Diabetes and Oral Health – A Review.

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ABSTRACT

Oral health is an integral component of the general health. There is increasing evidence to suggest that oral health and systemic health are interrelated to each other. Evidence indicates that diabetes mellitus has a major role in altering the oral environment. The oral manifestation is generally seen in uncontrolled or poorly controlled diabetic patients due to reduction in defense mechanism and the increased susceptibility to infections. Both advanced periodontal disease and dental caries may lead to the loss of teeth. Xerostomia is also a common symptom in diabetics. Hypo salivation and changes in salivary composition may contribute to the increased susceptibility to oral infections, impaired wound healing and increased rate of dental caries in diabetics. Fungal infections are more common in type 1 and type 2 diabetics than in non-diabetics and the oral infections appear to be more severe in diabetics than in non-diabetics. Periodontal disease is considered as a sixth complication of diabetes mellitus. The other frequently encountered clinical signs are, gingival polyps, periodontitis, periodontal abscess, loosened teeth due to bone loss, delayed post surgical healing and striking enlargement and erythema of the attached gingiva.
It is worth to review the available literature so that one can have a comprehensive knowledge and understanding of the oral manifestation related to diabetes mellitus which in turn will help dentists in taking necessary precautions, while delivering oral health care services for patients with diabetes mellitus.

Keywords: Diabetes mellitus, Periodontal disease, Dental Caries

INTRODUCTION

Human beings are suffering from diseases and have been combating with diseases to control its evolution. Even though man has given remedies for many diseases, newer challenges still exist in the form of lifestyle related diseases. Under such disease, diabetes is considered to be a common systemic disease which is threatening human survival.
Diabetes mellitus designates a group of metabolic diseases characterized by hyperglycemia due to insufficient insulin secretion or reduced insulin sensitivity, associated with abnormal glucose, lipid and protein metabolism. The chronic hyperglycemia leads to an increased risk of developing microangiopathy, accelerated atherosclerosis, neuropathy and impaired wound healing.[1] It is well known that diabetes has profound impact on oral health status of an individual. [2]

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Diabetes currently affects almost 200 million people worldwide. International diabetic federation estimates that this figure will increase to 333 million by 2025.[3] India has the highest prevalence of diabetes and it is predicted to become "Diabetic capital of the world". Almost 1/6th of world's diabetic patients are found in India alone and only 10% of these patients in India receive appropriate treatment. [4]

Diabetes mellitus is a clinical syndrome which is characterized by different types.

Type 1 diabetes - Destruction of insulin-producing pancreatic β-cells due to a T-cell mediated autoimmune process. The cause may also be idiopathic and found usually in normal body weight or thin, mainly adolescents aged under 30 years. Often abrupt onset with symptoms of insulin deficiency (polyuria, polydipsia, weight loss, and fatigue), may present with ketoacidosis and exogenous supply of insulin is vital. [1]

Type 2 diabetes - Develops due to a combination of insulin resistance and impaired insulin secretion. Most individuals aged over 40 years, often overweight or obese, few classical symptoms of hypoglycaemia, not prone to ketoacidosis except during stressful periods. Exogenous insulin supply is not vital, but as the disease progresses and endogenous insulin secretion decreases, most patients will require insulin therapy, either alone or in combination with oral hypoglycemic agents. [1]

Other specific type's - Genetic β-cell functional defects, exocrine pancreatic disorders with secondary diabetes, autoimmune endocrinopathies with secondary diabetes, drug-induced diabetes, infection-induced diabetes, etc.

Gestational diabetes - Impaired glucose tolerance occurring during pregnancy. Usually disappears after delivery. Mostly affects women with a familial predisposition to diabetes and who are overweight. Women who have had gestational

diabetes are at increased risk for later developing type 2 diabetes.

Diabetes varies considerably around the world and it is related to differences in genetic and environmental factors. The initial classical symptoms of diabetes mellitus include polydipsia, polyphagia, polyuria, fatigue, weakness, irritability, weight loss and pruritus.

The diabetic complications are related to chronic hyperglycemia, which results in alterations in blood vessels, nerves and connective tissue. Diabetic complications affect eyes, kidneys, nerves, heart, blood vessels and oral cavity.[5]

Several studies have shown that both type 1 and type 2 diabetes mellitus are associated with an increased risk of developing oral diseases, including periodontal disease, oral candidiasis, dental caries, salivary gland hypofunction, sialosis and taste impairment.[6,19]

Conversely, control of periodontal infections seems to have beneficial effects on the metabolic control in diabetics.[7] Hence, an attempt has been made to know the association between diabetes mellitus and oral manifestations and the impact of diabetes on oral health.

