Article Review

CLINICAL MANAGEMENT OF SALIVARY DEFICIENCY A REVIEW ARTICLE

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توفر الخصائص المختلفة للعاب الفم بعض الحماية الطبيعية للأسنان ضد التسوس. لذلك عندما تنشأ نقص في كمية اللعاب أو اختلاف شديد في تركيبه يؤدي ذلك إلى تزايد واضح في تسوس الأسنان. وفي هذه الورقة مراجعة لبعض الخواص الدفاعية للعاب وبعض الأسباب المؤدية إلى نقص أو انعدام إفرازه وكذلك طرق المعالجة.

The physical, chemical and antibacterial properties of saliva provide protection to human dentition against dental diseases, Therefore, salivary deficiency has to be managed carefully. The causes of saliva deficiency are many and varied. It is worth mentioning that saliva flow rate is normally affected by physiologic condition, such as eating, resting, sleeping, cold or hot season etc, In this paper the protective role of saliva, etiologiy of saliva deficiency and its clinical management are discussed.

"My mouth and throat are dry, rough and sticky. I cannot wear my dentures. My mouth is always sore. I am hoarse; it is hard to talk, I have to sip fluids frequently so my tongue will not stick to the side or roof of my mouth and teeth. I cannot tell the positon of food in my mouth. I have difficulty tasting, and I have to add salt and sugar to my food. My fillings are falling out and my teeth are crumbling away,"[1] These are some of the complaints usually presented by xerostomic patients, They reflect the major functional roles of saliva; digestive and protective rotes.[1-2]

The physical, chemical and antibacterial properties of saliva provide protection to human dentition against dental caries,[3] The physical flow of saliva, if in sufficient quantity, and with help of muscular activity of the lips and tongue effectively remove a large number of bacteria and food debris from the teeth surfaces,[1,2] Clearance of carbohydrates from the mouth appears to be one of the most important functions of saliva with respect to prevention of dental caries. In addition to this physical effect, saliva also interferes with adherence of bacteria to teeth surfaces. The ability of saliva to minimize bacterial attachment is either by direct action of the secretory IgA system or by the presence of macromolecules, mucins, which mask bacterial adhesins or compete with them for attachment sites. The presence of mucins in saliva also serves as a trap to aggregate bacteria to the point where they can no longer effectively adhere to the oral tissues.[1,4]

Saliva contains a number of proteins which are known to have immediate effects on oral bacteria.[5-8] They are lysozyme, lactoferrin, lactoperoxidase and secretory IgA. Lysozyme can cause lysis of bacterial cell membrane, especially streptococcus mutans by interacting with anions of low-charge density, chaotropic ions, and with bicarbonates. This leads to destabilization of the bacteria! cell membrane probably through the activation of autolysins[6,9] Lactoferrin is effective against bacteria that require iron or copper for their metabolic processes. It can compete with bacteria for iron and copper and deprive them of some of their essential needs.[6,10] By combining with iron and copper, lactoferrin also protects the lysozyme action which is depressed by the presence of these elements.[3] Lactoperoxidase, in the presence of hydrogen peroxide, acts by oxidizing thiocyanate to form hypothiocyanite and hypothiocyanous acid.[1,8] These products in turn affect bacterial metabolism by oxidizing the sulfhydryl groups of the enzymes involved in glycolysis and sugar transport. This property of lactoperoxidase is significantly enhanced by interaction with secretory IgA. Although it needs more clarification, saliva may have some influence on viruses through secretory IgA. This action had been reported in the success of the oral polio vaccine.[1]

Saliva has also another important role of protection against dental caries. It maintains a relatively

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neutral pH in the oral cavity. In the bacterial plaque, where acid is the natural result of bacterial metabolism of carbohydrate, saliva helps to regulate pH in several ways. Phosphate, histidine-rich peptides and particularly sodium biocarbonate, present in saliva, raise the pH in the oral cavity by buffering action.[1,3] The biocarbonate in saliva is also capable of diffusing into dental plaque to neutralize the acid formed from carbohydrate by microorganisms. Furthermore, bacteria break down urea present in saliva to form ammonia which can also neutralize acid.[1,3]

