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Review Article **Dentistry**

Diabetes Mellitus in Dental Practice: Oral Manifestations and Clinical Management Considerations

Bashayr Faisal Alanazi[1]*, Mohammed Abdulrahman Alghamdi[2], Fai Hamad Aloraini[3], Noura Mohammed Abdulrahman Aljadaan[4], Hebah Sabih Alenazi[5], Kadi Saleh Alharbi[6], Farah Nabil Khayraldeen[7], Nadeen Tariq Alreefi[8], Thamer Abdulrahim Alqurashi[9], Reema Omar Alluqmani[10], Ruba Mualla Aljohani[11], Ghadeer Ali Asiri[12], Yasir Abdullatif Mansour[13], Abdulaziz Mohammed Altalhi[14]

[1]General Dentistry, Vision College, Dentistry Department, Al Riyadh, Saudi Arabia
[2]General Dentistry, King Abdulaziz University, Faculty of Dentistry, Jeddah
[3]General Dentistry, King Saud bin Abdulaziz University for Health Science, College of Dentistry, Riyadh, Saudi Arabia
[4]General Dentistry, King Saud bin Abdulaziz University for Health Science, College of Dentistry, Riyadh, Saudi Arabia
[5]General Dentistry, King Saud bin Abdulaziz University for Health Science, College of Dentistry, Riyadh, Saudi Arabia
[6]Dental intern, Qassim University, Dentistry College. Qassim, Saudi Arabia
[7]General Dentistry, Taibah University, Dentistry College, Al-Madinah Al-Munawwara, Saudi Arabia
[8]General Dentistry, Taibah University, Dentistry College, Al-Madinah Al-Munawwara, Saudi Arabia
[9]General dentistry, Umm Alqura University, Dentistry College, Makkah, Saudi Arabia
[10]General Dentist, Ibn Sina National College, Dental Department, Jeddah, Saudi Arabia
[11]General Dentist, Ibn Sina National College, Dental Department, Jeddah, Saudi Arabia
[12]General dentist, King Khalid University, Dentistry College, Abha, Saudi Arabia
[13]Dental Intern, King Abdulaziz University, Dentistry College, Jeddah, Saudi Arabia
[14]Periodontics consultant, Ministry of Health, Riyadh, Saudi Arabia

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***Corresponding author:** Bashayr Faisal Alanazi
General Dentistry, Vision College, Dentistry Department, Al Riyadh, Saudi Arabia

Abstract

Diabetes mellitus (DM) is a global metabolic disorder characterized by chronic hyperglycemia resulting from impaired insulin secretion, insulin resistance, or both. Its prevalence continues to rise worldwide, with substantial morbidity linked to microvascular and macrovascular complications that influence overall health and the delivery of dental care. In dental practice, DM is clinically significant because it modifies host immunity, vascular function, inflammatory regulation, and tissue metabolism mechanisms that collectively increase susceptibility to oral infections and compromise healing. Common orofacial manifestations include increased risk and severity of gingivitis and periodontitis with alveolar bone loss, salivary gland dysfunction and xerostomia, oral mucosal lesions (including candidal infections and lichenoid reactions), dysgeusia, burning mouth symptoms, and, in severe settings, opportunistic deep fungal infections and osteomyelitis. These changes directly affect treatment planning across specialties. Prosthodontic management requires careful attention to salivary hypofunction, denture-related candidiasis, mucosal fragility, residual ridge resorption, and delayed wound healing, with emphasis on atraumatic techniques, hygiene reinforcement, and appropriate scheduling. Endodontic practice must consider the bidirectional relationship between apical periodontitis and glycemic control, the possibility of slower periapical healing, increased residual lesions, and the need for meticulous infection control and stress reduction to limit hyperglycemic episodes. Orthodontic therapy, particularly in patients with suboptimal glycemic control, demands thorough periodontal screening, the use of light physiological forces, close monitoring, and coordination with the patient's medical team to reduce risks of infection, impaired healing, and hypoglycemic emergencies. This review synthesizes systemic and oral evidence to provide practical, clinically oriented recommendations for safe and effective dental management of patients with DM, with glycemic control and interprofessional collaboration as central determinants of favorable outcomes.

Keywords: Diabetes Mellitus; Oral Manifestations; Xerostomia; Periodontitis; Prosthodontics; Endodontics; Orthodontics; Wound Healing; Glycemic Control.

INTRODUCTION

The human body is designed in an incredible manner, with a unique ability to maintain a stable and constant internal environment. It depends on feedback mechanisms and internal signals such as hormones and chemical signaling pathways to respond to external stresses and challenges like a change in temperature, pH and blood glucose levels, through its highly and minutely organized and well-regulated endocrine system. This steady state is termed as "homeostasis." However, many times this delicate mechanism is disturbed leading to a disorder or a disease.

Diabetes mellitus (DM) is a complex metabolic disorder characterized by the disturbances in carbohydrate, protein and lipid metabolism. The primary feature of this disorder is an elevated blood glucose level, either due to a reduction in the insulin secretion or an increase in the target tissue resistance to the action of insulin, or both the cases [1].

Globally, an estimated 9.5 million people are living with type 1 diabetes (T1D) in 2025, an increase of 1.1 million people (13 %) since the previous estimate of 8.4 million in 2021 [2]. Out of the 9.5 million people suffering from this silent killer disease, a major concern is the significant proportion of this burden being shared by the youth aged < 20 years who are mostly represented in low- and low to middle-income countries. Globally, the number of people living with T1D in 2025 would be 13.9 million or 46 % higher, if the mortality of people with T1D matched that of the general population in their countries.

