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Review Article

Nutritional aspects of oral disorders- A review

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ABSTRACT

The relationship between nutrition and oral health is multifaced. Nutrition has both local and systemic impacts on oral cavity. The oral cavity is often the first sites where nutrient deficiencies can be clinically noted. Any alterations in the structure & function of oral cavity may compromise intake and contribute to the development of a nutrient deficiency state. The present article reviews all the clinical manifestations of nutrient deficiencies that can have significant impact on the function of the oral cavity.
"Let thy food be thy medicine and thy medicine be thy food"- Hippocrates (400BC)

1. Introduction

Nutrition is related with assimilation of food and its effect on the metabolic processes of the body. According to the W.H.O: "Nutrition is the science of food and its relationship to health"[1,2] It is a science that examines relationship between diet & health. Malnutrition is the condition that develops when the body does not get the right amount of the vitamins, minerals, and other nutrients which it needs to maintain healthy tissues and organ functions.

The concept of oral health correlated to quality of life comes from the definition of health that the WHO gave in 1946. Health is understood to be "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity".[3]

There exists a biunique relationship between diet and oral health: a balanced diet is correlated to a state of oral health (periodontal tissue, dental elements, quality, and quantity of saliva). Vice versa an incorrect nutritional intake correlates to a state of oral disease.[4–7]

Oral health is an important determinant of overall health, and can impact and be impacted by dietary and/or nutritional factors. Therefore, all healthcare professionals need to understand the potential relationships among nutrition, oral health, and general health and adopt an interdisciplinary approach to provide optimal patient care.[8] Nutrition plays a fundamental role in health & the dental community has the opportunity to be a critical link between discovery & wellness. Many of the world's most significant health problems are linked to poor dietary practices, under nutrition & over nutrition.

Thus, the evidence base for casual links between nutrition & specific disorders including oral diseases is under exploration. As oral medicine specialists (dentists) are in prime position to screen patients for many disorders relative to nutrition & provide appropriate referrals in health care systems.

The aim of the present review is to expose the best cutting edge science that links nutrition with oral disorders & that supports the evidence based integration of scientific

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advances in nutrition into clinical practice of dentistry. The beneficiary will be the dental patient & the outcome will be prevention of disease & promotion of an integrated health care system.

2. Discussion

2.1. Nutritional risks in various oral diseases

2.1.1. Recurrent apthous stomatitis (RAS)

Fergusson et al. found that out of 47 RAS patients, 23 were deficient in iron, 7 in folic acid, 6 in Vitamin B12 & in addition 11 had combined deficiencies. Koybasi et al. proposed serum vitamin status to be a predisposing factor for RAS & upto 20% cases may have atleast one hematinic deficiency. They found that 12 out of 34 patients to be deficient in vitamin B12. They proposed that diseases commonly associated with RAS do this through diminishing intestinal absorption of vitamin B 12 resulting in its deficiency.[9] Haisraeli – Shalish et al. studied,vitamin B1 levels in 70 patients with recurrent aphthous stomatitis and in 50 as thiamine pyrophosphate effect on transketolase activity in red blood cell lysates. Low levels of vitamin B1 were detected in 49 patients but in only 2 members of the control group. These low levels were not associated with patient age, sex or underlying disease causing RAS. Their finding suggests an association between thiamine deficiency & RAS.[10]

2.1.2. Periodontal disease

Riya Toger Decker referred that compromised host defense responses associated with malnutrition may make the periodontium more susceptible to infectious organisms that are a normal component of the oral flora. During periods of malnutrition, the magnitude of the inflammatory response is limited, resulting in an impaired host response. This could result in a greater amount of periodontal destruction, leading to a compromised dentition. Along with dental & or pharmacological treatment of the underlying local etiologic factors, nutritional management goals focus on provision of adequate calories, protein and nutrients to promote tissue repair, restoration of the host defense mechanisms, and overall well-being.[11] Nishida M et al. evaluated the role of dietary calcium intake as a contributing risk factor for periodontal disease. The association of low dietary calcium intake with periodontal disease was found for young males and females & older males. The dose response was also seen in females, where there was 54% greater risk of periodontal disease for lowest level of dietary calcium intake (2-499mg) and 27% greater risk in dietary calcium (500-799mg) as compared to those who took 800mg or more dietary calcium per day. They concluded with the results that low dietary intake of calcium results in more severe periodontal disease.[12] Nishida M, Grossi SG et al. Vitamin C has long been a candidate for modulating periodontal disease. Studies of scorbutic gingivitis and the effects of Vitamin C on extracellular matrix and immunologic and inflammatory responses provide a rationale for hypothesizing that vitamin C is a risk factor for periodontal disease. They evaluated the role of dietary vitamin C as a contributing risk factor for periodontal disease & found a relationship between reduced dietary vitamin C and increased risk for periodontal disease for the overall population. They concluded that dietary intake of vitamin C showed a weak but statistically significant relationship to periodontal disease in current and former smokers as measured by clinical attachment. Those taking the lowest levels of vitamin C and who also smoke are likely to show the greatest clinical effect on the periodontal tissues.[13]

