**REVIEW ARTICLE**

# ORAL MANIFESTATIONS OF DIABETES MELLITUS: AN UPDATED CLINICAL AND PATHOPHYSIOLOGICAL REVIEW

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## ABSTRACT

Diabetes mellitus is a chronic metabolic disorder with important effects on oral health. This review summarizes the major oral manifestations associated with diabetes, including periodontal disease, xerostomia, dental caries, oral candidiasis, oral mucosal alterations and delayed wound healing. These complications arise from interacting mechanisms such as persistent hyperglycaemia, oxidative stress, advanced glycation end-product formation, vascular dysfunction, impaired immune responses, altered salivary function and defective tissue repair. Particular attention is given to the bidirectional relationship between diabetes and periodontal disease, wherein diabetes may increase susceptibility to periodontal inflammation, while persistent periodontal inflammation may adversely influence systemic inflammatory burden and glycaemic control. Xerostomia and salivary dysfunction may further increase susceptibility to dental caries and oral infections, while impaired immune responses may facilitate Candida colonization. Diabetes-associated vascular and cellular abnormalities may also delay healing after oral surgical procedures. Recognition of these manifestations is important for both dental and medical professionals. Preventive oral care, periodontal management, appropriate treatment of infections, regular dental assessment and adequate glycaemic control should be incorporated into comprehensive diabetes care. A multidisciplinary approach involving physicians and dental professionals can facilitate early recognition and management of oral complications and may improve the quality of life and health outcomes of patients with diabetes.

**Keywords:** Diabetes mellitus, Oral manifestations, Periodontitis, Xerostomia, Dental caries, Oral candidiasis, Wound healing, Glycaemic control.

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## INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from impaired insulin secretion, impaired insulin action or a combination of both. It is associated with a wide range of metabolic, vascular and immunological abnormalities that can affect multiple organ systems. Because the oral cavity is highly vascularized and innervated, the systemic metabolic disturbances associated with diabetes may have important consequences for oral tissues and oral health.[1]

The relationship between diabetes mellitus and oral health is complex and multifactorial. Individuals with diabetes may develop a broad spectrum of oral manifestations

involving the periodontal tissues, teeth, oral mucosa, salivary glands and supporting structures. Periodontal disease, xerostomia, salivary dysfunction, dental caries, oral candidiasis, mucosal alterations, taste disturbances and impaired wound healing have all been described in association with diabetes. The occurrence and severity of these manifestations may be influenced by the type and duration of diabetes, the degree of glycaemic control, oral hygiene, systemic complications, medications and other patient-related factors.[1,2]

Among the various oral complications, periodontal disease has received particular attention because of its close and potentially bidirectional relationship with diabetes. Chronic hyperglycaemia may increase susceptibility to periodontal tissue destruction through alterations in host immune responses, vascular function, connective-tissue metabolism and inflammatory pathways. Conversely, persistent periodontal inflammation may contribute to systemic inflammatory burden and adversely influence glycaemic control. Periodontal health therefore represents an important component of comprehensive diabetes management.[2,3]

The biological basis of the oral complications of diabetes involves several interconnected mechanisms. Persistent hyperglycaemia can promote metabolic and vascular alterations, impaired immune function, changes in tissue metabolism and abnormalities in wound repair. Increased formation of advanced glycation end products and associated alterations in inflammatory and vascular pathways may contribute to tissue damage and impaired periodontal homeostasis. Changes in salivary secretion and composition may additionally reduce the protective functions of saliva and influence the oral microbial environment, thereby increasing susceptibility to dental and infectious complications.[2]

Diabetes may also affect the oral mucosa, and a variety of mucosal abnormalities have been reported in affected individuals, including ulcerative lesions, pigmentation, candidiasis, angular cheilitis, recurrent aphthous ulceration and leukoplakia.[4,5] The clinical presentation may vary considerably between individuals, highlighting the importance of recognizing oral abnormalities within the broader context of the patient's metabolic status.

