

ORAL CAVITY AND SYSTEMIC DISEASES – *DIABETES MELLITUS*

Assya Krasteva¹, Vladimir Panov², Adriana Krasteva³, Angelina Kisselova¹, Zachary Krastev⁴

¹Medical University Sofia, Faculty of Dental Medicine, Department of Imaging and Oral Diagnostic, Sofia, Bulgaria

²Medical University “Prof. Dr. P. Stoyanov”, Faculty of Dental Medicine, Department of Conservative and Pediatric Dentistry, Varna, Bulgaria

³Medical University Sofia, Faculty of Pharmacy, Department of Pharmacology and Toxicology, Sofia, Bulgaria

⁴Medical University Sofia, Clinic of Gastroenterology “St. Ivan Rilski”, Sofia, Bulgaria

Correspondence to: Assya Z. Krasteva

E-mail: asyakrasteva@gmail.com

ABSTRACT

Diabetes mellitus (DM) is a metabolic disease which affects many organs. Often in the oral cavity undergo changes that are associated with diabetes.

Many dental practitioners are often not aware of the attendant oral manifestations in diabetic patients, which results in prescription of a non-accurate treatment.

The aim of the review is to focus on main oral manifestations related to diabetes in order that the proper solution for treatment is to be taken.

Untreated oral infections can adversely affect metabolic control. Patients may present with oral conditions that suggest undiagnosed diabetes: progressing severe periodontitis, enlarged gums which bleed easily, multiple periodontal abscesses. Diabetic patients with poor oral hygiene, a history of smoking, rare visits to dentists, high carbohydrate intake are more likely to present caries and periodontitis and to respond poorly to dental treatment. If the doctor suspects undiagnosed diabetes, the patient should be examined to reveal the history of polydipsia, polyuria, polyphagia, unexplained weight loss, and family history of diabetes.

Biotechnol. & Biotechnol. Eq. 2011, **25**(1), 2183-2186

Keywords: diabetes mellitus, oral health, dental practitioners

Oral manifestations in patients with diabetes mellitus

Diabetes is a metabolic disease which affects many organs, and also the structure of the mucosa tissue in the oral cavity. A number of oral manifestations have been associated with DM, particularly in patients with poor disease control.

Oral signs that are seen in individuals with diabetes are:

- burning mouth and taste disturbances;
- xerostomia;
- decreased salivary secretion;
- multiple carious lesions and caries in unusual places (root caries);
- delayed wound healing;
- increased incidence of infection;
- enlarged gingival tissues bleeding easily upon manipulation;
- periodontal disease (occurs more frequently and progress rapidly than in normal patients);
- multiple periodontal abscesses;
- oral candidiasis (most commonly erythematous, together with atrophy of lingual papillae);
- ulcers and irritation fibromas;

- lichen planus and lichenoid reactions;
- erythema migrans;
- diabetic sialadenosis (diffuse bilateral enlargement). Enlargement of the parotid glands;
- altered oral microflora, necrotizing external otitis caused by *Pseudomonas aeruginosa*, abnormal tooth eruption (early or late);
- faster alveolar bone resorption.

Xerostomia may result more often from use of medications than from bad metabolic control. Dry mucous membranes are easily irritated and are associated with “burning mouth” syndrome. Moreover, they provide a favourable environment for fungal organisms.

There is an increased predisposition to fungal infections which are manifested as oral candidiasis, including median rhomboid glossitis, denture stomatitis and angular cheilitis. This predisposition may be due not only to xerostomia, but also to increased salivary glucose levels or immune dysregulation. Mucormycosis is a rare but serious systemic fungal infection that may occur in patients with uncontrolled DM. Oral involvement usually appears as palatal ulceration or necrosis (17).

Hyperglycaemia state has shown a positive association with dental caries. Recent studies show higher frequency of caries in diabetic individuals which is associated with dry mouth or increased amount of glucose in gingival crevicular fluid.