Adequate nutrition, along with other host factors, is necessary to maintain resistance to periodontal disease. Although the role of diet and nutritional factors in development of periodontal disease is unclear, it is known that defense mechanisms of gingival and saliva can be affected by nutritional intake and status. For eg. Healthy gum tissue normally prevents penetration of bacteria that can lead to gingivitis. Deficiencies of vitamin C, folic acid and zinc may increase the permeability of gingival tissue, making these clients more susceptible to bacterial plaque that causes periodontal disease. Studies examining vitamin C and calcium intake are at increased risk for periodontal disease. The effect is accentuated in current and former tobacco users with suboptimal vitamin C intakes who show an even greater risk of periodontal disease. Oral health professionals are in a position to identify possible nutrient defiencies by looking for signs like stomtitis, glossitis, burning sensation of tongue, bleeding gingival, angular cheilosis and oral ulcerations.[14]

2.1.3. Dental caries

Selwitz, Ismail, et al. suggested that caries is related to one's lifestyle and behavioral factors. These factors include poor oral hygiene; poor dietary habits i.e. frequent consumption of refined carbohydrates, frequent use of oral medicines that contain sugar; and inappropriate methods of feeding infants. Other factors related to caries risk include poverty, depression or social status, number of years in education; dental insurance coverage, use of dental sealants; use of orthodontic appliances and poorly designed or ill-fitting partial dentures.[15]

Marshall TA, Eichenberger-Gilmore JM, et al. suggested that children with caries had lower family incomes, less educated parents, heavier mothers and higher soda-pop intakes at 2, 3 and for 1-5 years than children without caries. 'Overweight' children had less educated fathers and heavier parents than 'normal' weight children. Children 'at risk' of overweight had higher caries rates than 'normal' or 'overweight' children. Thus, they conducted caries and obesity coexist in children of low socioeconomic status.

Public health measures to improve dietary education and access to appropriate food stuffs could decrease the risk of both diseases.[16]

2.1.4. Angular cheilitis

Although the disease has an unknown etiology, the sores of angular cheilitis may become infected by the fungus. Candida aibicans (thrush) or toher pathogens. Studies have linked the initial onset with nutritional deficiences, namely riboflavin, vitamin B12 and iron deficiency anemia, which in turn may be evidence of poor diets or malnutrition (e.g.- celiac disease). Zinc deficiency has also been associated with angular cheilitis. Cheilosis may also be part of a group of symptoms (upper esophageal web, iron deficiency anemia, glossitis and cheliosis) defining the condition called Plummer-Vinson syndrome (aka Paterson-Brown-Kelly syndrome).[17] Don Levy referred that Cheilitis may also be caused by a lack of vitamin B2 (riboflavin), B3 (niacin), B6 (pyridoxine) or B12 (cyanocobalamin) along with a deficiency in iron or an immune system that has been weakened by something such as HIV. Toothpastes and cosmetics have also been known to cause this lip problem. Even some foods and drinks such as orange juice can make the problem worse. If there is lack of B vitamins it can be cured by either supplements or by focusing on foods rich in B vitamins such as dairy products, cereals and leafy vegetables for B2. For B3 and B6 eat foods such as beans, rice, fish, eggs, etc.[18]