The presence of calcium, phosphorus, magnesium, fluoride and some other trace elements in saliva provides the newly-erupted teeth with necessary ions for their post- eruption maturation. The diffusion of these elements (ions) into the surface enamel increases surface hardness, decreases permeability and increases resistance to caries.[11]

Another important protective role against dental caries played by saliva is the process of remineralization.[12] To understand this phenomena, it is important to remember that the first step in the caries process is the loss of mineral ions from the enamel. Initially, the acid solubilizes the magnesium and carbonate ions, followed by the removal of calcium, phosporus, and other ions that are part of the enamel crystals. This process creates microcavities. The loss of mineral ions from the enamel is termed demineralization. The process of demineralization is reversible under certain conditions. There is a continuous exchange of minerals between enamel surfaces and the oral environment. The direction of mineral movement is governed by the relative mineral concentration and the pH at this interface. Dental caries occurs if the amount of ion removal surpass the redeposition of minerals. However, when the net flow of mineral salts is back into the enamel from the oral fluids, repair or healing of pre-existing microcavities occurs. This process is known as remineralization and it enables the repair of enamel rod structure following acidogenesis. Saliva, because it contains calcium and phosphate, serves as a potential source for the minerals required in this process. Fluoride, if present, will enhance the process of remineralization.[12] Although saliva contains a minute amount of fluoride, plaque usually contains fluoride from previous demineralization.[13,14]

Etiology of Salivary Deficiency

The causes of saliva deficiency are many and varied. Salivary flow rate is normally affected by physiologic conditions, such as eating, resting, sleeping, cold or hot seasons etc.[3,15] However, there are certain conditions in which salivary flow is greatly depressed. Patients suffering from dehydrating diseases such as fever, diabetes, prolonged diarrhea, anemia, uremia, blood disorders like sideropenic anemia and some other systemic conditions have a dramatic reduction in salivary flow.[16,17]

Neuroleptics, sedatives, diuretics, hypnotic, anticholinergic and many other drugs may cause salivary flow deficiency. These drugs affect salivary flow by mimicking the autonomic nervous system actions or by directly acting on the cellular processes necessary for salivation. Drugs can also indirectly affect salivation by altering the fluid and electrolyte balance or by affecting blood flow to the glands.[18]

There are certain local diseases that affect salivary glands and cause reduction in salivary flow. Chronic sialadenitis more commonly affects the submandibular and the parotid glands. This disease causes degeneration of the acini and ductal obstruction.[16] Salivary gland cysts and tumor, both benign and malignant, can cause compression of the ductal structures of salivary glands and thus affect salivary flow. Sjorgen's syndrome, a chronic inflammatory and autoimmune disorder in which the acinar cells of the salivary glands are replaced by lymphocytes also causes xerostomia.[19]

The salivary gland parenchyma is sensitive to irradiation. Radiotherapy of head and neck malignant lesions has been found to cause varying degrees of permanent damage to the salivary glands exposed to the path of radiotherapy. Histologically, irradiated salivary glands undergo initial edema followed by varying degrees of fibrosis and fatty generation accompanied by progressive degeneration of fine vasculatures. As the pathology advances, the acini degenerate and necrose, and the glands shrink.[20,21] The return of the salivary function after radiotherapy is extremely variable and is related to the dose received.[18]

Recently, it has been reported that AIDS patients have presented with xerostomia. Furthermore, it has been observed that irradiation therapy to

reduce the discomfort of intra-oral Kaposi's sarcoma can additionally contribute to salivary gland dysfunction in certain patients with AIDS.[22]

Finally, surgical procedures and traumatic injuries can both result in the loss of salivary glands innervation, damage to gland parenchyma, damage to the ducts and/or damage to salivary glands blood supply which may compromise salivary secretion.

