Journal of Indian Dental Association Madras (JIDAM), 2024; Volume No: 11 , Issue No: 4

Review Article | ISSN (O): 2582-0559

Dental Caries And Periodontal Disease– Nutrition And Oral Health

Revant H Chole[1], Swati Balsaraf[2*]

[1]Department of Oral and Maxillofacial surgery and Diagnostic Sciences, Faculty of Dentistry,Najran University, Saudi Arabia.
*[2]Department of Public Health Dentistry, Ex- professor , College of Dental Sciences & Hospital, Rau, Indore, India. *Corresponding Author's Email: smilecare123@rediffmail.com

Abstract

Oral health is important for overall health of the body. We are what we eat. We should be aware of the nutrition and balanced diet we take. Numerous oral health disease are a result of lack of knowledge, care and poor diet. Diet should be rich in micro as well as macro nutrients. In this article we have discussed various factors associated with the common oral diseases. It's causes and role of nutrition. The role of sugars in dental caries is well known but still it's prevention is not controlled due to it's multifactorial behaviour. Periodontal diseases which are accumulated over the years and present late in life has many risk factors which are discussed at length.

Introduction

Oral health depends on the same nutritional factors as the overall bodily health. It includes routine maintenance, a healthy non sedentary lifestyle and nutritional food.

Healthy nutrition influence the progression and development of oral diseases and conditions such as enamel erosion, gingivitis, periodontal disease and dental caries.1

Impact of nutrition on oral health-

CARIES: The standard classification of sugars by Committee on Medical Aspects of Food Policy (COMA) is based on the location of sugar molecule within the food or drink.

Total sugars

Intrinsic Sugar: Sugar molecules inside the cell e.g. fresh fruit & vegetables.

Extrinsic Sugar: Sugar molecules outside the cell. This is of two types

MILK SUGARS: E.g. lactose in dairy products.

NON-MILK EXTRINSIC SUGARS (NMES) e.g. table sugar, confectionery, honey fruit juice.

According to Rugg Gunn 1993 NMES are highly cariogenic. Milk extrinsic sugars are virtually non cariogenic. Non- sugar sweeteners are non cariogenic.According to the recommendations on diet and caries the amount of NMES must be reduced and should be taken with meals in less frequency. Consumption of intrinsic sugars such as fresh fruits, vegetables, should be increased to 5 pieces/ portion of fruit or vegetable / day.According to Department of Health 1989; WHO 1990 the energy provided by Non milk extrinsic sugar (NMES) should not exceed 10% of total energy in diet.2

Various types of sugars available in eatable products

Coupling Sugar: When a mixture of starch & sucrose are allowed to act upon by enzyme cyclodextrin glucosyltransferase, derived from Bacillus megaterium,a mixture of glycosylsucrose, maltosylsucrose, monosaccharide & oligosaccharides is formed which is termed coupling sugar. The acid production by fermentation of this sugar is much less than obtained with glucose & sucrose. Very little insoluble glucans are produced for adherence of S mutans.

Sucrose: it is a non reducing disaccharide. It has a positive optical rotation & can be readily hydrolyzed. Sucrose being fermentable resists bacterial decomposition at higher concentrations.

(Received 12th October 2024; Accepted 17th November 2024; Published 30th November 2024)

Sugar cane & sugar beet are the main industrial sources of sucrose. Sucrose serves in the formation of insoluble extracellular polysaccharides & enhances plaque formation & microbial aggregation on tooth surface. The adhesion of S. mutans is promoted by the glucans which is the most important determinant of virulence of them on smooth surface.

Invert sugar: The resulting mixture of glucose & fructose is known as invert sugar which has a negative optical rotation. It is slightly sweeter than sucrose.3

Plaque pH: Stephans curve:

Robert Stephans 1940 was the first person to demonstrate changes in plaque pH after every consumption of food items which was responsible for demineralization procedure. In his experiments he demonstrated that after a rinse with sucrose solution (10 ml of 10% sucrose solution for 10 seconds). The mixed bacteria in dental plaque are responsible for uptake of sucrose and acid production and rapid drop in pH. pH in the range of 5-5.5 was termed critical pH as at this pH the saliva stops to get saturated with mineral such as calcium and phosphates. Initially there is drop in the plaque pH and it takes almost 20 minutes for the pH to attain its resting value. Depending upon the other factors influencing the drop in pH such as microbial composition of plaque, the type of carbohydrate consumed and rate of diffusion of substrate in plaque it may take even longer 40 minutes for the plaque to achieve the resting pH.20

Buffering System: The buffering agents are bicarbonates, the phosphates and the protein buffer systems.