The increase in the global number of prevalent cases i.e 9.5 million cases in 2025 compared to 8.4 in 2021 – a 13 % increase, is largely due to combined impact of increasing incidence (including improved diagnosis rates), population growth, aging and falling mortality. In lower-income countries (lower-middle and low-income countries), prevalent cases increased by 20 % from 1.8 million in 2021 to 2.1 million in 2025. There is a different trend in each country and the individual country data from countries, such as Kazakhstan [3], and Maldives [4] show substantially higher annual increases in incidence than the modelled incidence over the time estimate in their respective regions.

The global trend shows that the prevalence of diabetes is steadily increasing at a rate of 2.5% per year [5]. The number of people with diabetes is expected to exceed 570 million by 2025, and the number of deaths due to diabetes is expected to reach 1.59 million [6]. Most of the deaths occurring in younger age group i.e. in children and youth with this condition is due to non-diagnosis of the younger age group dying in ketoacidosis or misdiagnosed as another condition or not getting proper and professional care [7–12].

The impact of population size and age distribution is evident from the fact that India having the highest prevalence of T1D in youth aged < 20 years, and both India and China being in the 10 countries with the highest numbers of T1D in people of all ages, despite having lower reported incidence rates of T1D when compared to European populations such as Finland and Sweden [13].

As already mentioned, the peak age of onset is in childhood but most people diagnosed with T1D are adults. Also, with the median global age for a person living with T1D being 36 years, and 1.1 million people living with T1D are aged ≥ 60 years, efforts to improve T1D care should focus on adult as well as younger age-groups [14].

Pathogenesis and Classification

In 2010 and 2013, Wilson et al and Aguirre et al have classified Diabetes Mellitus (DM), into four main categories. Type 1 DM, type 2 DM, gestational DM and other causes. Their classification is on the basis of etiology with type 1 DM representing a total destruction of B-cell which leads to severe deficiency in insulin secretion and Type 2 representing a deficiency in the amount of insulin secretion or the poor cellular response to the hormone. The third category is the Gestational DM which is seen in pregnant women with the pregnant women being commonly affected by diabetes during their second and third trimester. Lastly, there are a large variety of diabetes due to other causes which do not fall in any of the above case. They represent themselves as monogenic diabetes syndromes and genetic defects, disease of exocrine pancreas or drug, eg, cyclosporine or chemical diabetes. [15,16]

In addition, according to the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, in 1998 classified it on the basis of its etiology. DM is divided into primary and secondary. Primary DM involves type 1 and type 2 where type 1 can involve type A immune mediated or type B idiopathic DM whereas Type 2 DM includes insulin resistance or an insulin secretory defect. Secondary diabetes can be due to multitude of reasons such as pancreatic disease, hormonal imbalance, severe illness, gestational diabetes, genetic syndromes, drug therapy, autoimmune endocrinopathies, and insulin resistance etc. [17].

Diabetes Mellitus can be a result of two main patho-physiological processes. Firstly, it can be due to a deficiency in insulin secretion and this is considered the most common one. The second process is that the insulin receptors do not work properly; therefore, as a compensatory mechanism, there is an increase in insulin secretion. Or it can have a genetic defects or environmental reasons or both leading to a state of insulin resistance [18].

Pathogenesis: Type 1 DM

Destruction of insulin producing cells as a result of autoimmune disease is the prime cause of type1DM. Macrophages, $CD4^+$ and $CD8^+$ T cells are the primary cells that are involved in this self destruction [19].

Circulating anti-insulin antibodies are detected even before receiving insulin therapy in most of these patients. The glutamic acid decarboxylase, located within pancreatic B cells are targeted by islet cell antibodies [20]. As a result of pancreatic β-cells destruction, insulin production is affected which leads to metabolic derangement. Moreover, pancreatic α-cells also show aberrant behavior in which excessive secretion of glucagon takes place. Physiologically, there is a reverse relationship between hyperglycemia and glucagon production. However, this process is disturbed in patients with type 1 DM in which glucagon level is elevated with hyperglycemia [21].

Furthermore, insufficient insulin secretion has a direct effect on metabolism of lipid leading to uncontrolled lipolysis and an increase in the amount of free fatty acids in the plasma, leading to further decrease in glucose metabolism in peripheral tissues such as skeletal muscles [22].

Moreover, insulin deficiency can cause genetic defects affecting the expression of a number of genes essential to respond normally to insulin. Glucokinase in liver and the GLUT 4 class of glucose transporters in adipose tissue are affected leading to an impairment in the metabolism of glucose, lipid and protein [22].

Pathogenesis: Type 2 DM

In this type of diabetes there is either a disturbance in the insulin production or a disturbance leading to tissue resistance to insulin.[17]

The outcome of insulin resistance and hyperinsulinemia is Glucose tolerance discrepancy. However, in the case of maturity onset diabetes of the young (MODY), a type of type 2 diabetes mellitus the mechanism is still under study but it could be a mutation in glucokinase gene on chromosome 7p. [23]

MODY is known as "hyper-glycemia diagnosed before the age of twenty-five years and treatable for over five years without insulin in cases where islet cell antibodies (ICA) are negative" [24].

Complications of Diabetes Mellitus

The complications associated with diabetes are extensive and elaborate and can be categorized into micro vascular and macro vascular. Micro vascular complications include retinopathy, nephropathy, and neuropathy, which occur when hyperglycemia directly affects small blood vessels, such as capillaries and small arteries. Conversely, macro vascular complications include coronary artery disease (CAD), cerebrovascular disease, and peripheral arterial disease (PAD), which occur when hyperglycemia promotes atherosclerosis, resulting in damage to the macrovasculature [25-28].