Periodontal Disease

Epidemiological and clinical evidence demonstrates that periodontal disease is more prevalent and more severe in both type 1 and type 2 diabetics as compared to non-diabetics.[8,9]

Periodontal disease has been referred to as the sixth complication of diabetes mellitus.[10] In addition study has reported an association between poor metabolic control and worst periodontal status in both type 1 and 2 diabetics,[11] but this association has not been supported by the results of several other studies. [12]

In type 2 diabetes, the risk for periodontal disease is estimated to be two to three-fold that of non-diabetics.[13] It has been reported that the risk for periodontal disease and its progression increases with the patient's age, duration of diabetes,[9] the presence and severity of diabetic complications and the degree of metabolic dysregulation[10,11,14] found significantly more periodontal disease in terms of probing depths (≥5 mm) or attachment loss in diabetics than in non-diabetic controls in the age of 40-49 years, and consequently concluded that age at time of diagnosis has an impact on the extent of periodontal disease. However, it has also been shown that the prevalence and extent of periodontal disease in diabetics is not associated with age. [8,13,15]

Children and adolescents with poorly controlled type 1 diabetes appear to have more advanced gingivitis as compared to non-diabetics, despite comparable plaque indices and level of oral hygiene.[14] The extent of gingival bleeding is most prominent in patients with newly diagnosed type 1 diabetes, and improvement of the metabolic control by intensified insulin treatment reduces the extent of gingival bleeding.[14] Furthermore, some studies have found that children and adolescents with type 1 diabetes have significantly deeper periodontal pockets and more extensive attachment loss than age-matched controls,[16] while others have not found any differences.[17] However, substantial evidence indicates that adult type 1 diabetics with poor metabolic control have more extensive attachment loss,[12] alveolar bone loss[18] and deeper periodontal pockets than well controlled type 1 diabetics.[19] Moreover, the severity of periodontal disease seems not only related to long-term poor metabolic control, but also to the presence of diabetic complications.[19]

Poor oral hygiene, poor metabolic control, current cigarette smoking and irregular dental care with long-term diabetic complications seem to have more risk for developing periodontal disease.[20] Health behavior which is closely related to psychological characteristics such as self-efficacy, is an important factor that should be considered, when the risk for periodontal disease is evaluated. Thus, poor adherence to both diabetes regimens and dental treatment is associated with poor metabolic control and oral diseases. [21]

The response to non-surgical periodontal treatment has been shown to be equal among diabetics and non diabetics, provided regular plaque control is ensured by frequent dental visit follow-ups.[22] Evidence suggests that elimination of periodontal infection improve tissue sensitivity to insulin and thereby the metabolic control. Indeed, study has shown that non-surgical periodontal treatment can have a positive effect on HbA1c levels in diabetics, reducing the need for insulin.[7] In a study the metabolic status improved most significantly in patients who received systemic antibiotic therapy with doxycycline in addition to conventional periodontal treatment. The reduced HbA1c levels were ascribed to the beneficial effect of antibiotics on the periodontal pathogens.[23]

The underlying pathogenic mechanisms behind the increased risk for developing periodontal disease in diabetes are still not clear. The advanced periodontal disease has been ascribed to a number of structural and functional hyperglycaemia-related alterations, such as thickening of the basement membranes of blood vessels (microangiopathy), which leads to deterioration of the microcirculation in the periodontal tissue and consequently to decreased supply of oxygen and nutrients to the tissues and accumulation of harmful metabolites; impaired functions of polymorphonuclear leukocytes leading to abnormalities of adherence, phagocytosis and chemotaxis; impaired gingival fibroblast proliferation and collagen synthesis; enhanced collagenase activity; and formation of AGEs, which bind to monocyte receptors, thereby

inducing production of inflammatory mediators such as tumour necrosis factor, prostaglandin E-2 and interleukin-1, and genetic predisposition for Gram-negative infections.[6,16] These mechanisms may lead to impaired host resistance to infection and accelerates inflammatory host response which results in loss of periodontal fibres, alveolar supporting bone and eventually loss of teeth.[24]