The oral cavity may therefore provide clinically relevant information on the systemic health of individuals with diabetes. Recognition of characteristic oral findings may facilitate appropriate dental management and, in selected patients, may prompt assessment of glycaemic status and other systemic risk factors. Conversely, effective management of oral disease, particularly periodontal inflammation, may contribute to improved overall health and metabolic control, which emphasizes the importance of collaboration between dental and medical professionals in the comprehensive care of patients with diabetes.[1,2]

In recent years, increasing attention has been directed toward the molecular basis of the oral–systemic relationship in diabetes, including inflammatory signalling, oxidative stress, altered immune responses, tissue-remodelling pathways, microbial dysbiosis and changes in salivary composition. These developments have expanded the understanding of diabetes-associated oral disease beyond the description of clinical manifestations and have created opportunities for identifying novel biomarkers and potential therapeutic targets.

The present review therefore aims to provide an integrated overview of the clinical spectrum of oral manifestations associated with diabetes mellitus, their underlying pathophysiological mechanisms, the bidirectional relationship between diabetes and periodontal disease, and their clinical implications.

## PATHOGENESIS OF DIABETES-ASSOCIATED ORAL CHANGES

The pathogenesis of diabetes-associated oral changes is multifactorial and involves the interaction of persistent hyperglycaemia, metabolic disturbances, vascular alterations, immune dysfunction and chronic inflammation. The severity of diabetic complications is generally related to the degree and duration of hyperglycaemia, and the oral cavity may be particularly susceptible because of its extensive vascular and neural supply.[6,7]

Persistent hyperglycaemia can initiate several intracellular pathways involved in diabetic tissue injury. Increased intracellular glucose promotes mitochondrial production of reactive oxygen species (ROS) and activates the polyol pathway, the advanced glycation end product (AGE)–RAGE pathway, the protein kinase C pathway and the hexosamine pathway. These mechanisms contribute to oxidative stress, endothelial dysfunction and a pro-inflammatory state, which may adversely affect oral tissues.[7]

Immune dysfunction is another important mechanism. Patients with diabetes may exhibit impaired polymorphonuclear leukocyte function, including abnormalities in adhesion, chemotaxis, phagocytosis and bactericidal activity, together with alterations in antigen responses and T-lymphocyte function.[6,7] These changes reduce the effectiveness of host defence against microorganisms and may increase susceptibility to periodontal disease and oral infections. Increased adherence of microorganisms to epithelial cells may further facilitate microbial colonization.

Diabetes is also associated with a persistent pro-inflammatory state. Metabolic and vascular disturbances can activate endothelial cells and promote the expression of adhesion molecules and chemotactic factors, resulting in recruitment of inflammatory cells into affected tissues. The subsequent release of inflammatory mediators, including interleukin-1β and interleukin-6, may amplify and sustain local inflammation.[7] In periodontal tissues, impaired immune regulation and persistent inflammatory activity may contribute to increased tissue destruction and progression of periodontal disease.

Vascular alterations may further compromise oral tissue homeostasis. Diabetes-associated endothelial dysfunction can produce abnormal blood flow and vascular changes, potentially affecting tissue perfusion and repair. These abnormalities, together with impaired immune responses, may contribute to delayed healing and increased susceptibility to infection after tissue injury.[7]

Salivary dysfunction provides another important link between diabetes and oral disease. Hyposalivation can reduce the mechanical cleansing, buffering and antimicrobial functions of saliva, thereby increasing susceptibility to oral infections such as candidiasis. Diabetes-associated oral changes may therefore result from the combined effects of metabolic, vascular, inflammatory, immune and salivary disturbances rather than from hyperglycaemia alone.