1. Diabetic Retinopathy (DR)

DR is the most common micro vascular complication and a leading cause of vision loss globally. It affects approximately one-third of diabetic patients.

- Pathophysiological Mechanisms: [25]
  - Hyperglycemia and oxidative stress
  - Activation of the polyol pathway
  - Vascular endothelial growth factor (VEGF)

- The Clinical Presentation and Diagnosis: [25]
  - Non-proliferative DR (NPDR) is characterized by microaneurysms, intraretinal hemorrhaging, and cotton-wool spots, indicating early retinal ischemia.
  - Proliferative DR (PDR) is characterized by neovascularization, vitreous hemorrhaging, and tractional retinal detachment, which can lead to severe vision loss if left untreated.

Fundoscopic examinations and optical coherence tomography are essential tools for diagnosing and monitoring the progression of DR. Fluorescein angiography can also be used to assess retinal blood flow abnormalities.

- Current and Emerging Treatments [25]
  - Anti-VEGF therapy
  - Corticosteroid injections
  - SGLT2 inhibitors

2. Diabetic Neuropathy (DN)

DN is a leading cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD) affecting approximately 30%-40% of patients with diabetes and is characterized by progressive albuminuria, a declining glomerular filtration rate (GFR), and the development of glomerulosclerosis [29].

- Pathophysiological Mechanisms
  - Glomerular hyperfiltration
  - Renal hypertrophy and inflammation
  - AGEs [30]
  - Dysregulation of the renin-angiotensinaldosterone system (RAAS) [31]

- The Clinical Presentation and Diagnosis: [25]
  - Microalbuminuria: (30-300 mg/day) indicating initial glomerular damage
  - Macroalbuminuria: ($>$ 300 mg/day) signaling advanced kidney damage.
  - Declining GFR

- Treatment Strategies [25]
  - RAAS inhibition
  - SGLT2 inhibitors [32,33]
  - GLP-1 receptor agonists

3. Diabetic Neuropathy

Diabetic neuropathy affects up to 50% of patients with diabetes

• Clinical Manifestations: Diabetic Neuropathy is of the following types:

- ○ Peripheral neuropathy: Symptoms include numbness, tingling, and burning pain, which typically start in the feet and progress proximally.
- ○ Autonomic neuropathy: Involves dysfunction of the autonomic nervous system, leading to gastroparesis, orthostatic hypotension, and bladder dysfunction.

• Treatment Approaches [25]

- ○ Pain management: First-line treatments include anticonvulsants (e.g. gabapentin and pregabalin) and antidepressants (e.g. duloxetine), which help alleviate neuropathic pain [34].
- ○ Emerging therapies: GLP-1 receptor agonists and SGLT2 inhibitors [35].

4. Cardiovascular Diseases (CVD)

It includes coronary artery disease, myocardial infarction, and heart failure. Diabetic patients due to insulin resistance, dyslipidemia, chronic inflammation, and endothelial dysfunction have a two- to four-fold increased risk of CVD compared to non-diabetic individuals [36, 37].

• Pathophysiological Mechanisms [25]

- ○ Insulin resistance and lipid dysregulation
- ○ Chronic inflammation
- ○ Oxidative stress and endothelial dysfunction:
- ○ AGEs and RAGE activation

• Therapeutic Approaches [25]

"Guidelines for the Prevention of Atherosclerotic Cardiovascular Diseases 2022 was published by The Japan Atherosclerosis Society with the aim of preventing atherosclerotic diseases [38].

- ○ Statin therapy
- ○ Antiplatelet agents
- ○ SGLT2 inhibitors and GLP-1 receptor agonists

5. Cerebrovascular Disease [25]

Diabetes increases the risk of stroke.

• Pathophysiological Mechanisms

- ○ Hyperglycemia - induced endothelial dysfunction
- ○ Enhanced thrombogenesis
- ○ Vascular fragility and hemorrhagic stroke

• Clinical Management

- ○ Anticoagulant therapy
- ○ Blood pressure control

6. Peripheral Arterial Disease (PAD) [25]

Patients with diabetes have a four-fold increased risk of developing PAD

• Pathophysiological mechanisms

- ○ Microvascular compromise
- ○ Inflammatory pathways

• Diagnosis and Advanced Therapies:

The ankle-brachial index (ABI) is a non-invasive diagnostic tool for PAD.

- ○ Revascularization techniques
- ○ Emerging pharmacotherapies: SGLT2 inhibitors and anti-inflammatory agents

Diagnosis

According to the American Diabetic Association, at least one of the following conditions must exist to establish the diagnosis of DM: [39]

1. Fasting plasma glucose ≥126 mg/dl (≥7 mmol/L)
2. Presence of the classic symptoms of diabetes (polyuria, polyphagia, polydipsia, visual blurring, thrush, lethargy, and unexplained weight loss), with unequivocal hyperglycemia (random plasma glucose ≥200 mg/dl [11 mmol/L])
3. Abnormal Oral Glucose Tolerance Test (OGTT): This involves giving the patient 75 g of glucose in 300 ml of water to drink the morning after an overnight fast. Blood glucose is measured every 30 min for 2 h. The test is positive if the 2-hour plasma glucose ≥200 mg/dl (11 mmol/L)
4. Hemoglobin A1c (HbA1c) ≥6.5%: HbA1c is a form of hemoglobin that is measured primarily to identify the average plasma glucose concentration over a period of 2–3 months. This test therefore is useful to monitor glycemic control in diabetic patients.