The burning may be due to peripheral neuropathy, xerostomia or candidiasis.

Good glycemic control may alleviate the burning sensation—some diabetic patients have a mild impairment of the sweet taste sensation. This may be related to xerostomia or disordered glucose receptors (1, 9, 15, 16).

Poorly controlled diabetics more often have also gingivitis and periodontitis compared to well-controlled diabetic or non-diabetic patients (1, 9, 15).

Increased amount of glucose in crevicular fluid may also alter wound-healing events in periodontal tissue. Diabetes changes the functions of polymorphonuclear leukocytes (PMNs), monocytes and macrophages. Monocytes and macrophages produce significantly more pro-inflammatory cytokines and mediators (11, 12, 19, 20).

Risk factors for periodontal disease are poor oral hygiene and smoking. Accumulated deposits of glycosylated proteins cause collagen storage and thickening of the membranes. These changes decrease tissue perfusion and oxygenation, change the response to periodontal pathogens, leading to destruction of tissues and a decrease in the recovery potential. Diabetes not only increases the prevalence and severity of periodontitis, but the progression of bone loss and loss of attachment over time (7, 21).

Periodontitis is also associated with an increased risk of other complications of diabetes such as nephropathy and macrovascular disease. Periodontal infection, itself may adversely affect glycemic control as increased risk of poor glycemic control up to six times. In diabetic patients with periodontitis, periodontal therapy with a combination of mechanical debridement (scaling and planing root) and systemic doxycycline antibiotic therapy may have beneficial effect on glycemic control (5, 6, 13).

The prevalence of oral lichen planus is significantly higher in diabetic patients than in control subjects. However, this may be a side effect of oral hypoglycemic agents or antihypertensive medications. Grinspan Syndrome is triad of lichen planus, DM and vascular hypertension.

People with type 1 DM have a higher prevalence of oral traumatic ulcers and irritation fibromas. These findings may be related to altered wound healing patterns in these patients. Delayed healing of wound due to microangiopathy and utilisation of protein for energy, may retard the repair of tissues (16, 17).

General dental treatment rules

Metabolic control

The dental practitioner should be familiar with glycemic control before starting a treatment. Patients should carry their glucometer to the dental office. Patients with poor glycemic control do not respond well enough to non-surgical treatment and periodontal surgery. The short term improvement of periodontal health is often followed by relapse. Dental doctors should be aware of values of glycated hemoglobin, as it characterizes the glycemic control in the last 2-3 months. HbA1c values that are less than 8% indicate relatively good glycemic control and values greater than 10% indicate poor control (8, 10).

Pain control and stress reduction

It is necessary to ensure adequate control of pain and stress reduction, for example, by anesthesia, which in addition reduces pain and decreases endogenous release of epinephrine. Withdrawal of consciousness is used in extremely anxious individuals. The quantity of epinephrine in local anaesthetics is very small and does not affect blood glucose (2, 3, 14).

Antibiotics

It is recommended that antibiotic prophylaxis precedes invasive dental treatment. If glycemia is poorly controlled: a single preoperative oral dose of 2 g amoxicillin or 600 mg clindamycin if the patient is allergic to penicillins, eventually followed by a 6-hour postoperative dose. Besides antibacterial effects tetracycline reduces host collagenase production and degradation of collagen, which is important in the treatment of periodontitis (4, 18, 22, 23).

Insulin

It is advisable to reduce the dose of insulin that precedes long or major dental procedures. Diabetic patients should receive dental treatment in the morning. Often, it is not possible to plan dental treatment so as to avoid peak insulin activity. It is best to plan dental treatment either before or after periods of peak insulin activity (8).

The greatest risk of hypoglycaemia will occur in 30 to 90 minutes after injecting of Rapid-acting insulin, 2 to 3 hours after Short-acting insulin, and 4 to 10 hours after Intermediate insulin (Table 1).