Bicarbonates are most important because it can buffer rapidly by losing carbon dioxide. Its pK is close to that encountered in plaque & therefore it is more effective. As flow rate increases bicarbonate concentration increases dramatically whereas phosphate falls slightly. After removal of bicarbonate by a current of CO2-free air at pH 5, the buffering capacity of saliva is markedly reduced. Sialin is an argenine peptide which rapidly clears glucose from plaque, increases base formation and increases pH.

The human saliva are supersaturated with calcium and phosphates which is mediated by salivary proteins eg statherin, the acidic Prolin rich proteins, cystatins and histatinshistatins. 3,4

Trace elements:

Zinc: Zinc helps in arresting demineralization of tooth LYNCH RJ.Studies indicate role of zinc in prevention of dental cariesUCKARDES .Lower level of zinc systemically was associated with high caries prevalence ATASOY, however few studies do not support the utility of zinc in the prevention of dental caries.5, 6,7

Strontium: The deficiency of strontium may help form caries.The role of strontium in preventing dental caries was much studied in 1970s and 80s but till date the results of studies are inconclusive.8, 9

Iron: Iron interferes with the sucrose metabolism of streptococcus mutans thus reducing the production of extracellular polysaccharides and inhibits enzyme glucosyl transferase. 10

Saliva: Saliva is a mixture of major and minor salivary glands secretions. Saliva contains inorganic molecules such as electrolytes, mucin, immunoglobulins and various enzymes. Salivary antioxidant system is made of peroxidase, catalase, superoxide dismutase glutathione, peroxidase and small molecules, uric acid, Vitamin E & C. Immunoglobulins acts as defence against bacteria, viruses, fungi and promotes mucosal healing and enzymes. Antioxidants are helpful in prevention and progression of dental caries.15,16,There is a possible connection between dental caries & mucin(MUC1 & MUC5B) in saliva.17The main oral innate defence factors are the peroxidase systems,Lysozyme,Lactoferrin and histatins.they limit bacterial or fungal growth, intereference with glucose metabolism of bacterias, promote aggregation and elimination of bacteria.11

Lactoferrin: Iron binding basic protein.Tends to bind and limit the amount of free iron which is essential for microbial growth, this salivary protein is an active host defense mechanism.

Igs: They could bind to salivary pellicle blocking glucan-induced irreversible adherence or they could agglutinate bacteria. IgGs opsonize bacteria

permitting phagocytosis by polymorphonuclear leukocytes or possibly macrophages.

Lysozyme: Small highly positive enzyme-catalyzes the degradation of the negatively charged peptidoglycan matrix of microbial cell wall. Binds to hydroxyapetite and maintains its activity after binding. Strong ionic interactions with bacterial cell walls and with the mucin in saliva.

Lactoperoxidase: Kills microorganisms-catalyzing the hydrogen peroxide mediated oxidation of a variety of substances in the microbes. High affinity for enamel surface and it forms an important defense mechanism limiting early microbial colonization of tooth surfaces. 12,11

Fluoride dentifrices:-

Tinanoff N 2005 in a CDE program in Chennai in 2005 pointed out the fluoridated dentifrices as the single most important factor for the global decline of caries. Downer reviewed caries prevalence in UK in the 20 yrs from 1973-93 and observed a 53% reduction of caries experience in 5 yr old children. He attributed this to the widespread use of fluoridated dentifrices. Brown regarded widespread use of fluoridated tooth pastes in reduction of dental caries prevalence in NZ. Changing levels of dental caries experience (DMFT) among 12-year-olds in developed and developing countries

Increased dental awareness:-

As stated by the joint working group of FDI and WHO in 1985, the increased dental awareness and the organized oral health education programmes may be few of the most probable reasons for dental caries decline.

- Adoption of preventive approach by the practitioner.
- The criteria for filling the teeth have changed over time with the advent of Minimal Intervention Dentistry (MI), smart restorative materials, Nanotechnology, etc.
- Increased use of professionally applied fluorides.
- Advent of sealants and preventive resin restorations in the routine dental practice.

NUTRITIONAL RISK FACTORS FOR PERIODONTITIS:

Nutritional factors: vitamin deficiency, protein deficiency, starvation: Refined carbohydrate

Leads to Scorbutic, Rachitic, non-detergent diet gingivitis. Vitamin C deficiency leads to bleeding gums. Gastrointestinal disorders, syphilis, nephritis, liver disease, tuberculosis show signs in the mouth.