Orofacial Manifestations [40].

1. Dento-alveolar: increased risk and severity of gingivitis, periodontitis, and alveolar bone loss.
2. Salivary glands: Salivary gland swelling (sialosis) and hypofunction (xerostomia).
3. Oral mucosa: lichenoid white lesion, candidal infection, and tongue depapillation.
4. Orofacial sensations: burning tongue or mouth, altered taste, and circumoral paraesthesia.
5. Jaw bones and paranasal sinuses: increased risk of osteomyelitis and deep fungal infections (mucormycosis).
6. Cranial nerves: cranial nerves function, especially oculomotor nerve, might be affected as a result of diabetic mononeuropathy.
7. Halitosis (bad breath) due to diabetic ketoacidosis

Dental considerations:

I) Prosthetic considerations:

- ➢ The factors that should be considered in a diabetic patient before and during the prosthodontic treatment are discussed below:

a. Salivary hypofunction [41]:

Diabetes Mellitus causes a qualitative and a quantitative effect on the parenchyma of major salivary glands leading to hypo salivation. The possible mechanism could be the substitution of normal glandular tissue by adipose tissue decreasing saliva production [42]. Patient has difficulty in wearing the denture and has a constant irritation and burning sensation in the oral mucosa [43].

b. Candida infections and Denture stomatitis:

Candidal infection occurs due to change in pH, increased salivary glucose levels and immune dysregulation in diabetic patients, over the normal hypo salivation seen in diabetics. The manifestations of oral candidiasis may occur in as median rhomboid glossitis, atrophic glossitis, denture stomatitis and angular chelitis [44].

c. Poor wound healing:

Poor blood supply to the tissues, microvascular angiopathic changes, reduced oxygen to the cells, reduction of collagen production, increased collagenase activity leads to impaired or delayed wound healing [41]. So, care needs to be taken while planning pre-prosthetic surgery or dental implant placement, and they should be performed only when normal glycemic levels are achieved [43].

e. Burning mouth syndrome

Altered taste sensation, burning mouth syndrome and dysphagia is commonly experienced by diabetic patients and this could be due to the variations in the salivary flow and changes in the buffering capacity of saliva. Even peripheral neuropathy may play a role in this condition [45].

f. Increased caries risk

In diabetes mellitus, there is increased risk of caries and periodontal problems [43]. Decreased salivary flow and pH and increased pathogenic bacterial growth in the mouth increases the risk of caries due to a change in the oral environment [41].

g. Residual ridge resorption

In diabetic patients decreased blood supply to the tissues because of microvascular angiopathy increase the amount of residual ridge resorption [43].

h. Antibiotic consideration

Antibiotic coverage (Penicillin V of Erythromycin) is strictly recommended in diabetic patients before implant placement or invasive surgery [43].

i. Anaesthetic Consideration

Epinephrine results in the precipitation of hyperglycemia by breaking down glycogen to glucose. So, excessive amount of epinephrine in local anesthesia and in gingival retraction cords should be avoided [43]. In place of epinephrine, either alumina or zinc chloride-based retraction cords are preferred in these patients [46].

➢ **PROSTHETIC THERAPY IN DIABETICS:**

1. **Medical History:** It is important to take proper medical history of the patient. Patients Blood glucose levels both fasting and HbAIC, medication, dosage and timing of medication taken should be enquired. In all the patients older than 45 years of age it is very important to assess glucose level at the initial appointment [41].
2. **Diet:** always ensure that patient has had his/her breakfast and medication before prosthetic treatment as it can take a little while. [41].
3. **Scheduling of the Patient's Visit:** Diabetic patients should be scheduled preferably in the morning as endogenous cortisol level is higher during morning time and they in turn increases blood glucose levels. [41]. Patient must be instructed to consult his or her physician before initiating any procedure [47].
4. **Complete denture**: Diabetic patients lose resilience of Oral mucous membrane due to xerostomia which indirectly affect the retention of complete denture. So, a salivary reservoir can be made into a denture that provides slow, sustained, and continuous release of salivary substitute but it has a disadvantage of that the patient should manually refill the reservoir at regular intervals and high degree of precision is mandatory to ensure accurate and smoothly fitting of the reservoir lid [48].

Frequent relining and rebasing of complete denture is required due to more ridge resorption and this can be decreased by having broad area of coverage under the denture base [43]. Also, a decrease in the number of artificial teeth, decrease in the buccolingual width, improved occlusal tooth design are some of the other techniques that may also be used [49].

Due to more residual ridge resorption in diabetic patient, mucostatic or minimal pressure impression technique or neutral zone impression technique is recommended for impression making in such patients [43].

In diabetic patients, liquid supported denture bases are good alternatives then conventional dentures. Liquid supported dentures combine the features of being flexible in adapting to the mucosa and during function it is rigid. Liquid supported dentures causes preservation of residual ridge, better retention, stability and support, prevention of chronic soreness with comfort to the patients, and improved patient tolerance. Thus, on a long-term basis it provides benefit of soft liners and tissue conditioners [50].

5. **Removal Partial Denture (RPD):** All components of RPD must be designed appropriately such that prosthesis is tissue friendly. Proper oral hygiene and

denture hygiene or maintenance instructions should be given to the patients [41].

6. **Fixed Partial Denture (FPD):** It is better to keep the supragingival finish line to avoid damaging soft tissue. The chamfer margin applies less force or stress on weakened tooth so it is preferred and care should be takern to follow Ante's law. Proper flossing should be done to maintain the oral hygiene. During tooth preparation, care should be taken to avoid trauma to the soft tissue as diabetes patients have poor wound healing. Hygienic pontic should be preferred for their ease of cleansing action [41].
7. **Implant or Implant Supported Dentures**: Implant supported prosthesis are not indicated for uncontrolled diabetic patients. Proper medication must be provided before and after implant placement. Patient should maintain their sugar level even after the surgical placement of implants [41].