Early microbiological reports indicate that the oral microbial flora is changed due to increased levels of glucose in saliva and crevicular fluid. It has been shown that type 1 diabetics have more Capnocytophaga in their periodontal pockets, [25] and a higher proportion of gram-negative bacteria in their dental plaque than non-diabetics, but other studies have not supported these findings, since they found no significant changes in the microbial flora in type 1 diabetics.[26] It has also been reported that the duration of diabetes, type of diabetes and metabolic control of the disease have no significant influence on the prevalence of periodontal pathogens such as Actinobacillus actinomycetemcomitans, Fusobacterium nucleatum, Eiknella corrodens, Porphyromonas gingivalis and Prevotella intermedia.[27]

In summary, strong evidence suggest that diabetes is associated with an increased risk for developing periodontal disease, especially in diabetics with inadequate metabolic control. Conversely, periodontal treatment seems to improve the metabolic control of diabetes suggesting a bidirectional relationship between the two diseases. The increased susceptibility to periodontal disease may be explained by hyperglycaemia related alterations in the inflammatory host response.

Dental Caries

Results are conflicting as to whether diabetics have an increased risk of developing dental caries. Thus, some cross-sectional and controlled studies have found no difference in the prevalence of caries between diabetics and non-diabetics.[28] A higher caries experience and incidence has, however, also, been demonstrated among diabetics as compared to non-diabetics.[29] An association between metabolic control and dental caries has been reported in type 1 diabetics.[30] Increased caries activity and experience have been found in children and adolescents with type 1 diabetes of short duration and poor metabolic control, i.e., HbA1c values >10%, but once metabolic control has been stabilized, caries activity often decreases.[28] Elevated salivary glucose concentrations reduced salivary flow rates and low salivary pH is well-known risk factors for dental caries, salivary flow rates and salivary glucose levels have been found to be inversely correlated in type 1 diabetics. High salivary glucose concentrations have also been shown to correlate with high blood glucose concentrations. In addition, increased salivary glucose concentrations have been related to an increased number of lactobacilli and yeasts in the saliva.[31]

Literature provides a good evidence base to suggest that diabetics have a higher predilection for periodontal disease and gingival recession. The study showed that root caries scores were more among diabetics compared to non-diabetics. [32] For this reason, microorganisms which are less aciduric and acidogenic than lactobacilli and mutans streptococci can be assumed to play a role in the development of root surface caries. Some strains of A. Viscous and A. Naeslundii have shown to induce root caries in experimental animals and these species are consistently present in most plaque samples obtained from human root surfaces.[33]

A study has shown a positive correlation between the frequency of carbohydrate intake and root surface caries.[32] Other caries promoting factors such as mouth movements and sugar clearance may, however, be of great importance in connection with the food intake of elderly.

In conclusion, evidence suggests that an increased risk for dental caries in diabetes is related to poor metabolic control, salivary gland hypofunction, and high salivary glucose concentrations, which promotes growth of Streptococcus mutans and Lactobacillus.

Tooth Loss

A significant correlation between tooth loss and type 1 diabetes has been reported. Diabetics are about 5 times more likely to be partially edentulous than non diabetics.[34] Both advanced periodontal disease and dental caries may lead to the loss of teeth. An association between diabetic peripheral neuropathy and autonomic parasympathetic neuropathy and tooth loss has been found. A possible explanation for this association may be that autonomic neuropathy can reduce salivary flow rate, which can lead to tooth decay and hence tooth loss. The consequences of being partially or completely edentulous not only include changes in personal appearance, but also impairment of masticatory function and avoidance of certain foods, and ultimately poor diet quality and poor nutritional status. Thus, replacement of missing teeth, particularly the posterior tooth pairs, is important in order to avoid persistent oral health problems.[35] Placement of endosseous dental implants may be a preferable alternative to conventional dentures in partially or completely edentulous diabetics, who have susceptibility to oral candidiasis, traumatic ulcers and who suffer from hyposalivation. Placement of dental implants has been reported to be just as successful as in the general population, provided that the patients exhibit adequate metabolic control and adherence to oral hygiene regimens. On the other hand, the

failure rate appears to increase after about one year, which could indicate that implant failure is related to uncovering of the implants and to the early phase of implant loading. Diabetes-related microangiopathy leading to impaired immune response and reduced bone turnover has been suggested as an important risk factor to implant failure. [36]