TABLE 1

Types of Insulin

Types of Insulin	Names of Insulin	How Fast They Start	When the Action Peaks	How Long They Last
Rapid Acting	Humalog/Lispro Novolog/Aspart	5-15 minutes	30-90 minutes 1-3 hours	3-5 hours
Short Acting	Regular	1/2-1 hour	2-4 hours	6-8 hours
Intermediate	NPH	1-2 hours	6-10 hours	10-16 hours
Long Acting	Lantus/Glargine Levemir/Detemir	1-2 hours	No peak action	24-36 hours

Risk of hypoglycaemia

Metformin and the thiazolidinediones rarely cause hypoglycaemia. The greatest risk would occur in a patient who fails to eat or eats less than the usual amount. It is reasonable to check the blood glucose level (using patient's glucometer) and to have a source of carbohydrates readily available.

Factors which increase the risk of hypoglycaemia:

- missed or delayed food intake;
- injection of high dose of insulin;
- increased exercise level;
- alcohol consumption;
- previous history of hypoglycaemia.

Questions to assess the risk of hypoglycaemia:

1. Have you ever had a severe hypoglycaemia before?
2. What diabetic medication do you take?
 - 1.1 Did you take it today?
 - 1.2 Is this the same amount as you normally take?
3. What did you eat today before coming to the dental office?
 - 1.1 At what time did you eat?
 - 1.2 Do you normally eat at this time?
 - 1.3 Do you often skip a meal?

Diabetic emergencies in the dental office

The most common diabetic emergency in the dental office is hypoglycaemia: confusion, sweating, tremors, agitation, anxiety, dizziness, and tachycardia. Severe hypoglycaemia may result in seizures or loss of consciousness. If a glucometer is unavailable, the dental practitioner should give the patient approximately 15 g of oral carbohydrate in a form that will be absorbed rapidly (**Table 2**).

TABLE 2

Treatment of hypoglycaemia

Number	Action
1.	If the patient is able to take food by mouth, give <u>1 tablespoon sugar or sugar-containing beverages</u>
2.	If the patient is unable to take food, 40% glucose intravenously as a bolus dose of 40-50 ml should be given; if the need for hospitalization is assessed, a 10-20% glucose infusion is needed to maintain blood glucose between 6 and 10 mmol/L
3.	One mg glucagon subcutaneously or intramuscularly can be applied
4.	Patients with hypoglycaemia and sulfonylurea antidiabetics should be hospitalized because of continuous glucose infusion need

Signs of hypoglycaemia should resolve in 10 to 15 minutes and the patient should be observed for 30 to 60 minutes after recovery. Marked hyperglycaemia may present with symptoms mimicking hypoglycaemia. These symptoms must be treated as hypoglycaemia because the small amount of extra glucose will not have a significant negative effect. Diabetic ketoacidosis and hyperosmolar acidosis require immediate hospital treatment (8, 10).

Practical recommendations

Two clinical diabetic cases are presented. To start with, a medical consultation is needed to control the overall disease.

1. Massive candida infection (cheilitis angularis, **Fig. 1**; and oral candidiasis, **Fig. 2**) is microbiologically demonstrated by saliva and throat secretion examination.



Fig. 1. Cheilitis angularis



Fig. 2. Oral candidiasis

Administration of Mycomax tabl. 150 mg was suggested once weekly, for a period of 3 weeks. The oral cavity is treated with Mycozanole 2% peroral gel twice daily, for at least 15 days, and daily salt mouthwash. After discontinuation of treatment a new microbiological examination is needed.

2. The second clinical case was with gingivitis (**Fig. 3**)



Fig. 3. Gingivitis

General recommendations

Patients with gingivitis (such as patients with periodontitis) need at least 3 times per year professional cleaning of dental plaque and tartar. After each procedure laser periodontal therapy is recommended and should be applied for at least 4 weeks. In individual assessment Metronidazole (250 mg tabl.) is prescribed (twice per year) for 7 or 10 days and daily use of mouth wash.