Several criteria like systemic glycemic level, glycemic control on bone, periodontal condition are considered for diabetic patients. The threats of poor wound healing, impaired osseointegration, increased chance of infection and periodontitis interfere with successful implant therapy. Proper patient selection with well controlled glycemic level and adequate antibiotic administration improves survival of dental implants in patients with diabetes [42].

II) Endodontic considerations

Apical Periodontitis (AP) is a common dental condition influenced by various systemic factors, including DM [51]. AP is prevalent worldwide, with individual-level rates reaching 52%. In a previous study done the pooled prevalence of individuals with at least one tooth with AP in T2DM individuals was found out to be 75%, compared to nondiabetic individuals who had a prevalence of only 62% [52]. Similar results are seen various studies [53, 54].

a) Impact of glycemic control on apical periodontitis

Studies done to investigate the relationship between glycemic control, AP, and the outcomes of endodontic therapy in patients with DM have diverse results, some studies have found no significant difference in AP healing between patients with good and poor glycemic control [55]. Others have reported a higher prevalence of AP and poorer endodontic outcomes in individuals with long-term diabetes [56-59].

A study shows that poor glycemic control (A1c ≥6.5%) is associated with a higher prevalence of AP [60]. Conversely, a more recent study found that type 2 diabetic patients with poorly controlled blood sugar levels (A1c ≥6.5%) did not have a higher prevalence of AP [55]. T2DM patients, especially those with elevated A1c levels, experienced less favorable treatment outcomes for RCT, including reduced radiographic lesions.[45] Individuals with poorly controlled DM are more likely to have root canal-treated teeth.[7,9,50] The long-term duration of DM has been linked to a greater prevalence of AP and a higher number of in root-filled teeth with AP.[48,49]

b) Effect of apical periodontitis on diabetes mellitus

Persistent AP infection may contribute to a heightened and dysregulated inflammatory state, impairing glycemic control and increasing insulin requirements [61]. It has been demonstrated in animal studies that AP can reduce insulin signaling in the blood [62], resulting in increased insulin resistance [63, 64].

c) The effect of diabetes mellitus on the endodontic outcome

A higher prevalence of residual AP lesions in DM patient's root-filled teeth than in healthy individuals were seen [65]. Furthermore, the healing of periapical lesions following primary endodontic treatment is significantly slower in DM patients than in nondiabetic patients [60,66]. In addition, DM patients are at a greater risk of root-filled teeth extraction [67].

The underlying biological mechanisms by which DM influences periapical tissue healing and root canal treatment (RCT) outcomes include:

1. Impaired Innate Immunity [68]
2. Hyperglycemia [56]
3. Advanced Glycation End Products [69]

➢ **ENDODONTIC THERAPY IN DIABETICS**

a. Healing after endodontic treatment

Hyperglycemia and the systemic complications of diabetes can adversely affect the healing process after endodontic treatment [70]. Delayed or incomplete healing following endodontic procedures, leading to lower success rates are seen in a study compared to non-diabetic individuals [71].

One of the key factors contributing to impaired healing in diabetic patients is the reduced blood supply to the periapical tissues due to microvascular changes including basement membrane thickening and endothelial dysfunction, hindering the healing process [72]. Another factor to consider is the potential for altered bone metabolism in diabetic patients as Hyperglycemia has been shown to interfere with osteoblast function and reduce bone formation, which may impair the resolution of periapical radiolucencies following root canal treatment. [73].

b. **Medical History**

Medical history is very important. When reviewing medical histories, a clinician should be aware of the cardinal signs of DM and should refer to a physician for diagnosis and treatment [74].

In diabetic patients, clinicians should ascertain how well controlled the diabetic condition of the patient is [75] for which the doctor should ask regarding most

recent test results (e.g., glycosylated haemoglobin and postprandial blood glucose levels), frequency of hypoglycaemic episodes, medication, dosage and timing [76].

Well controlled diabetic patients free of any complication are candidate for endodontic treatment. Acute infections in diabetic patients should be managed using incision and drainage, pulpectomy, antibiotics and warm rinses [46].

c. Pretreatment sedation

Emotional and physical stress increases the amount of cortisol and epinephrine secretion that induces hyperglycaemia So, if the patient is very apprehensive, pre-treatment sedation should be given [76].

Before the procedure it has to be ensured that the patient has eaten normally and taken medication as usual [74].

d. Prophylactic antibiotics

Prophylactic antibiotic are not indicated for endodontic surgery in well-controlled diabetics [77] Whereas when endodontic surgery is required in a poorly controlled diabetic, prophylactic antibiotic should be considered, also the surgery may increase insulin resistance in the postoperative period, requiring prescription of antibiotic [75].

e. Appointment schedule

Lengthy appointments should be avoided. If a lengthy, especially surgical, procedure is to be undertaken, the patient's physician should be consulted. Blood glucose level should be constantly monitored during a lengthy surgical procedure [78]. For patients receiving insulin therapy, appointments should be scheduled so that they do not coincide with peaks of insulin activity, since this is the period of maximal risk of developing hypoglycaemia [76].

III) Orthodontic considerations

It is important to consider several factors when planning orthodontic treatment for patients with diabetes. These patients may be at a higher risk of complications, including increased infections, delayed wound healing, and poor blood glucose control [79].