Xerostomia, Salivary Gland hypofunction, and Sialosis

Xerostomia, the term for the subjective sensation of a dry mouth, is a common symptom in diabetics. The prevalence of xerostomia is 16% among type 1 diabetics with disease duration of 10 years, and 54% among type 2 diabetics with similar duration. [37] The marked variation in prevalence may be due to the fact that type 2 diabetics often are older, have more long-term diabetic complications and concomitant medical disorders, and take more medications that may cause xerostomia and hyposalivation than type 1 diabetics. Several studies of both type 1 and type 2 diabetics have shown that the sensation of oral dryness is related to a reduced flow rate of both unstimulated and stimulated whole saliva. [38] Poorly controlled (defined as HbA1c >9%) type 2 diabetes appear to have lower stimulated parotid flow rates than those who are well controlled. In children and adolescents with type 1 diabetes, high blood glucose values, but not high HbA1c values, are associated with low stimulated whole saliva flow rates and high salivary glucose concentrations. The glucose concentration in the saliva is assumed to increase at blood glucose values of 10-15 mmol/l.[39]

Salivary composition, including antimicrobial substances, total protein, electrolytes, salivary pH and buffer capacity has also been investigated, but the results are contradictory, regarding both whole saliva, and saliva collected separately from the parotid glands and the submandibular or sublingual glands.[40] The contradictory results may reflect differences in the selection of patients (age, duration of disease, metabolic control and medication) and methodology. Salivary flow rate, salivary pH, buffer capacity or microbial counts have not been found to correlate with the duration of diabetes. [38] A recent study found lower salivary pH, buffer capacity and salivary peroxidase activity in children with type 1 diabetes than in healthy controls. Total salivary protein concentrations have been found to be similar in diabetics as compared to non-diabetics. Increased concentrations of salivary potassium, salivary calcium and amylase have also been reported. [40]

The pathogenetic mechanisms behind diabetes-related changes in salivary gland function remain unclear. Dehydration, as the result of prolonged hyperglycaemia and consequently polyuria, is considered a major cause of xerostomia and salivary gland hypofunction

in diabetics. However, dehydration alone cannot explain the functional changes of the salivary glands. Lymphocytic infiltrates have been observed in labial salivary gland tissues of children with type 1 diabetes, indicating that the salivary gland tissue may be a target for the same autoimmune process as the pancreatic b-cells.[41] Gradual degeneration of salivary gland tissue can lead to salivary hypofunction and altered salivary composition. In addition, 10-25% of type 1 or type 2 diabetics may develop a bilateral asymptomatic enlargement of the parotid glands and, more rarely, of the submandibular glands, known as diabetic sialosis. [42] Histologically the salivary gland tissue from the enlarged parotid glands and the submandibular glands is characterized by fatty infiltration, fibrous tissue, enlargement of acinar cell and reduction in acinar tissue, but without signs of inflammation. [43]

The two frequently occurring degenerative complications of diabetes mellitus, autonomic neuropathy and microangiopathy, are thought to contribute to the development of structural alterations in the salivary gland tissue and thus the hypofunction of this gland by affecting the autonomic innervation and the microcirculation of the glandular tissue. However, patients with diabetic neuropathy have been reported to have both increased and decreased salivary flow rates.[44]

In summary, there is no consensus on the association between diabetes mellitus and salivary gland dysfunction. However, xerostomia and salivary gland hypofunction are commonly reported among diabetics, and may be indicative of poor metabolic control. In addition, hyposalivation and changes in salivary composition may contribute to the increased susceptibility to oral infections, impaired wound healing and increased rate of dental caries in diabetics.

Oral Candidiasis

Fungal infections are more common in type 1 and type 2 diabetics and appear to be more severe in diabetics than in non-diabetics.[45] Candida albicans is the most common species isolated from the oral cavity of diabetics, and of non-diabetics.

Substantial evidence suggests that the carriage frequency and the density of candidal colonization are increased in diabetics as compared to non-diabetics.[46] It remains controversial whether the diabetes itself increases the risk of candidal carriage and whether the increased candidal carriage actually reflects clinically manifest infection. Several studies have shown that poor metabolic control, high concentrations of glucose in blood and saliva, longer disease duration and the presence of diabetic complications (retinopathy) are associated with increased candidal carriage and clinical manifestations of candidiasis.[45] High

concentrations of glucose in the blood and saliva may promote growth and enhance adherence of yeasts to epithelial cell surfaces. Also the impaired functions of polymorphonuclear leukocytes leading to reduced phagocytosis, intracellular killing and chemotaxis may contribute to the increased colonization of Candida and increased susceptibility to oral candidiasis. However, a substantial number of local and systemic factors that are not related to diabetes may also influence candidal carriage status and the susceptibility to oral candidiasis. These factors include gender, oral hygiene habits, smoking habits, intake of medications, denture wearing, salivary flow rate, and salivary composition.[46] Inadequately controlled diabetics who wear dentures have a higher oral candidal load and higher prevalence of denture stomatitis than non-diabetic denture wearers.[45] Furthermore, low salivary flow rates and low salivary pH have been found associated with high incidence of Candida. [47]