## MAJOR ORAL MANIFESTATIONS OF DIABETES MELLITUS

### Periodontal Disease

Periodontal disease is one of the most important oral complications of diabetes mellitus and represents a clinically significant interaction between systemic metabolic dysfunction and oral inflammation. It includes gingivitis, characterized by inflammation of the gingival tissues without loss of periodontal attachment, and periodontitis, in which persistent inflammation results in destruction of the periodontal ligament, alveolar bone and other supporting structures. Diabetes is recognized as an important modifying factor that can increase susceptibility to periodontal disease and influence its severity and progression.[6,7]

The increased periodontal susceptibility of diabetic individuals is related to persistent hyperglycaemia, impaired host defence, vascular alterations and an exaggerated inflammatory response. Hyperglycaemia can impair neutrophil chemotaxis, phagocytosis and bactericidal activity, reducing the ability of periodontal tissues to control the subgingival microbial challenge. Altered immune responses may subsequently promote persistent inflammation and periodontal tissue destruction. Diabetes-associated microvascular changes may further compromise periodontal tissue perfusion and repair, while alterations in collagen metabolism can affect the integrity and turnover of connective tissues.

Clinically, diabetic patients may present with increased gingival inflammation, periodontal pocket formation, clinical attachment loss, alveolar bone destruction, tooth mobility and periodontal abscesses. Periodontal disease may be more severe in individuals with inadequate glycaemic control, and periodontal destruction may increase with longer duration of diabetes. Diabetes may therefore influence not only the occurrence of periodontal disease but also its progression and response to treatment.[6,7]

The relationship between diabetes and periodontal disease is considered bidirectional. Diabetes can increase susceptibility to periodontal inflammation and tissue destruction, while chronic periodontal inflammation may contribute to systemic inflammatory burden and impaired metabolic control. Periodontal inflammatory mediators and bacterial products can enter the systemic circulation and potentially influence insulin sensitivity and glucose metabolism, so persistent periodontal inflammation may make optimal glycaemic control more difficult to achieve.[8] Clinical studies have demonstrated an association between periodontal inflammatory burden and metabolic control in individuals with type 2 diabetes.[3]

This bidirectional relationship has important clinical implications. Periodontal assessment should form part of comprehensive oral care for patients with diabetes, particularly those with poor glycaemic control or long-standing disease. Maintenance of periodontal health through appropriate oral hygiene, professional periodontal care and control of local inflammatory factors may help reduce the oral burden of diabetes and support overall metabolic health.[8]

### Xerostomia and Salivary Gland Dysfunction

Xerostomia and salivary gland dysfunction are frequently reported oral complications in individuals with diabetes mellitus. Xerostomia refers to the subjective sensation of dry mouth, whereas hyposalivation represents an objectively reduced salivary flow. Although these conditions may occur independently, reduced salivary secretion can contribute substantially to the sensation of oral dryness and may compromise several protective functions of saliva.[6,9]

Diabetic individuals have been reported to experience xerostomia more frequently than non-diabetic individuals, and reduced salivary flow has been observed in both type 1 and type 2 diabetes. A systematic review found that the prevalence of xerostomia among diabetic patients ranged from 12.5% to 53.5%, compared with 0–30% among non-diabetic populations.[9] The association appears to be influenced by glycaemic control, as patients with poorer glycaemic control may have lower salivary flow and a greater prevalence of oral dryness.[7,9]

Several mechanisms may contribute to salivary dysfunction in diabetes. Persistent hyperglycaemia can cause polyuria and osmotic diuresis, potentially resulting in dehydration and reduced salivary secretion. Diabetes-associated autonomic or peripheral neuropathy may also influence salivary gland function, although the evidence for this mechanism remains inconsistent. In addition, structural and functional alterations of the salivary glands, increased oxidative stress, inflammatory changes and metabolic disturbances may contribute to impaired salivary function. Medications frequently used by patients with diabetes, including antihypertensive and other xerogenic drugs, may further aggravate oral dryness.[7]

Reduced salivary flow has important consequences for oral health because saliva provides lubrication, buffering, antimicrobial activity and mechanical clearance of microorganisms and food debris. Salivary dysfunction may therefore increase susceptibility to dental caries, oral candidiasis, mucosal discomfort, taste disturbances and other oral complications. Increased salivary glucose concentrations may additionally favour microbial proliferation and Candida colonization, potentially contributing to the increased occurrence of oral infections in susceptible individuals.[6,9]