- **Key considerations are [7]:**

a. Control of Blood Glucose Levels: Uncontrolled diabetes can lead to delayed wound healing and increased susceptibility to infections, which can complicate orthodontic procedures. So, it is essential for patients with diabetes to have well-controlled blood glucose levels before and during orthodontic treatment.

b. Oral Health Assessment: Orthodontists should conduct a thorough oral health assessment before starting treatment and work in conjunction with the patient's dentist or periodontist to manage any oral health issues as these patients are more prone to dental problems.

c. Medication Management: Orthodontists should be aware of the medications a diabetic patient is taking and their potential side effect.

d. Dietary and Nutritional Considerations: Orthodontic patients with diabetes should be educated about the importance of maintaining a balanced diet to help manage their blood glucose levels. Dietary recommendations should consider their orthodontic appliances, such as braces or aligners.

e. Oral Hygiene: In diabetics orthodontic appliances can make oral hygiene more challenging, and the increased risk of periodontal disease so, maintaining good oral hygiene is critical for such orthodontic patients.

f. Complications and Healing: Patients with diabetes experience slower wound healing and are more susceptible to infections. Therefore, In cases of surgical orthodontics, additional precautions may be necessary.

g. Regular Monitoring: Patients with diabetes should have their blood glucose levels regularly monitored throughout the course of orthodontic treatment. Orthodontists should coordinate with the patient's endocrinologist or primary care physician to ensure proper blood sugar control.

h. Communication with the Diabetes Care Team: Orthodontists should maintain open communication with the patient's diabetes care team to ensure a holistic approach to the patient's health. This collaboration can help in managing any complications and optimizing treatment outcomes

- Orthodontic considerations can also be categorized as [80]:

a. Considerations before deciding orthodontic treatment

Ensure good oral hygiene and dental health (most potent)
Tight control of diabetes
Exclude periodontitis
Monitor blood glucose before going into active orthodontic treatment

b. Considerations during the process of orthodontic treatment

Apply light physiological forces
Antibiotic prophylaxis before: orthodontic band placement;
Separator placement;
Screw insertion
Antibiotic prophylaxis is not needed in: simple adjustment of appliances;
Simple replacement of appliances

c. Considerations to prevent or manage emergencies during the process (especially hypoglycemia):

Morning meal on day of orthodontic treatment and if symptoms of hypoglycemia occurred: IV dextrose; IM glucagon 1 mg.

CONCLUSION

Diabetes mellitus exerts a broad and clinically meaningful impact on oral tissues and dental treatment

outcomes through persistent hyperglycemia-driven inflammation, immune dysregulation, microvascular compromise, and altered bone and soft-tissue metabolism. These systemic changes translate into a recognizable set of oral and orofacial manifestations most notably periodontal breakdown, xerostomia with secondary caries risk, increased susceptibility to candidal and mucosal infections, neuropathic symptoms such as burning mouth, and compromised healing potential. As a result, DM should be approached in dentistry not as a background medical label, but as a condition that actively shapes diagnosis, risk assessment, treatment planning, and follow-up.

Across prosthodontic care, attention to salivary dysfunction, denture-related mucosal injury, fungal overgrowth, and progressive ridge resorption is essential to improve comfort and reduce complications. In endodontics, clinicians must recognize that poor glycemic control may be associated with higher prevalence of apical disease and slower periapical healing, while persistent endodontic infection may further aggravate systemic inflammation and worsen metabolic control. Orthodontic treatment similarly requires strict periodontal screening, lighter force systems, enhanced hygiene protocols, and coordinated monitoring to mitigate infection risk and delayed healing, particularly in patients with unstable blood glucose levels. Ultimately, the cornerstone of safe dental management in patients with DM is effective medical–dental collaboration, appointment timing that reduces hypoglycemic risk, careful infection control, and individualized planning based on the degree of glycemic control and the presence of systemic complications. With these principles, dental treatment can be delivered predictably, safely, and with improved long-term outcomes for diabetic patients.