Oral infection with Candida may clinically present as median rhomboid glossitis, atrophic glossitis, denture stomatitis, pseudomembranous candidiasis and angular cheilitis. Oral candidiasis may be accompanied by burning mouth sensations, taste disturbances (usually metallic taste) and sensation of dry mouth. The presence of median rhomboid glossitis and denture stomatitis has been found to be related to long disease duration, long-term diabetic complications (nephropathy and retinopathy), poor metabolic control and smoking. [45]

In conclusion, diabetics have a high rate of oral candidal carriage and an increased risk for oral candidiasis, which is related to poor metabolic control, high concentrations of glucose in the blood and saliva, reduced salivary flow rates, low saliva pH and a reduction in antimicrobial substances in the saliva. However, other risk factors such as oral hygiene, smoking and dentures also have a substantial influence on oral candidal status in diabetics and their susceptibility to oral candidiasis.

Other Oral Mucosal Lesions

An increased occurrence of oral lichen planus has been observed in both type 1 and type 2 diabetics as compared to healthy controls.[48] However study has reported a low prevalence of oral lichen planus in diabetics and not being significantly different to that of healthy non-diabetic controls. The occurrence of diabetes mellitus in patients with oral lichen planus has also been examined and varies from 1.6- 37%.[49] The contradictory results may reflect differences in the criteria used for diagnosing both diabetes mellitus and oral lichen planus. An increased occurrence of oral leukoplakia has also been observed in both type 1 and type 2 diabetics as compared to healthy controls. Oral leukoplakia, as well as oral lichen planus lesions, was most prevalent in insulin-treated diabetics, who were smokers and approximately 2 years disease duration.[48]

An increased prevalence of fissured tongue has been observed in diabetics as compared to non-diabetics and especially in older type 1 diabetics with longer disease duration and complaints of dry mouth. [50]

In general, type 1 diabetics have an increased risk for oral mucosal lesions, in particular fissured tongue, irritation fibromas and traumatic ulcers as compared to healthy controls and the risk is related to age, duration of diabetes and the presence of diabetic complications.

Taste Impairment

Several reports indicate that the ability to detect and recognize sweet, salty and bitter taste is impaired in both type 1 and type 2 diabetics. Electrogustometric studies have revealed that hypogeusia and ageusia are significantly more prevalent in type 1 and type 2 diabetics than in non-diabetics. Accordingly, 33-73% of type 1 diabetics and 40% of type 2 diabetics fulfil the criteria for hypogeusia. Ageusia has been found in 3% of type 1 diabetics and 5% of type 2 diabetics. It has been suggested that the impaired taste acuity, especially for sweet, can lead to hyperphagia and increased intake of sugar, and hence obesity and impairment of the metabolic status.[51] In newly diagnosed type 2 diabetics, taste impairment of glucose and salt, partially reversed after correction of hyperglycaemia with diet and oral hypoglycaemic agents suggesting an association between blood glucose concentrations and taste acuity. Taste impairment has also been found associated with long disease duration and long-term diabetic complications, particularly peripheral neuropathy. However, taste impairment has also been observed in diabetic patients without peripheral and autonomic neuropathy. Several other factors such as salivary gland hypofunction, intake of medications and smoking may be responsible for impaired taste acuity and should be considered when evaluating the relationship between taste impairment and diabetes. [52]

Burning Mouth Sensation

Many diabetics complain of burning sensations in the oral cavity. The burning mouth sensations have been found related to increased candidal density and Candida-associated stomatitis. It has therefore recently been suggested that the sensation of burning mouth in diabetics occurs via stimulation of the capsaicin receptor by Candida metabolites, since capsaicin receptors are responsible for the detection of pain-producing chemical and thermal stimuli. An association between poor metabolic control and burning mouth sensations has also been suggested. The report further indicates that burning

mouth sensation is an early sign of undiagnosed diabetes.[53] On the other hand, burning mouth sensations have been associated with long disease duration. Thus, glossodynia has been reported in 18% of type 2 diabetics with disease duration of 13 years. It has been suggested that glossodynia is an oral manifestation of peripheral neuropathy, a common long-term diabetic complication. However, several local and systemic conditions may be accompanied by burning mouth sensations, including such as hyposalivation and oral habits like tongue thrusting and haematinic deficiencies. Persistent burning mouth sensations are also the cardinal symptom of burning mouth syndrome, a condition of remaining unknown etiology, although numerous potential etiological factors have been proposed including diabetes mellitus.[54]