### Dental Caries

Dental caries is another oral condition that may be influenced by diabetes mellitus, particularly when glycaemic control is poor. The association is multifactorial and may involve alterations in salivary flow, salivary composition, oral microbial ecology and metabolic control. Reduced salivary flow during periods of poor glycaemic

control can decrease the natural cleansing, buffering and antimicrobial functions of saliva, creating a favourable environment for cariogenic microorganisms and demineralization.[10,11]

Changes in the oral microbial environment may further contribute to caries development. Increased blood glucose levels may be accompanied by alterations in the salivary environment and increased colonization by acidogenic and aciduric bacteria. In a study of diabetic individuals with dental caries, increased *Streptococcus mutans* counts were observed together with higher caries indices, supporting an association between diabetes, oral microbiota and caries development.[12]

The relationship between diabetes and dental caries may also be influenced by metabolic control and salivary characteristics. Alterations in salivary pH, flow rate and other protective factors can modify caries susceptibility, while dietary habits, oral hygiene, age and duration of diabetes may act as additional contributing factors.[10,11] Dental caries in diabetic individuals should therefore be viewed as the result of an interaction between systemic metabolic disturbances and established local cariogenic factors rather than as a direct consequence of diabetes alone.

Regular caries-risk assessment, maintenance of adequate oral hygiene, dietary counselling and appropriate preventive dental care are therefore particularly important in diabetic patients, especially those with poor glycaemic control or reduced salivary flow.

**Oral Candidiasis and Other Oral Infections**

Oral candidiasis is an important opportunistic infection that may occur more frequently in individuals with diabetes mellitus. *Candida albicans* is the predominant species, although non-*albicans Candida* species may also be isolated. The increased susceptibility of diabetic patients is likely related to the combined effects of altered salivary function, elevated salivary glucose, impaired host immune responses and local environmental changes within the oral cavity.[13]

Hyperglycaemia may favour Candida colonization by increasing glucose availability in saliva, while reduced salivary flow and altered salivary pH can further impair the natural antimicrobial and cleansing functions of saliva. In addition, diabetes-associated impairment of polymorphonuclear leukocyte chemotaxis and phagocytosis may reduce the ability of the host to control fungal proliferation. These factors can facilitate Candida adherence, colonization, biofilm formation and the subsequent development of symptomatic infection.[13]

Clinical studies support increased oral Candida carriage among individuals with diabetes. Mohammadi et al. reported Candida colonization in 55% of diabetic patients compared with 35.5% of non-diabetic controls, with *C. albicans* being the most frequently isolated species; higher Candida loads were associated with increased salivary glucose, reduced salivary pH, xerostomia and poor oral hygiene.[13] Oral Candida infection has also been reported in diabetic patients with periodontal disease, suggesting that multiple diabetes-associated oral conditions may coexist and reinforce one another.[14]

Clinically, oral candidiasis may present as pseudomembranous or erythematous lesions, burning or soreness, altered taste and mucosal discomfort. Recurrent or persistent candidal infection should therefore prompt assessment of local predisposing factors as well as glycaemic control. Appropriate oral hygiene, management of xerostomia, control of diabetes and timely antifungal therapy are important components of management.

**Oral Mucosal Manifestations**

Diabetes mellitus has been associated with a range of oral mucosal alterations, although the occurrence and clinical significance of these lesions vary among individuals. Altered immune responses, vascular changes, xerostomia, increased susceptibility to infection and impaired tissue repair may contribute to mucosal abnormalities in diabetic patients. Commonly reported conditions include angular cheilitis, geographic tongue, fissured tongue, recurrent ulceration, oral lichen planus or lichenoid reactions and denture-related stomatitis.[6]