Physical disabilities: physical disabilities makes a person affected with it with lower neuromotor skills and thus poor maintenance of oral hygiene and thus rendering him more prone to gingival and periodontal disease.

Xerostomia : Xerostomia will lead to retention as well as lower clearance of food particle from mouth and reduced ability of natural oral clearance of food by the action of tongue, lips and cheeks thus increased chances of periodontitis.13

Drug induced disorders: Dilantin sodium used in treatment of epilepsy leads to gingival inflammation & enlargement. Chronic bismuth intoxication shows ulcerative gingivostomatitis.

Psychological or emotional factors: In such conditions a person is high strung and due to emotional upheaval salivary changes such as xerostomia or excess salivation can occur, lack of proper personal care can lead to poor oral hygiene and hence periodontitis or altered gingival conditions. Psychological factors has important role in necrotizing ulcerative gingivitis. Psychosomatic disorders exert their effect on periodontitis by the development of habits that are injurious to the periodontium.14

Metabolic conditions: pregnancy, puberty, menopause: Endocrine adjustments takes place during this time. There is generalized tissue enlargement with discoloration. Bleeding and mulberry like swelling and papillae become bulbous. There is adverse effect on periodontal structure during the course of pregnancy.

Endocrinal disturbances: diabetes mellitus, hyperparathyroidism and hyperthyroidism: The glucose level of saliva and blood in diabetic patients is more than normal which has the potential to alter the microflora towards more pathogenic bacterias. Due to polymorphonuclear

leucocytes deficiencies in diabetes which results in impaired chemotaxis, defective phagocytosis thus making the diabetic individual more susceptible to periodontal infection. Most of the studies show statistically significant relationship between severe gingival inflammation and loss of attachment in diabetic group as compared to normal individuals which is more when there is increase in glucose level. Periodontal disease in diabetics presents with very severe gingival inflammation, deep periodontal pockets, rapid bone loss, frequent periodontal abscesses, greater loss of attachment, increased bleeding on probing and increased tooth mobility. 15

1,25(OH)2D3 has an important role in maintaining calcium and phosphate level in blood. Its deficiency leads to increased secretion of parathyroid hormone leading to rickets and osteomalacia. In Mice, vitamin D receptor (VDR)-mediated induction of osteoblast RANKL may account for enhanced bone resorption. Osteoporosis results in lowered density in jaw bones leading to altered trabecular bone pattern and rapid alveolar bone resorption followed by invasion of periodontal pathogens. Periodontal infection increases the systemic release of proinflammatory cytokines, which accelerate systemic bone resorption. Hypopitutarism exhibits with crowding of teeth, enlarged gingiva, delayed resorption of deciduous teeth, delayed formation and eruption of teeth. Hyperpitutarism exhibits spacing of lower teeth-jaw size. Thus people with poor metabolic control have higher frequency of gingivitis and periodontitis.16

Hematologic disorders: There is occurrence of hemorrhagic gingival overgrowth with or without necrosis as a common early manifestation of acute leukemia. Chemotherapy associated with bone marrow transplantation also adversely affect gingival health.

Immune system disorder: leukocyte disorder, antibody disorders, HIV infection, persons on immunosuppressive drugs: Bacterial infections acts as a stimulus for systemic acute phase response resulting in increased production of acute phase proteins like C Reactive protein(CRP), macroglobulin and Serum amyloid. The bacterial endotoxins also stimulate the local host inflammatory mediators which finally results in serum antibody response to bacteria. Thus if the immune system is compromised it will be a risk factor for developing periodontitis. HIV infected patients exhibit Kaposi's sarcoma, necrotizing ulcerative gingivitis, necrotizing ulcerative stomatitis, necrotizing ulcerative periodontitis which results in inflammation and enlargement of gingiva.17

Stress: stress, depression and anxiety are considered potential risk factors to affect periodontal condition as these factors are risk indicators to take up health impairing habits such as smoking, tobacoo chewing, alcoholism, poor oral hygiene status and the pathophysiological factors that lead to higher glucocorticoid and catecholamine level which affects the immunological profile thus making the individual susceptible to periodontitis.10 stress related habits such as pencil biting are detrimental to periodontal health.17

Obesity: stress has been marked as a potential risk factor for uptaking unhealthy lifestyle which includes uptake of unhealthy food habits such as refined carbohydrates, fast food, which promotes plaque retention and thus periodontal and gingival diseases.18