Impaired Wound Healing

Impaired wound healing is a prevalent complication of diabetes mellitus. The underlying pathophysiological mechanisms of impaired wound healing in diabetics are, however, poorly understood. Wound healing is a multi-step process that includes an inflammatory response, granulation tissue formation, wound closure and angiogenesis, and tissue remodeling.[55] An adequate blood and nerve supply is required for efficient wound healing. In diabetics, several structural and functional hyperglycaemia-related abnormalities are thought to contribute to impaired wound healing. [56] Interaction of AGEs with the receptor for AGEs (RAGE) results in an exaggerated inflammatory response and compromised collagen production, which can lead to impaired wound healing.[57] Recent clinical studies indicate that the use of growth factors offer promising future therapeutic possibilities for enhancing wound healing. [58]

Dentists being health care providers are encountering an increasing number of patients with known or undiagnosed diabetes. Dentists can play an important role in identifying children and adults who are at risk of developing diabetes mellitus. Regular dental visits provide an obvious opportunity for prevention of the disease among healthy individuals and for screening children and adults with early signs and symptoms suggestive of diabetes. Early intervention in patients at particular risk, e.g., overweight children, may include dietary counselling to reduce sugar intake and thus contribute in reducing the risk of developing both obesity and dental caries. Diabetics with poor metabolic control are at higher risk for oral diseases, and may develop complications following dental procedures. Preventive measures that focus on decreasing the risk, incidence, and consequences of diabetes will help in reducing human sufferings from diabetes mellitus.

REFERENCES

1. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med 1998;15:539-553.
2. WHO chronic disease report. International Diabetic Federation press release Brussels 5th October 2005. www.idf.org.
3. World Health Organisation. Global burden of diabetes (www document). URL http:// www.who. Int/inf-pr-1998/en/pr98-63.html (last accessed 30 april,2006).
4. Bjork S, Kapur A, Sylvett C, Kumar D, Nail J, Kelkar S. Presentation made at forum 9 Mumbai 12 to 16th September 2005. www.global forum health.org.
5. Saadoun, A.P. Diabetes and periodontal disease: a Review and Update, Periodont Abstr. 1980; 28:116-139.
6. Oliver RC, Tervonen T. Diabetes - A risk factor for periodontitis in adults? J Periodontol. 1994;65:530-538.
7. Miller LS, Manswell MA, Newbold D, Reding ME, Rasheed A, Blodgett J, et al. The relationship between reduction in periodontal inflammation and diabetes control: a report of 9 cases. J Periodontol. 1992;63:843-848.
8. Schlossman M, Knowler WC, Pettitt JJ, Genco RJ. Type 2 diabetes mellitus and periodontal disease. J Am Dent Assoc. 1990;121: 532-536.
9. Thorstensson H, Hugoson A. Periodontal disease experience in adult long-duration insulin-dependent diabetics. J Clin Periodontol 1993;20:352-358.
10. Löe H. Periodontal disease. The sixth complication of diabetes mellitus. Diabetes Care 1993;16:329-334.
11. Tsai C, Hayes C, Taylor GW. Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population. Community Dent Oral Epidemiol. 2002;30:182-192.
12. Bridges RB, Anderson JW, Saxe SR, Gegory K, Bridges SR. Periodontal status of diabetic and non-diabetic men: effects of smoking, glycemic control and socioeconomic factors. J Periodontol. 1996;67:1185-1192.
13. Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in noninsulin- dependent diabetes mellitus. J Periodontol. 1991;62: 123-131.
14. Karjalainen KM, Knuuttila MLE. The onset of diabetes and poor metabolic control increases gingival bleeding in children and adolescents with insulin-dependent diabetes mellitus. J Clin Periodontol. 1996;23:1060-1067.
15. Dahms WT. An update in diabetes mellitus. Pediatr Dent. 1991;3- 4:79-82.
16. Leeper SH, Kalkwarf KL, Strom EA. Oral status of ‘‘controlled’’ adolescent Type 1 diabetics. J Oral Med. 1985;40:127-133.
17. Sbordone L, Ramaglia L, Barone R, Ciaglia RN, Iacono VJ. Periodontal status and subgingival microbiota of insulin-dependent juvenile diabetics: A 3-year longitudinal study. J Periodontol. 1998;69:120-128.
18. Seppala B, Seppala M, Ainamo J. A longitudinal study on insulindependent diabetes mellitus and periodontal disease. J Clin Periodontol. 1993;20:161-165.
19. Karjalainen KM, Knuuttila MLE, von Dickhoff KJ. Association of the severity of periodontal disease with organ complications in type 1 diabetic patients. J Periodontol. 1994;65:1067-1072.
20. Syrjala AM, Ylostalo P, Niskanen MC, Knuuttila ML. Role of smoking and HbA1c level in periodontitis among insulin-dependent diabetic patients. J Clin Periodontol. 2003;30:871-875.
21. Syrjala AM, Ylostalo P, Niskanen MC, Knuuttila ML. Relation of different measures of psychological

