred in the fat depleted group, but that tissue changes among high fat group were mostly a degenerative type, characterized by gingival inflammation with very thin epithelial covering and loss of tissue detail, irregular fibrosis, bone resorption and the presence of proliferate tissue replacing some of the cementum and bone [16].

Studies have correlated the blood cholesterol levels with periodontal health and gave the following results [17]:

- There's a positive association between CPITN and Cholesterol levels
- There's a negative association between CPITN and HDL cholesterol levels.
- There's a positive association between CPITN and LDL cholesterol levels.

Vitamins

Vitamins are organic substances that the body required in small amounts for its metabolism. Their absence from diets results in deficiency diseases. The human body can't make these essential accessory food substances at least not in sufficient quantity and therefore most acquire them in foods. The vitamins are generally considered under two major subdivisions: water soluble, and fat soluble vitamins.

Water Soluble Vitamins

Vitamin C:- The body stores of ascorbic acid in healthy, well fed men approximate 1500mg and are used at an average daily rate of 3% of existing pool [18]. The effect of ascorbic acid on periodontal health has been investigated more than that of any other nutrient. Dietary deprivation of vitamin-c for 3 months precedes marked depletion of the body stores; at this stage the amount available for daily catabolism is not enough to prevent scurvy. Although 30mg/day is recommended in take for normal persons, a daily intake of 10mg is believed to be necessary to prevent scurvy in healthy adults. This opinion is supported by the observation that a daily dose of only 6.5mg of ascorbic acid produces remarkable improvement in experimentally induced scurvy in adult moles [19].

Among the signs and symptoms often observed in marked ascorbic acid deficiency are - ocular hemorrhages, Sjogren syndrome, femoral neuropathy, impaired wound healing, scorbutic arthritis and gingivitis.

ETIOLOGIC RELATION BETWEEN ASCORBIC ACID AND PERIODONATAL DISEASE

1. Low levels of ascorbic acid influence the metabolism of collagen within the periodontium thereby affecting the ability of tissue to regenerate and repair itself.

2. Ascorbic acid deficiency interferes with bone formation, leading to loss of periodontal bone. Changes that do occur in alveolar-bone and other bones as result of failure of osteoblasts to form osteoid take place very late in deficiency state [20].

3. Ascorbic acid deficiency increases the permeability of the oral mucosa to tritiated endotoxin and tritiated inulin and of normal human crevicular epithelium to tritiated dextran [21].

4. Increasing levels of ascorbic acid enhance both chemotactic and migratory action of leucocytes without influencing their phagocytic activity.

5. An optimal level of ascorbic acid is apparently required to maintain the integrity of the periodontal microvasculature, as well as the vascular response to bacterial irritation and wound healing [22].

6. Depletion of vitamin C may interfere with the ecologic equilibrium of bacteria in plaque and thus increase its pathogenicity.

B-COMPLEX DEFICIENCY

The vitamin B complex includes thiamin, riboflavin, niacin, pyridoxine (B_6), biotin, folic acid, and cobalamin (B_{12}) oral disease is rarely due to a deficiency in just one component of B-complex group; the deficiency is generally multiple.

Thiamin

Also known as “antineurotic factor” due to its antagonistic pharmacologic action against acetylcholine. Deficiency results in a disease called ”beri-beri”, a condition marked by multiple neuritis, edema, and serous effusion. Oral manifestations include [23]:

- Hypersensitivity of the teeth and oral mucosa.
- Gingival may become “dusty-rose” in colour.
- Loss of gingival stippling.
- Aphthous ulcerations.

Riboflavin Riboflavin deficiencies affect the ectodermal tissues. Ocular, dermal and mucosal lesions occur. Angular-cheilosis is common but nonspecific since other factors in the vitamin B complex as well as microbial infections can result in a similar condition.