Cardiovascular disorders: There is increase in C-reactive proteins and fibrinogen in periodontitis. Furthermore there is an increase in level of systemic markers of inflammation such as C-reactive protein and IL-6 in cardiovascular disease. Bacteremia from periodontitis and dental disease is known to be the primary cause of infective endocarditis.SALIVA: several inorganic and organic factors in saliva are important for bacteria and their products in oral environment. Saliva like GCF contains antibodies that are reactive with indigenous oral bacterial species. Salivary enzymes such as hyaluronidase , lipase, beta-glucuronidase, chondroitin sulfatase are present in increased concentrations in periodontal disease. Proteolytic enzymes in the saliva are generated by both host and oral bacteria which are contributors to the initiation and progression of periodontal disease.To combat these enzymes saliva contains antiproteases that inhibit cystine proteases such as cathepsins and antileucoproteases. Saliva contains coagulation factors that hastens blood coagulation and protects wound from bacterial invasion. Saliva exerts a major role in plaque initiation,

maturation and metabolism. Calculus formation, periodontal disease and caries are dependent on salivary flow. An increase in inflammatory gingival disease, rapid tooth destruction are a consequence of decrease salivary gland secretion.19

Conclusion

Oral health is a gateway to overall health. Mouth is the entrypoint which decides what a person will become as the adage goes we are what we eat. Oral diseases are preventable if proper lifestyle is adapted. A few changes in eating habits can save oral and overall health.

Reference

1. Shafer et al. Textbook of oral pathology; fourth edition.
2. Clifford M. Sturdevant. The art and science of operative dentistry. Third Edition. Harcourt Brace and company Asia pte ltd 1995
3. Newbrun. Cariology. third edition: Quintessence publishing company;1989
4. M.Lenander-Lumikari, V.Loimaranta.saliva and Dental caries.Adv.Dent Res 14:40-47,December 2000
5. ELynch RJ .Zinc in the mouth, its interactions with dental enamel and possible effects on caries; a review of the literature. Int Dent J. 2011 Aug;61 Suppl 3:46-54.
6. The effect of systemic zinc supplementation on oral health in low socioeconomic level children.Uçkardeş Y1, Tekçiçek M, Ozmert EN, Yurdakök K. Turk J Pediatr. 2009 Sep-Oct;51(5):424-8.
7. Atasoy HB1, Ulusoy ZIPediatr Dent. The relationship between zinc deficiency and children's oral health. 2012 Sep-Oct;34(5):383-6.
8. Riyat M1, Sharma DCAnalysis of 35 inorganic elements in teeth in relation to caries formation.. Biol Trace Elem Res. 2009 Summer;129(1-3):126-9.
9. Lippert F1, Hara AT.Strontium and caries: a long and complicated relationship.Caries Res. 2013;47(1):34-49.
10. Cecília Cláudia Costa Ribeiro: The effect of iron on Streptococcus mutans biofilm and on enamel demineralization. Braz. oral res. vol.26 no.4 São Paulo July/Aug. 2012
11. M.Lenander-Lumikari, V.Loimaranta.saliva and Dental caries.Adv.Dent Res 14:40-47,December 2000
12. Peter S. Essentials of Preventive & Community Dentistry. 4th Edition. Arya Publishers; June 2009
13. Naseem Shah. Oral and dental disease: causes, prevention and treatment stratigies. NCMH background papers-Burden of disease in India
14. JB John,V Prabhurajan,A.Stalin,M.Krishnan. Masochistic habits in a child patient: A case report and its management. Int J Crit Illn Inj Sci 2013;3:211-3
15. Sammartino G, M.Tia, Bucci T, Wang HL. Prevention of mandibular third molar extraction-associated periodontal defects: a comparative study. J.Periodontol 2009 Mar;80(3): 389-96
16. Y Amano, K Komiyama, and M Makishima. Vitamin D and periodontal disease. Journal of Oral Science, Vol. 51, No. 1, 11-20, 2009
17. A.Dilsiz,and T.Aydin. Self Inflicted gingival injury due to habitual fingernail scratching: A case report with a 1 year follow up. Eur J Dent Apr 2009;3(2):150-154
18. GMG Patricia,Am Juliane , APC Cristiane, HCSP Silvia. Obesity and periodontitis; Systematic review and Metaanalysis.Cienc.Saunde coletiva Vol. 19 No. 6, Rio. De Janciro. June 2014
19. Newman MG. Takei HH, F.A Carranza 's clinical periodontology.Ninth edition,Saunders 2006 pg 138-142;pg 105-106