characteristics to oral health habits, diabetes adherence and related clinical variables among diabetic patients. Eur J Oral Sci. 2004;112:109-114.
22. Westfelt E, Rylander H, Blohme G, Jonasson P, Lindhe J. The effect of periodontal therapy in diabetics, result after 5 years. J Clin Periodontol. 1996;23:92-100.
23. Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: A two-way relationship. Ann Periodontol. 1998;3:51-61.
24. Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M. Glycemic control and alveolar bone loss progression in type 2 diabetes. Ann Periodontol. 1998;3:30-39.
25. Zambon JJ, Reynolds H, Fisher JG, Shlossman M, Dunford R, Genco RJ. Microbiological and immunological studies of adult periodontitis in patients with noninsulin-dependent diabetes mellitus. J Periodontol. 1988;59:23-31.
26. Sastrowijoto SH, Hillemans P, van Steenbergen TJ, Abraham-Inpijn L, de Graaff J. Periodontal condition and microbiology of healthy and diseased periodontal pockets in type 1 diabetes mellitus patients. J Clin Periodontol. 1989;16:316-322.
27. Tervonen T, Karjalainen K, Knuuttila M, Huumonen S. Alveolar bone loss in type 1 diabetic subjects. J Clin Periodontol 2000; 27:567-571.
28. Moore PA, Weyant RJ, Etzel KR, Guggenheimer J, Mongelluzzo MB, Myers DE, et al. Type 1 diabetes mellitus and oral health: assessment of coronal and root caries. Community Dent Oral Epidemiol. 2001a; 29:183-194.
29. Jones RB, McCallum RM, Kay EJ, Kirkin V, McDonald P. Oral health and oral health behaviour in a population of diabetic outpatient clinic attenders. Community Dent Oral Epidemiol. 1992; 20:204-207.
30. Twetman S, Nederfors T, Stahl B, Aronson S. Two-year longitudinal observations of salivary status and dental caries in children with insulin-dependent diabetes mellitus. Pediatr Dent. 1992; 14:184-188.
31. Karjalainen KM, Knuuttila ML, Kaar ML. Salivary factors in children and adolescents with insulin-dependent diabetes mellitus. Pediatr Dent. 1996;18:306-311.
32. Hintao J, Teanpaisan R, Chongsuvivatwong V, Dahlen G, attarasarn C. Root surface and coronal caries in adults with type 2 diabetes mellitus. Community Dent Oral Epidemiol. 2007;35(4):302-9.
33. Fure S, Romaniec M, Emilson CG, Krase B. Proportions of Streptococcus mutans, lactobacilli and Actinomyces spp in root surface plaque. Scand J Dent Res. 1987;95:119-23.
34. Syed SA, Loesche WJ, Pape HL Jr, Grenier E. Predominant cultivable flora isolated from human root surface caries plaque. Infect Immun. 1975;11:727-31.
35. Newrick PG, Bowman C, Green D, O'Brien IA, Porter SR, Scully C, et al. Parotid salivary secretion in diabetic autonomic neuropathy. J Diabet Complications. 1991;5:35-37.
36. Olson JW, Shernoff AF, Tarlow JL, Colwell JA, Scheetz JP, Bingham SF. Dental endosseous implant assessments in a type 2 diabetic population: a prospective study. Int J Oral Maxillofac Implants. 2000;15:811-818.
37. Moore PA, Guggenheimer J, Etzel KR, Weyant RJ, Orchard T. Type 1 diabetes mellitus, xerostomia and salivary flow rates. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001b;92:281- 291.
38. Meurman JH, Collin H-L, Niskanen L., Töyry J, Alakuijala P, Keinänen S, et al. Saliva in non-insulin-dependent diabetic patients and control subjects. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1998;86:69-76.