Clinical management of the irradiated patient

"Radiation caries is a serious complication of radiation therapy caused by a drastically reduced production of saliva and change in its compositions."[3] For optimal preventive result, dental care for patients receiving radiotherapy should begin prior to the onset of radiation therapy.[3-23] Pre-radiation evaluation of each patient is of paramount importance to the management of these patients. Many *of* the complications *associated with* radiotherapy can be either reduced or eliminated by the establishment of a good working relationship between the radiotherapist and the dentist.[24,25] The patient must be given a full and realistic information about the radiotherapy sequelae.[3,25] Patients must be adequately trained to remove plaque. During the period of plaque control instructions, a daily plaque index should be completed to ensure that it is as close to zero as possible because acceptable plaque index score *for a normal* individual is not satisfactory for patient receiving radiotherapy.[3]

All teeth must be evaluated. The teeth to be retained must be restored to an optimal condition to keep post-radiation complications to a minimum. Teeth with poor prognosis should be extracted at least two weeks prior to radiation treatment to eliminate a probable development of osteoradionecrosis. Patients with poor oral hygiene and motivation may require more teeth extraction than the highly motivated patients.

It is not *an easy task for* a depressed cancer patient to concentrate on complicated dental prophylaxis. These patients present with excessive caries activity related to radiation therapy. Such a patient could be assisted with weekly supportive professional therapy using fluoride-containing tooth paste or fluoride gel application with a fabricated flexible custom-made tray.

Numerous studies have reported the effectiveness of daily self-application of fluoride in the prevention of radiation caries. Sodium fluoride gels (1 %) or stannous fluoride gels (0.4%) were recommended and they were applied with custom tray for five minutes every day. Patients were also instructed to brush and floss their teeth, rinse twice a day with remineralization solution, stimulate salivation by chewing a sugarless chewing gum, and use artificial saliva substitute, containing at least 1 ppm fluoride, to prevent dryness of the mouth.[23,25-27]

Katz's[28] study which was conducted on patients who did not have any oral prophylaxes before radiotherapy, reported that prevention of dental caries and remineralization of incipient existing caries were achieved by professional application of topical fluoride-chlorhexidine solution. Katz's management for irradiated patient with excessive caries activity are as follows:

1. After prophylaxis, teeth are isolated by cotton roll. Then, a 1.0% sodium fluoride - 1.0% chlorhexidine digluconate solution is applied with cotton applicator to each dental arch and will last for four minutes.
2. A series of four applications, once every week, is performed.
3. After each application, the patient is advised not to rinse, drink or eat for 30 minutes.
4. The patient is instructed to rinse his or her mouth every night, after brushing, with a 0.05% sodium fluoride - 0.2% chlorhexidine digluconate solution for one minute.[28]
5. These patients require a frequent recall, after the initial management. Recall visits are first scheduled at 3 weeks, 6 weeks, 3 months, 6 months, and then at 6-month intervals. If the caries appears, the preventive program will be reinstituted.

Furthermore, if radiation-induced caries are extensive and require restorations, all soft caries should be excavated and temporarily treated with zinc oxide/eugenol cement to aid in changing the nature of oral microflora. The permanent restorative material of choice is glass ionomer because of the long term fluoride release from this material. Furthermore, radiation caries usually *affects* cervical and incisal areas in which glass ionomer can fulfill esthetic considerations.[29] Rubber dam isolation may not be necesary because of the reduction in the salivary flow and the possibility of soft tissue injury by rubber dam clamps. Amalgam restoration

is also acceptable in inter-proximal posterior areas because of the inability of glass ionomer to withstand masticatory forces.[30] Surgical removal of decayed teeth should be avoided because of the possibility of development of osteoradionecrosis. If pulpal pathology is involved, the tooth can be treated by conventional endodontic therapy. During treatment procedure, the patient should be given a course of antibiotic as a preventive measure against infection and possible osteoradionecrosis.[29,20]

Conclusion

It is evident from the above discussion that saliva is essential for the maintenance of oral health. Therefore, salivary deficiency has to be managed carefully. A strict and meticulous preventive program, including maintainance of good oral hygiene and standard fluoride application, may minimize some of the salivary deficiency complications. However, more effective methods are needed in some cases.