REFERENCES

1. Diabetes Canada Clinical Practice Guidelines Expert Committee, Punthakee Z, Goldenberg R, Katz P. Definition, classification and diagnosis of diabetes, prediabetes and metabolic syndrome. Can J Diabet 2018;42 Suppl 1:S10-5.
2. Gregory GA, Robinson TIG, Linklater SE, Wang F, Colagiuri S, de Beaufort C, et al. Global incidence, prevalence, and mortality of type 1 diabetes in 2021 with projection to 2040: a modelling study. Lancet Diabetes Endocrinol 2022;10(10): 741–60.
3. Beisembinova N, Kosherbayeva L, Balmukhanova A, Hailey D, Tolganbayeva K, Seyduanova L. Diabetes and diabetic retinopathy in Kazakhstan from 2013-2018. Curr Pediatr Res 2021;25(7).
4. Majeed NA, Shiruhana SA, Maniam J, Eigenmann CA, Siyan A, Ogle GD. Incidence, prevalence and mortality of diabetes in children and adolescents aged under 20 years in the Republic of Maldives. J Paediatr Child Health 2020;56(5):746–50.
5. Kotwas A, Karakiewicz B, Zabielska P, *et al*. Epidemiological factors for type 2 diabetes mellitus: Evidence from the Global Burden of Disease. Arch Public Health 2021; 79: 110.
6. Lin X, Xu Y, Pan X, *et al*. Global, regional, and national burden and trend of diabetes in 195 countries and territories: An analysis from 1990 to 2025. Sci Rep 2020; 10: 14790.
7. Ogle GD, von Oettingen JE, Middlehurst AC, Hanas R, Orchard TJ. Levels of type 1 diabetes care in children and adolescents for countries at varying resource levels. Pediatr Diabetes 2019;20(1):93–8.
8. Ogle GD, Middlehurst AC, Silink M. The IDF Life for a Child Program Index of diabetes care for children and youth. Pediatr Diabetes 2016;17(5):374–84.
9. Sandy JL, Besançon S, Sidib´e AT, Minkailou M, Togo A, Ogle GD. Rapid increases in observed incidence and prevalence of Type 1 diabetes in children and youth in Mali, 2007-2016. Pediatr Diabetes 2021;22(4):545–51.
10. Sagna Y, Bagbila W, Sawadogo N, Savadogo PPC, Zoungrana L, S´er´e L, et al. Incidence, prevalence, and mortality of type 1 diabetes in children and youth in Burkina Faso 2013-2022. Diabetes Res Clin Pract 2024;207:111086.
11. Jasem D, Majaliwa ES, Ramaiya K, Najem S, Swai ABM, Ludvigsson J. Incidence, prevalence and clinical manifestations at onset of juvenile diabetes in Tanzania. Diabetes Res Clin Pract 2019;156:107817.
12. Ludvigsson J, Edna M, Ramaiya K. Type 1 diabetes in low and middle-income countries - Tanzania a streak of hope. Front Endocrinol (Lausanne) 2023;14: 1043370.
13. Ruiz PLD, Chen L, Morton JI, Salim A, Carstensen B, Gregg EW, et al. Mortality trends in type 1 diabetes: a multicountry analysis of six population-based cohorts. Diabetologia 2022;65(6):964–72.
14. Committee ADAPP. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes 2025. Diabetes Care 2024;48(Supplement_1):S27–S49.
15. Aguirre F, Brown A, Cho NH, et al. IDF diabetes atlas; 2013.4.
16. Wilson MH, Fitpatrick JJ, McArdle NS, Stassen LFA. Diabetes mellitus and its relevance to the practice of dentistry. *J Ir Dent Assoc*. 2010;56 (3):128–133.5
17. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care*. 2010;33(Suppl 1):S62–S69. doi:10.2337/dc10-S06211
18. Baynes HW. Classification, pathophysiology, diagnosis and management of diabetes mellitus. *J Diabetes Metab*. 2015;6(5):1–9.
19. Al Homsi MF, Lukic ML. An update on the pathogenesis of diabetes mellitus. *Dubai Diabetes Endocrinol J*. 1993;1:1–2.
20. Raju SM, Raju B. *Illustrated Medical Biochemistry*. 2nd ed. New Delhi, India: Jaypee Brothers Medical Publishers ltd; 2010.