Nicotinic Acid

Deficiency of niacin or of tryptophan is responsible for pellagra. Gingivitis, attributable to deficiency of niacin, is characterized by extremely painful, wedge-shaped, punched out ulcers involving the interdental-papillae and marginal gingiva. The lesions in humans are necrotic, exudative, and foul smelling. In dogs severe inflammatory changes in the oral mucosa, including the sulcular epithelium, capillary dilatation, and osteoporosis of alveolar bone, have been reported [24].

Folic Acid

Folic acid deficiency is characterized by lesions in cells with rapid rate of renewal, which demonstrates the importance of this vitamin in the synthesis of DNA. Folic acid deficiency in animals demonstrate

- Necrosis of gingiva
- Necrosed periodontal ligament
- Necrosis of alveolar bone

Although there is necrosis, there are no signs of inflammation [25]. The absence of inflammation is the result of deficiency induced granulocytopenia. In a series of human studies, a significant reduction of gingival inflammation has been reported after systemic or local use of folic acid, when compared with placebo [26,27].

FAT-SOLUBLE-VITAMINS

Vitamins A, D, K and E are fat soluble vitamins required in human diet. This section describes observations of deficiency of these vitamins.

VITAMIN A DEFICIENCY

Deficiency of Vitamin A results in dermatologic, mucosal, and ocular manifestations.

In experimental animals, Vitamin A deficiency results in hyperkeratosis and hyperplasia of gingiva with a tendency for increased periodontal pocket formation. The following periodontal changes have been reported in vitamin A-deficient rats:

- Hyperplasia and hyper keratinization of gingival epithelium with proliferation of junctional epithelium.
- Retardation of wound healing [28].

VITAMIN D

Vitamin D, or calciferol, is essential for the absorption of calcium from the gastrointestinal tract and the maintenance of the calcium-phosphorus balance. The effect of deficiency of imbalance on periodontal tissues of young dogs results in:

- Osteoporosis of alveolar bone
- Osteoid that forms at normal rate but remains uncalcified.
- Failure of osteoid to resorb, which results in its excessive accumulation.
- Reduction in width of periodontal ligament space.
- A normal rate of cementum formation, but defective calcification and some cementum resorption.
- Distortion of growth pattern of alveolar bone.

Little is known regarding involvement of human periodontal tissues in hypervitaminosis-D. Among the

outstanding features seen in young rats subjected to hyper vitaminosis-D are large numbers of enlarged osteocytes engaged in lacunar resorption, enlarged cementocytes with presence of cementoid, and increased amounts of connective tissue fibers of periodontium. Calcification of periodontal ligament fibers and ankylosis were noted in rats fed on excessive amounts of Vitamin-D [29].

VITAMIN K

Vitamin K compounds have been found to be required for growth of Bacteroides melaninogenicus, an organism closely associated with periodontal disease [30]. It is speculated that a suitable antimetabolite of Vitamin K might interfere with the growth of this organism, and consequently, prevent the occurrence of periodontal disease [31].

VITAMIN E

The prominent deficiency sign in humans is increased tendency to hemolysis and affect the cross linking of collagen [3].

MINERALS

Calcium and Phosphorus

Calcium and phosphorus have been discussed earlier with regard to nutritional secondary hyper-parathyroidism. Among the findings in calcium deficient young rats are:

- Osteoporosis of alveolar bone.
- Reduction in amount of secondary cementum.
- Reduction in size and number of periodontal fibers.

In rats fed diets deficient in phosphorus, slight rachitic and osteomalacic alterations were observed in young and adult animals [3].

Iron and Other Minerals

Abnormalities of the mouth such as angular Cheilitis and atrophic changes on the dorsum of tongue and buccal mucosa have been noted in iron deficiency. It is also worthy to not that one of the two enzymes intimately involved with antimicrobial activities of the phagocytic cell is an iron-containing enzyme called myloperoxidese. Higashi and Coworkers showed that patients with iron deficiency have a deficiency of this enzyme and a consequent decrease in bactericidal activity. The possible effects of such a situation in terms of the defense of periodontal tissues against plaque microorganisms should not be overlooked.