2.1.5. Burning tongue

A burning tongue is a constant burning sensation in all or part pf the tongue. There are two medical names, glossodynia and gossopyrosis. If there is also burning in other areas it is called burning mouth syndrome. The patient may or may not have decreased or altered taste. Being deficient in nutrients, such as iron, zinc, folate (vitamin B9), thiamin (vitamin B1), riboflavin (vitamin B2), pyridoxine (vitamin B6) and cobalamin (vitamin B12), may affect the oral tissues and cause a burning mouth.[19]

Painful burning tongue is characterised by inflammation and defoliation. The loss of filiform papillae produces a painful erythematous and granular apperance of tingue and eventual complete atrophy of papillae produces a smooth or blad tongue. The condition has been associated with vitamin B1, B2, B6, B12 or folic acid deficiency.[20] Burning tongue can be a very irritating and painful symptom of menopause. Burning tongue affects women seven times as often as men. Women going through hormonal transitions such as the time leading upon to menopause, are at an even greater risk of developing the symptom, because hormonal imbalance is known to cause burning tongue.[21]

2.1.6. Osteoporosis

Edwards BJ, Migliorati CA referred that osteoporosis and related fractures are more common than coronary disease, stroke and breast cancer. Fractures resulting from osteoporosis can effect patient's quality of life severely, and fractures result in functional impairment and increased health care cost and mortality. Medical management of osteoporosis includes diet control, with appropriate intake of calcium and vitamin D, weight-bearing exercise, discontinuation of tobacco and alcohol intake, and use of medications, including selective estrogenreceptor modulators, calcitonin, anabolic agents and bisphosphonates. Bisphosphonates have been associated with the development of osteonecrosis of the jaws. Oral health maintenance is important in patients with osteoporosis.[22]

Benjamin, Eslick, at al suggested that the social and the economic burden of osteoporotic fractures is increasing worldwide, as the population ages. Calcium alone, or in combination with vitamin D, has been suggested as an inexpensive treatment to prevent osteoporotic bone loss and fractures.[23]

2.1.7. Acure necrotizing ulcerative gingivitis (A.N.U.G)

Folayan MO suggested that in developing countries, however, the condition is commonly diagnosed clinical lesion because of the persistently poor nutritional status. Because of the current campaign for increased focus on global health issues, ANUG, a lesion of significant interest for the developing countries where malnutrition is high and for developing countries because of the AIDS, a global pandemic has resurfaced.[24]

2.1.8. Drugs

Drug usage has the potential of adverse side effects, interactions with other medications and also interactions with other medications and also interactions with other medications and also interactions with nutrient intake and nutritional status of the individual taking the drug. Dugs have direct effect on a person's nutrient intake, metabolism and can induce an actual clinical nutritional deficiency. Drug-induced malnutrition is most likely to develop in those patients receiving long-term drug therapy who take medication to suppress symptoms of a chronic disease. Clinical nutritional deficiency states related to drug intake usually result from a combination of factors: patients who have a marginal nutritional status before the drug is prescribed, patients who have a catabolic disease (weight loss of their ideal body weight), poor dietary intake (alcoholism, anorexia, colitis or irritable bowel syndrome), increased nutritional requirements as a result of a recent major operation, serious bout with the flu, pneumonia, AIDS, cancer, prolonged periods of stress (physical and emotional) etc. those who tall into these categories are

more likely to be in nutritional jeopardy. Drugs can directly affect the intake of food nutrients through their influence on the appetite. As an example, amphetamines act on the central nervous system to suppress the appetite, however the individual usually develops a tolerance to the suppressive effect after about 10 days. These same families of drugs are used to control behavioural problems in hyperactive children.[25]