39. Reuterving CO, Reuterving G, Hagg E, Ericson T. Salivary flow rate and salivary glucose concentration in patients with diabetes mellitus influence of severity of diabetes. Diabetes Metab. 1987;13:457-462.
40. Dodds MW, Dodds AP. Effects of glycemic control on saliva flow rates and protein composition in non-insulin-dependent diabetes mellitus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997;83:465-470.
41. Markopoulos AK, Belazi M. Histopathological and Immunohistochemical Features of the Labial Salivary Glands in Children With Type I Diabetes. J Diabet Complications. 1998;12:39-42.
42. Russotto SB. Asymptomatic parotid gland enlargement in diabetes mellitus. Oral Surg. 1981;52:594-598.
43. Lindeberg A, Andersen L. Size and composition of the submandibular glands in late onset diabetes. Arch Otorhinolaryngol. 1987;244:100-103.
44. Lamey PJ, Fisher BM, Frier BM. The effects of diabetes and autonomic neuropathy on parotid salivary flow in man. Diabet Med. 1986;3:537-540.
45. Guggenheimer J, Moore PA, Rossie K, Myers D, Mongelluzzo MB, Block HM, et al. Insulin-dependent diabetes mellitus and oral soft tissue pathologies. II. Prevalence and characteristics of Candida and candidal lesions. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000b;89:570-576.
46. Budtz-Jorgensen E. Candida-associated denture stomatitis and angular cheilitis. In: Samaranayake LP, Macfarlane TW (eds). Oral candidosis. London: Wright. 1990:156-183.
47. Banoczy J, Albrecht M, Rigo O, Ember G, Ritlop B. Salivary secretion rate, pH, lactobacilli and yeast counts in diabetic women. Acta Diabetol Lat. 1987;24:223-228.
48. Albrecht M, Banoczy J, Dinya E, Tamas G Jr. Occurrence of oral leukoplakia and lichen planus in diabetes mellitus. J Oral Pathol Med. 1992;21:364-366.
49. Grinspan D, Diaz J, Villapol LO, Schneiderman J, Berdichesky R, Palese D, et al. Lichen ruber planus of the buccal mucosa. Its association with diabetes. Bull Soc Fr Dermatol Syphiligr. 1966; 73:898-899.
50. Moore PA, Weyant RJ, Mongelluzzo MB, Myers DE, Rossie K, Guggenheimer J, et al. Type 1 diabetes mellitus and oral health: assessment of tooth loss and edentulism. J Public Health Dent. 1998;58:135-142.
51. Stolbova K, Hahn A, Benes B, Andel M, Treslova L. Gustometry of diabetes mellitus patients and obese patients. Int Tinnitus J 1999;5:135-140.
52. Perros P, MacFarlane TW, Counsell C, Frier BM. Altered taste sensation in newly-diagnosed NIDDM. Diabetes Care. 1996;19: 768-770.
53. Vitkov L, Weitgasser R, Hannig M, Fuchs K, Krautgartner WD. Candida-induced stomatopyrosis and its relation to diabetes mellitus. J Oral Pathol Med. 2003;32:46-50.
54. Pedersen AML, Smidt D, Nauntofte B, Christiani CJ, Jerlang BB. Burning mouth syndrome: Aetiopathogenetic mechanisms, symptomatology, diagnosis and therapeutic approaches. Oral Biosci Med. 2004;1:3-19.
55. Hunt TK, Hopf H, Hussain Z. Physiology of wound healing. Adv Skin Wound Care. 2000;13:6-11.
56. Silhl N. Diabetes and wound healing. J Wound Care. 1998;7:47- 51.
57. Wear-Maggitti K, Lee J, Conejero A, Schmidt AM, Grant R, Breitbart A. Use of topical RAGE in diabetic wounds increases neovascularization and granulation tissue formation. Ann Plast Surg. 2004;52:519-521.
58. Bennett SP, Griffiths GD, Schor AM, Leese GP, Schor SL. Growth factors in the treatment of diabetic foot ulcers. Br J Surg. 2003; 90:133-146.

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