References

1. Mandel ID. The functions of saliva. J Dent Res 1987;66(Spec Iss):623-7.
2. Schwarz WH. The rheology of saliva. J Dent Res 1987;66(Spec Iss):660-4.
3. Harris N, Cristen A. Primary preventive dentistry. 2nd ed. East Norwalk, CT: Appleton & Lange, 1987:262.
4. Tabak LA, Levine MJ, Mandel ID, Ellison SA. Role of salivary mucins in the protection of the oral cavity. J Oral Patho 1982;11(1):1-17.
5. De Jong MH, Van Der Hoeven JS. The growth of oral bacteria on saliva. J Dent Res 1987;66(2):498-505.
6. TenovuoJ, Grahn E, Lehtonen O, Hyyppa T, Karhuvaara L, Vilja P. Antimicrobial factors in saliva: Ontogeny and relation to oral health. J Dent Res 1987;66(2):475-9.
7. Arnold R, Brewer M, Gauthier JJ. Bacteriocidal activity of human lactoferrin: Sensitivity of a variety of microor-ganisms. Infect Immun 1980;28:893-8.
8. Tenovou J, Mansson-Rahemtulla B, Pruitt K, Arnold R. Inhibition of dental plaque acid production by salivary lactoperoxidase system. Infec Immun 1981;34:208-15.
9. Pollock JJ, Lotardo S, Gavai R, Grossbard B. Lysozyme-protease-inorganic monovalent anion lysis of oral bacterial strains in buffers and stimulated whole saliva. J Dent Res1987;66(2):467-74.
10. Lassiter MO, Newsome AL, Sams LD, Arnold RR. Characterization of lactoferrin interaction with S. mutans. J Dent Res1987;66(2):480-5.
11. Shaw JH. The influence of sialoadenectomy in rats on food and water consumption. J Dent Res 1958;37:805-13.
12. Silverstone LM. Significance of remineralization in caries prevention. J Canad Dent Assoc 1984;50(2):157-67.
13. Silverstone LM. Remineralization phenomena. Caries Res 1977;11(Suppl 1):59-84.
14. Park KK. Associate Professor, Department of Preventive Dentistry, Indiana University, School of Dentistry: Lecture Oct. 15, 1987.
15. Dawes C, Physiological factors affecting salivary flow rate, oral sugar clearance and the sensation of dry mouth in man. J Dent Res 1987;66(Spec Iss):648-53.
16. Tandler B. Salivary gland changes in diseases. J Dent Res 1987;66(2):398-406.
17. Blitzer A. Inflammatory and obstructive disorders of salivary glands. J Dent Res 1987;66(Spec Iss);675-9.
18. Schubert MM, Izutsu KT. Iatrogenic causes of salivary gland dysfunction. J Dent Res 1987;66(Spec Iss):680-8.
19. Talal N. Overview of Sjogren's syndrome. J Dent Res 1987;66(SpecIss):672-4.
20. Shannon IL, Starcke EN, Wescott WB. Effect of radiotherapy on whole saliva flow. J Dent Res 1977;56(6):693.
21. Kaplan P. Mantle irradiation of the major salivary glands, J Prosthet Dent 1985;54(5):681-6.
22. Silverman P. Oral findings in people with or at high risk for AIDS. J Am Dent Assoc 1986;112:187-95.
23. Rothwell BR. Prevention and treatment of the oral complications of radiotherapy, J Am Dent Assoc 1987; 114:316-22.
24. Ritchie JR, Brown JR, Guerra LR, Mason G, Dental care for irradiated cancer patient. Quintessence Int 1985; 12:837-42.
25. Toljanic JA, Saunders VW, Jr. Radiation therapy and management of the irradiated patient. J Prosthet Dent 1984;52(6):852-8.
26. Makkonen TA, Edelman L, Forsten L. Salivary flow and caries prevention in patient receiving radiotherapy. Proc Finn Dent Soc 1986;82:93,
27. Wright WE. Management of oral sequelae. J Dent Res 1987;66(Spec Iss):699-702.
28. Katz S. The use of fluoride and chlorhexidene for the prevention of radiation caries, J Am Dent Assoc 1982;104(2):164-70.
29. Arafa SA. A clinical study of two restorative materials for possivle use in treating post-radiation cervical caries. Master's thesis, Indiana University, School of Dentistry, 1982.
30. Jackson MJ. Treatment of complication of radiation therapy. J of Massachusetts Dent Soc 1980;29:214-21.