**Angular cheilitis** is characterized by erythematous, fissured lesions at the corners of the mouth and may be associated with saliva accumulation, Candida colonization, denture use and reduced vertical dimension. In diabetic individuals, impaired host defence and a favourable local environment may increase susceptibility to this condition. A study of oral mucosal alterations reported angular cheilitis in approximately 15% of individuals with type 2 diabetes, compared with 9% of controls.[15]

**Tongue abnormalities** have also been described in diabetic patients. Geographic tongue, or benign migratory glossitis, presents as well-demarcated erythematous areas resulting from loss of filiform papillae and has been reported more frequently in some diabetic populations. Fissured tongue may also occur, with xerostomia and reduced salivary flow potentially contributing to the condition.[16]

Other mucosal findings include recurrent traumatic or aphthous ulceration, oral lichen planus, lichenoid reactions and denture stomatitis. Denture stomatitis may be particularly relevant in diabetic denture wearers because altered salivary function and increased Candida colonization can promote mucosal inflammation, and higher frequencies of denture stomatitis have been reported among diabetic individuals than among controls.[15,16]

Although many of these lesions are not specific to diabetes, their presence, particularly when recurrent or accompanied by xerostomia, candidiasis, periodontal disease or poor glycaemic control, may warrant evaluation of the patient's systemic and metabolic status. Careful examination of the oral mucosa and tongue should therefore form part of routine dental assessment in patients with diabetes.[6]

**Delayed Oral Wound Healing**

Delayed wound healing is an important oral complication of diabetes mellitus and may become clinically relevant after tooth extraction, periodontal surgery, implant placement and other invasive dental procedures. Diabetes can adversely affect several stages of wound repair, resulting in prolonged inflammation, impaired tissue regeneration and delayed restoration of normal tissue architecture.[2]

Persistent hyperglycaemia and the accumulation of advanced glycation end products (AGEs) contribute to oxidative stress and an exaggerated inflammatory response, which can interfere with the normal progression of wound healing. Diabetes-associated abnormalities in cellular function may affect epithelial cells, fibroblasts, endothelial cells and other cells involved in tissue repair.[17]

Vascular alterations also play an important role in impaired healing. Damage to small blood vessels and impaired angiogenic responses can compromise the delivery of oxygen and nutrients required for tissue regeneration, and altered immune function and a reduced ability to control infection may further prolong the inflammatory phase and increase the risk of postoperative complications.[2]

Experimental evidence suggests that diabetes can also interfere with growth factor-mediated tissue repair. In a diabetic animal model, epithelial migration after tooth extraction was delayed compared with controls, and expression of transforming growth factor-beta (TGF-β1, TGF-β2 and TGF-β3) and their receptors was substantially downregulated during healing, suggesting that impaired TGF-β signalling may contribute to delayed oral wound repair in diabetes.[18]

Clinically, impaired healing may manifest as prolonged healing of extraction sockets, persistent soft-tissue defects, increased susceptibility to infection or delayed osseous healing. Evaluation of glycaemic status, appropriate treatment planning, meticulous infection control and careful postoperative monitoring are therefore particularly important when invasive dental procedures are performed in patients with diabetes.[17]

## DIABETES–PERIODONTITIS: A BIDIRECTIONAL RELATIONSHIP

The relationship between diabetes mellitus and periodontal disease is increasingly recognized as bidirectional, with each condition potentially influencing the onset, progression and severity of the other. Diabetes increases susceptibility to periodontal inflammation through hyperglycaemia-associated immune dysfunction, vascular alterations and an altered inflammatory response, while persistent periodontal inflammation may contribute to systemic inflammatory burden and adversely influence glycaemic control.