2.1.9. Bleeding diathesis

Pernicious anemia (also known as Biermer's anemia, Addison's anemia, or Addison-Biermer's anemia) is a form of megaloblastic anemia due to vitamin B12 deficiency, caused by impaired absorption of vitamin B-12 due to the absence of intrinsic factor in the setting of atrophic gastritis, and more specifically of loss of gastric parietal cells. Vitamin B-12 cannot be produced by the human body, and must therefore be obtained from diet. Normally, dietary vitamin B-12 can only be absorbed by the ileum when it is bound by the intrinsic factors produced by parietal cells of the gastric mucosa. In pernicious anemia, this process is impaired because of loss of parietal cells, resulting in insufficient absorption of the vitamin, which over a prolonged period of the time ultimately leads to vitamin B-12 deficiency and thus megaloblastic anemia. This anemia is a result of the body's inability to produce DNA in sufficient quantities for blood cell synthesis, due to interruption of a biochemical pathway that is dependent on vitamin B-12 and/or folic acid as cofactors, which synthesizes thymine, a DNA component. Iron deficiency anemia occurs when the dietary intake or absorption of iron is insufficient and hemoglobin, which contains iron, cannot be formed. Women of childbearing age have iron deficiency anemia, compared with only 2% of adult men. The principal cause of iron deficiency anemia in premenopausal women blood is lost during menses. Iron deficiency anemia can be caused by parasitic infections, such as hookworms. Intestinal bleeding caused by hookworms can lead to fecal blood loss and heme/iron deficiency. Chronic inflammation caused by parasitic infections contributes to anemia during pregnancy in most developing countries. Iron deficiency ranges from iron depletion, which yields little physiological damage, to iron deficiency anemia, which can affect the function of numerous organ systems. Iron deficiency anemia is characterized by pallor (reduced amount of oxyhemoglobin in skin or mucous membrane), fatigue and weakness. Because it tends to develop slowly, adaptation occurs and the disease often goes unrecognized for some time. In severe cases, dyspnea (trouble breathing) can occur. Unusual obsessive food cravings, known as pica, may develop. Pagophagia or Pica for ice is a very specific symptom and may disappear with correction of iron deficiency anemia. Hair loss and lightheadedness can also be associated with iron deficiency anemia.[26]

2.2. Habits

2.2.1. Smoking

The results of the study showed that smoking reduced levels of 25-hydoxy Vitamin D and 1,25-dihydroxy Vitamin D by 10%, and levels of parathyroid hormone by 20% in addition, smokers had a 50% greater incidence of suboptimal vitamin D status compared with non-smokers. It can be seen that cigarette smoking has a significant effect on calcium and vitamin D metabolism, which cannot be explained by other lifestyle factors. Vitamin E is believed to inhibit the oxidation of LDL and VLDL thereby preventing the buildup of atherosclerotic plaques. To look at this further, American scientists carried out a two-month trial to examine lipoprotein oxidation in 40 middle-aged smoking men each supplementing with 200mg of natural Vitamin E daily. Compared to the placebo group, those receiving the active supplement had elevated levels of Vitamin E in their LDL and VLDL. The elevated levels were shown to increase their resistance to oxidation and the likelihood of them "sticking" to the artery walls. This suggests that smokers may reduce their risk of developing heart disease by supplementing with Vitamin E. Smokers have reduced plasma and tissue concentrations of Vitamin C, so smoking leads to both decreased natural antioxidants and an increased oxidative burden.[27]

People who smoke often have deficiencies in numerous nutrients, including zinc, calcium, folate, vitamin C and E, beta-carotene, lycopene and essential fatty acids in the omega-3 and omega-6 families. There are many possible causes for this depletion, including free radicals in cigarette smoke that destroy natural antioxidants; however for some nutrients the most important single cause might be poor diet rather than smoking itself (smokers have on an average a less well-balanced diet than non-smokers). In addition, some evidence suggests that folate or vitamin C supplements may improve arterial function in smokers, thereby potentially helping to prevent heart disease high dosage of vitamin E have not proven helpful for preventing heart disease or lung cancer in smokers. However, vitamin E consumption has shown promise for reducing risk of prostate cancer in smokers. For all these reasons, many smokers undoubtedly benefit from general nutritional support in the form of a multivitamin/mineral tablet. However, high doses of the antioxidant vitamin beta-carotene may not be helpful for smokers, and could even cause harm.[28]

2.2.2. Alcoholism

Three types of malnutrition may be observed in alcoholics, primary malnutrition due to decreased intake of nutrients, secondary malnutrition caused by impairment in the digestion and absorption of nutrients and tertiary malnutrition due to an alteration in the ability to convert nutrients to their active coenzyme forms, resulting in