IMPLEMENTATION OF CLINICAL NUTRITION IN PERIODONTAL PRACTICE

To implement an effective clinical nutrition program in periodontal practice, the clinician must appreciate the role of diet. The practitioner must known how to evaluate the nutritional status of a patient and how to perform a comprehensive nutritional analysis. Finally, the clinician must employ counselling and supplementation techniques when required.

The nutritional status of the host is not always a factor in periodontal disease. In fact, relatively few such patients can be identified. Although routine dietary modification or supplementation for all patients would be inappropriate, it would be inconsistent to examine the patient for endocrinologic, occlusal and psychosomatic factors while ignoring nutrition.

Triphasic Nutritional Analysis

A number of integrated systems for nutritional analysis and therapy in dental practice are available. None of these systems has been sufficiently tested to warrant comparisons. One system, however, does have the distinct advantage of offering several options or “phases” so that the clinical nutrition program can be tailored to the needs of the patients. This system is called triphasic nutritional analysis.

The first phase includes the standard medical/social history, clinical examination and a qualitative dietary analysis. Many of these procedures are a part of the process of diagnosis in the dental office. It is only necessary to include nutritional aspects to gain important insights about the status of the patient. In addition, a careful clinical examination may reveal some of the signs of nutritional deficiency. Particularly important in the clinical examination is the oral bioassay. This assay, which is clearly based more on “art” than “science”, is the subjective comparison of the amount of plaque present around the teeth and the degree of periodontal destruction. Any exaggerated response of the periodontal tissues to the amount of local irritant should increase the practitioner’s suspicion of systemic involvement and suggest the need for nutritional diet diary, taken over several days, which can be transformed into diet patterns and food groups. This transformation is difficult and should be conducted in constrained hygienist. If the clinician does not

employ a trained auxiliary, the patient may be referred to a consulting nutritionist or a computer-assisted diet analysis (described next) may be performed.

The second phase of the triphasic nutritional analysis includes a semiquantitative dietary analysis and routine blood chemistry with differential blood count and glucose tolerance test. This second level of analysis is required when, based on the first phase, the following conditions are noted: (1) the diet appears to be inadequate, but not clinical signs of malnutrition are present; (2) the diet appears adequate, but the patient may have increased needs because of a stress factor: or (3) the clinician desires to correlate specific nutrient intakes with clinical evidence.

The semiquantitative analysis is a computer-based extension of the diet diary in which the intake of various nutrients is estimated more accurately. This analysis should be based on a diet diary and not the less accurate, frequency intake questionnaire. The analysis combined with routine laboratory blood tests is useful is diagnosing nutritional anemias. Clinical trials, periods during which the patient is supplemented with a specific nutrient, are useful adjuncts to phase two analysis. These trials provide a basis for correcting the problem and serve to confirm the diagnosis.

The third phase of analysis, reserved for complex metabolic problems and usually conducted in consultation with a physician. Other publications discuss dietary modification and supplementation techniques.

The complexities of accurate nutritional diagnosis underscore the fact that the casual approach of many practitioners to nutrition is an inadequate basis for diagnosis. A simple question concerning diet no longer meets the ethical obligation for nutritional analysis in periodontal practice.

CONCLUSION

Many controversies concerning the role of nutrition in periodontal disease and problems in the design and analysis of nutrition-periodontal disease studies have been reviewed. In addition, recent advances in the interactions of nutrition, host defence, and infection have been conceptually related to the pathogenesis of periodontal disease. Finally, nutritional factors relevant to the diagnosis and clinical management of patients with periodontal disease have been discussed. The concepts developed with respect to experimental design and the host defences approach to periodontal disease, will hopefully serve to encourage definitive studies, which may delineate a more precise role for nutrition in periodontal disease.

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