The patients are recommended to use two different toothpastes- one in the morning and one in the evening (priority of those with dental preventive effects on periodontal and children's toothpastes).

In serious cases and these who do not respond to the treatment, pre-examination of periodontal flora is needed and after results are taken into account an accurate antibiotic treatment should be prescribed.

In diabetic patients with lichen planus or when fibroma laser removal of lesions is recommended, the smaller risk of bleeding, bactericidal effect of laser stimulation and biological tissue effects should be considered.

In any case, the focus should be on the clinical findings and the approach is always individual, conform to the general condition of patients.

Conclusions

The intimate relationship between oral health and systemic health in individuals with diabetes suggests a need for increased interaction between the dental and medical professionals who are charged with the management of these patients. Vascular changes seen in the retina, glomerulus, and perineural areas also occur in the periodontium. Oral health assessment and treatment should become as common as the eye, foot, and kidney evaluations that are routinely performed as part of preventive medical therapies

REFERENCES

3. Ficara A., Levin M., Grower M., Kramer G. (1975) *J. Periodontol. Res.*, **10**, 171-175.
4. Ghezzi E. and Ship J. (2000) *J. Public Health Dent.*, **60**, 289-296.
5. Ghezzi E., Chavez E., Ship J. (2000) *Spec. Care Dentist.*, **20**, 81-92.
6. Golub L., Lee H.M., Ryan M. (1998) *Adv. Dent. Res.*, **12**, 12-26.
7. Grossi S., Skrepcinski F., DeCaro T. et al. (1996) *J. Periodontol.*, **67**, 1094-1112.
8. Grossi S., Skrepcinski F., DeCaro T. et al. (1997) *J. Periodontol.*, **68**, 713-719.
9. Holm-Pedersen P., Agerbaek N., Theilade E. (1975) *J. Clin. Periodontol.*, **2**, 14-24.
10. Ketterl W. (1983) *Int. Dent. J.*, **33**, 262-271.
11. Kisselova A., Zekova M., Krashev Z. (2009) In: *Oral medicine (I. Sapunjev EOOD, Ed.)*, Sofia, 362-368.
12. Little J. et al. (2002) *Masby Inc., Elsev. Sc.*, 6th ed.
13. Manoucher-Pour M., Spagnuolo P., Rodman H., Bissada N. (1981) *J. Periodontol.*, **52**, 410-415.
14. McMullen J., Van Dyke T., Horoszewicz H., Genco R. (1981) *J. Periodontol.*, **52**, 167-173.
15. Miller L., Manwell M., Newbold D. et al. (1992) *J. Periodontol.*, **63**, 843-848.
16. Niessen L., Jones J., Zocchi M., Gurian B. (1985) *J. Am. Dent. Assoc.*, **110**, 207-209.
17. Nishimura F., Takahashi K., Kurihara M. et al. (1998) *Ann. Periodontol.*, **3**, 20-29.
18. Panov V., Krasteva A., Kisselova A. (2011) In: *Oral lesions (I. Sapunjev EOOD, Ed.)*, Sofia, 179-301.
19. Ponte E., Tabaj D., Maglione M., Melato M. (2001) *Acta Diabetologica*, **38**, 57-62.
20. Rao D., Desai A., Kulkarni R., Gopalkrishnan K., Rao C. (2010) *Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod.*, **110**, 7-12.
21. Salvi G., Collins J., Yalda B. et al. (1997) *J. Clin. Periodontol.*, **24**, 8-16.
22. Salvi G., Yalda B., Collins J. et al. (1997) *J. Periodontol.*, **68**, 127-135.
23. Taylor G., Burt B., Becker M. et al. (1996) *J. Periodontol.*, **67**, 1085-1093.
24. Tong D. and Theis J. (2008) *N. Z. Med. J.*, **121**, 45-52.
25. van Winkelhoff A. (2010) *Ned. Tijdschr. Tandheelkd.*, **117**, 519-523.