In individuals with diabetes, chronic hyperglycaemia can impair neutrophil function and alter the host response to periodontal microorganisms. Changes in leukocyte chemotaxis, phagocytosis and bactericidal activity may reduce effective microbial control and facilitate persistent periodontal inflammation, and diabetes-associated vascular abnormalities and alterations in connective-tissue metabolism may further compromise periodontal tissue integrity and repair.[6]

The influence of periodontitis on diabetes is thought to involve the systemic effects of chronic periodontal inflammation. Inflamed periodontal tissues can release pro-inflammatory mediators and other inflammatory products into the circulation, potentially contributing to systemic inflammation and impaired insulin sensitivity. The inflammatory burden of untreated periodontitis may therefore make metabolic control more difficult in susceptible individuals.[8]

Clinical evidence supports this interaction. Romano et al. demonstrated an association between periodontal inflammatory burden and metabolic control in patients with type 2 diabetes, highlighting the potential relationship between periodontal inflammation and glycaemic status.[3] This relationship is clinically relevant because poor glycaemic control may worsen periodontal disease, while persistent periodontal inflammation may in turn contribute to deterioration of metabolic control.

Management of diabetes should therefore include attention to periodontal health, and patients with periodontitis should be evaluated for diabetes-related risk factors when clinically appropriate. An integrated approach involving physicians and dental professionals can facilitate early recognition, prevention and treatment of both conditions. Regular periodontal assessment, effective plaque control, professional periodontal therapy and appropriate management of diabetes may help reduce the combined systemic and oral inflammatory burden.[7]

## CLINICAL IMPLICATIONS AND MULTIDISCIPLINARY MANAGEMENT

The close relationship between diabetes mellitus and oral disease highlights the need to integrate oral health into routine diabetes care. Dental professionals should be alert to periodontal disease, xerostomia, recurrent oral infections, mucosal lesions and delayed wound healing, particularly in patients with poor glycaemic control. Early identification and appropriate dental management may help reduce oral morbidity and improve quality of life.[7]

Management should emphasize preventive oral care, periodontal treatment, control of oral infections and regular dental follow-up, together with adequate glycaemic control. Patients with xerostomia may require measures to maintain oral moisture and reduce caries risk, while recurrent candidiasis should prompt attention to local predisposing factors and metabolic control.[2,9]

A multidisciplinary approach involving physicians, dentists and patients is essential. Before invasive dental procedures, glycaemic status, existing infections, medications and healing capacity should be considered, because diabetes-associated vascular and immune alterations may delay wound healing.[17] Effective communication between medical and dental professionals can facilitate timely referral, optimization of metabolic and periodontal health and comprehensive long-term management.

## FUTURE PERSPECTIVES

Future research should focus on strengthening the integration of oral and systemic diabetes care. Greater attention to oral findings in patients with diabetes may facilitate earlier recognition and management of periodontal disease, salivary dysfunction, oral infections and other complications. Further prospective studies are needed to clarify the bidirectional relationship between periodontal inflammation and glycaemic control and to determine the extent to which effective management of oral disease can contribute to improved metabolic outcomes.[3,7]

The potential use of salivary and oral biomarkers for monitoring metabolic status and identifying individuals at increased risk of oral complications also warrants further investigation. Larger, well-designed longitudinal studies may help to establish clinically useful biomarkers and clarify the mechanisms linking hyperglycaemia with oral tissue changes. Future approaches should ultimately emphasize personalized prevention, early diagnosis and coordinated medical–dental care to reduce the oral and systemic burden of diabetes.

## CONCLUSION

Diabetes mellitus is associated with a broad spectrum of oral manifestations resulting from the complex interaction of hyperglycaemia, immune dysfunction, vascular alterations, inflammation, salivary dysfunction and impaired tissue repair. Periodontal disease represents the most clinically significant oral complication, with evidence supporting a bidirectional relationship between periodontal inflammation and glycaemic control. Other manifestations, including xerostomia, dental caries, oral candidiasis, mucosal alterations and delayed wound healing, may further affect oral health and quality of life.

Recognition and management of these oral complications should therefore form an integral part of comprehensive diabetes care. Regular dental assessment, preventive oral care, appropriate periodontal and infection management and adequate glycaemic control are important for reducing oral morbidity. Close collaboration between dental and medical professionals, taking into account both the oral and systemic aspects of diabetes, can facilitate early detection, appropriate intervention and long-term management, and may ultimately improve overall patient outcomes.

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