nutritional complications that potentiate the direct effects of alcohol. Excessive drinking depletes the body of nutrients leading to kidney damage and cirrhosis of the liver and ultimately to death. Alcohol has no net benefit to health and is not an essential nutrient in any way. Impairments in the metabolism of vitamin A have been reposted in alcoholics. Even moderate alcoholic disease is associated with severely decreased vitamin A concentrations. Riboflavin, niacin, vitamin D and vitamin E deficiency as well as lower serum levels of ascorbic acid have been documented in alcoholics. Alcohol also inhibits the absorption of vitamin B6 and its release from the liver. Folate deficiency is probably the most common vitamin deficiency among alcoholics. Alcohol directly inhibits enzymes involved in folate metabolism and increase urinary excretion of folate. Nutritional changes have been used during the withdrawal or detoxification stage of drugs cessation Treatment and for Re- nutrition to enhance withdrawal from alcohol. Dietary supplementation helps restore brain chemicals modified by alcohol. Because of multiple nutrient deficiencies associated with alcoholism and heavy drinkers who quit alcohol should supplement with a high potency multivitamin for at least several months after the detoxification period.[29]

Alcoholics often eat poorly and limit their supply of essential nutrients and affect both energy supply and structure maintenance. Furthermore, alcohol interferes with the nutritional process by affecting digestions storage utilization and excretion of Nutrients. Alcohol inhibits the breakdown of nutrients into usable molecules by decreasing secretion of digestive enzymes from the pancreas. Alcohol impairs nutrient absorption by damaging the cells lining the stomach and intestine's and disabling transport of some nutrients into the blood. In addition nutritional deficiencies themselves may lead to further absorption problems for example folate deficiency alters the cells lining. The small intestine which in turn impairs absorption of water and nutrients including glucose sodium and additional follow it. Even if nutrients are digested and absorbed alcohol can prevent them from being fully utilized by altering their transport storage and excretion. Decreased liver stores of vitamins such as vitamin a and increased excretion of nutrients such as fat indicate impaired utilization of nutrients by alcoholics. Even when food intake is adequate alcohol can impair the mechanisms by which the body controls blood glucose levels resulting in either increased or decreased blood glucose. In non-diabetic alcoholics increased blood sugar or hypoglycaemia caused by impaired insulin secretion is usually temporary and without consequence. Decreased blood sugar or hypoglycaemia can cause serious injury even if this condition is short lived. Hypoglycaemia can occur when a fasting on malnourished person consumes alcohol.

When there is no food to supply energy stored sugar is depleted and the products of alcohol metabolism inhibit the formation of glucose from other compounds such as amino acids. As a result alcohol causes the brain and other body tissues to be deprived of glucose needed for energy and function. Alcoholics even without liver disease 10 to have clinical or laboratory signs of deficiencies in certain vitamins particularly vitamin B1 that is the thiamine, vitamin B2 (ribosomes) and bitaminB6 (pyridoxin) and vitaminC (ascorbic acid) as well as folic acid. The severity of these deficiencies correlates with the amount of alcohol consumed and with the corresponding disease in vitamin intake.

2.2.3. Tobacco chewing

Oral submucous fibrosis is a chronic disease which is commonly found in patients in the Asian subcontinent and as the Far East and is characterized by the buildup of constricting bands of collagen in the cheeks and adjacent structures of the mouth. The precise cause is unknown but chewing of betel quid as well as other areca nut containing products excessive use of chilies and spices or poor nutrition and vitamin and identification C have been suggested.[30] Rajendran et al. who reported that vitamin and iron deficiency together with malnourished state of the host leads to Dearrangement in the inflammatory repetitive response of the laminar propria with resultant effective healing and scarification which ultimately leads to OSMF.[31]

Leukoplakia is white patch or plaque which cannot be scrapped off clinically or pathologically as any other disease except caused by use of tobacco. One of etiology implicated in its development is nutritional deficiencies. Vitamin A and its analogues have been tried in its management.[32]

3. Conclusion

There is a clear synergy between oral health and nutrition; nutrition plays a crucial role in maintaining in integrity and normal function of the oral cavity. Nutrient deficiencies and oral manifestations of disease which alter that integrity contribute to compromised dietary intake, resulting in deficiency states, malnutrition and poor wound healing following procedures or surgeries. It is the responsibility of us as health professionals to be alert to risk factors for and clinical signs of nutrient deficiencies in our patient populations. Collaborative approaches to patient assessment, management and referral are critical to the emerging role in today's scenario as a health care professional.

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5. Conflict of Interest

The author declares that there is no conflict of interest.

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