21. Holt RI. Diagnosis, epidemiology and pathogenesis of diabetes mellitus: an update for psychiatrists. *Br J Psychiatry Suppl*. 2004;47:S55–S63. doi:10.1192/bjp.184.47.s55
22. Mealey BL, Ocampo GL. Diabetes mellitus and periodontal disease. *Periodontol 2000*. 2007;44:127–153. doi:10.1111/j.1600-0757.2006.00193.x
23. Mahler RJ, Adler ML. Clinical review 102: type 2 diabetes mellitus: update on diagnosis, pathophysiology, and treatment. *J Clin Endocrinol Metab*. 1999;84(4):1165–1171. doi:10.1210/jcem.84.4.5612
24. Sekikawa A, Tominaga M, Takahashi K, et al. Prevalence of diabetes and impaired glucose tolerance in Funagata area, Japan. *Diabetes Care*. 1993;16(4):570–574. doi:10.2337/diacare.16.4.570
25. Iwasaki H, Yagyu H, Shimano H. A Comprehensive Analysis of Diabetic Complications and Advances in Management Strategies. J Atheroscler Thromb, 2025; 32: 550-559.
26. ElSayed NA, Aleppo G, Aroda VR, et al : Addendum. 2. Classification and Diagnosis of Diabetes: Standards of Care in Diabetes-2023. Diabetes Care, 2023; 46: 1715
27. Brownlee M: The pathobiology of diabetic complications: a unifying mechanism. Diabetes, 2005; 54: 1615-1625
28. Holman RR, Bethel MA, Mentz RJ, et al: Effects of Once-Weekly Exenatide on Cardiovascular Outcomes in Type 2 Diabetes. N Engl J Med, 2017; 377: 1228-1239
29. Perkovic V, Jardine MJ, Neal B et al: Canagliflozin and Renal Outcomes in Type 2 Diabetes and Nephropathy. N Engl J Med, 2019; 380: 2295-2306
30. Alicic RZ, Rooney MT, Tuttle KR: Diabetic Kidney Disease: Challenges, Progress, and Possibilities. Clin J Am Soc Nephrol, 2017; 12: 2032-2045
31. Malek V, Suryavanshi SV, Sharma N et al: Potential of Renin-AngiotensinAldosterone System Modulations in Diabetic Kidney Disease: Old Players to New Hope! Rev Physiol Biochem Pharmacol, 2021; 179: 31-71
32. Heerspink HJL, Stefánsson BV, Correa-Rotter R et al. N Engl J Med, 2020; 383: 1436-1446
33. Herrington WG, Staplin N, Wanner C et al: Empagliflozin in Patients with Chronic Kidney Disease. N Engl J Med, 2023; 388: 117-127
34. Vinik AI, Nevoret ML, Casellini C, Parson H: Diabetic neuropathy. Endocrinol Metab Clin North Am, 2013; 42: 747-787
35. Riemma MA, Mele E, Donniacuo M et al: Glucagon-like peptide-1 receptor agonists and sodium-glucose cotransporter 2 inhibitors, anti-diabetic drugs in heart failure and cognitive impairment: potential mechanisms of the protective effects. Front Pharmacol, 2024; 15: 1422740
36. Davies MJ, Aroda VR, Collins BS et al: Management of Hyperglycemia in Type 2 Diabetes, 2022. A Consensus Report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes Care, 2022; 45: 2753-2786
37. Sarwar N, Gao P, Seshasai SRK et al: Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: a collaborative meta-analysis of 102 prospective studies. Lancet, 2010; 375: 2215-2222
38. Okamura T, Tsukamoto K, Arai H et al: Japan Atherosclerosis Society (JAS) Guidelines for Prevention of Atherosclerotic Cardiovascular Diseases 2022. J Atheroscler Thromb, 2024; 31: 641-853
39. American Diabetes Association. 2. Classification and diagnosis of diabetes: Standards of medical care in diabetes-2018. Diabetes Care 2018;41 Suppl 1:S13-27.
40. Mauri-Obradors E, Estrugo-Devesa A, Jané-Salas E, Viñas M, López-López J. Oral manifestations of Diabetes Mellitus. A systematic review. Med Oral Patol Oral Cir Bucal 2017;22:e586-94.
41. Katariya C. Diabetes mellitus and prosthodontic care. Int J Multidis Res Mod Edu,2017:3(1):294-296.
42. Rahman B. Prosthodontic Concerns in a Diabetic Patient. Int J Health Sci Res,2013:3(10):117-120.
43. Rathee M et al. Role of diabetes in the prosthodontic management of a completely edentulous patient. Int J Dent Res,2021:3(1):50-52.
44. Guggenheimer J, Moore PA, Rossie K et al. Insulindependent 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,2000:89(5):570-576.
45. Ship JA. Diabetes and oral health: an overview. J Am Dent Assoc,2003:134:410.
46. Kostić I, Najman S, Kostić M. Comparative review of gingival retraction agents. Acta Medica Medianae,2012:51(1).
47. Habib SS, Almas K. Management of Diabetic patients in dental practice. J Pak Dent Assoc. 2002; 11:101-106.
48. Joseph M A, Joseph S, Mathew N. Functional salivary reservoir in maxillary complete denture – technique redefined.Clin Case Rep,2016:4(12):1082-1087.
49. Samyukta, Abirami G. Residual Ridge Resorption in Complete Denture Wearers. J. Pharm. Sci. & Res, 2016:8(6):565-569.
50. Kaur J. Liquid-supported dentures: A delicate treatment modality for flabby ridges. Indian J Multidiscip Dent.2018 [cited 2020 Aug 26]:8:48-51.
51. Alsalleeh, Fahd. Diabetes and apical periodontitis: A comprehensive narrative overview of their interconnectedness. Saudi Endodontic Journal 15(2):p 120-127, May–Aug 2025. | DOI: 10.4103/sej.sej_247_24
52. Tibúrcio-Machado CS, Michelon C, Zanatta FB, Gomes MS, Marin JA, Bier CA. The global

prevalence of apical periodontitis:A systematic review and meta-analysis. Int Endod J 2021;54:712–35
53. Segura-Egea JJ, Cabanillas-Balsera D, Jiménez-Sánchez MC, Martín-González J. Endodontics and diabetes:Association versus causation. Int Endod J 2019;52:790–802.
54. Pérez-Losada FL, Estrugo-Devesa A, Castellanos-Cosano L, Segura-Egea JJ, López-López J, Velasco-Ortega E. Apical periodontitis and diabetes mellitus type 2:A systematic review and meta-analysis. J Clin Med 2020;9:540.
55. Pérez-Losada FL, López-López J, Martín-González J, Jané-Salas E, Segura-Egea JJ, Estrugo-Devesa A. Apical periodontitis and glycemic control in type 2 diabetic patients:Cross-sectional study. J Clin Exp Dent 2020;12:e964–71.
56. Davidović B, Krunić J, Mladenović I, Stojanović N, Hannig M, Vitkov L. Effects of apical periodontitis treatment on hyperglycaemia in diabetes:A prospective cohort study. Int Endod J 2024;57:1099–109.
57. Falk H, Hugoson A, Thorstensson H. Number of teeth, prevalence of caries and periapical lesions in insulin-dependent diabetics. Scand J Dent Res 1989;97:198–206.
58. Mesgarani A, Haghanifar S, Eshkevari N, Ehsani M, Khafri S, Nafarzade S, et al. Frequency of odontogenic periradicular lesions in diabetic patients. Caspian J Intern Med 2014;5:22–5.
59. Sánchez-Domínguez B, López-López J, Jané-Salas E, Castellanos-Cosano L, Velasco-Ortega E, Segura-Egea JJ. Glycated hemoglobin levels and prevalence of apical periodontitis in type 2 diabetic patients. J Endod 2015;41:601–6.
60. Arya S, Duhan J, Tewari S, Sangwan P, Ghalaut V, Aggarwal S. Healing of apical periodontitis after nonsurgical treatment in patients with type 2 diabetes. J Endod 2017;43:1623–7.
61. Cintra LT, Samuel RO, Azuma MM, Ribeiro CP, Narciso LG, de Lima VM, et al. Apical periodontitis and periodontal disease increase serum IL-17 levels in normoglycemic and diabetic rats. Clin Oral Investig 2014;18:2123–8.
62. Astolphi RD, Curbete MM, Colombo NH, Shirakashi DJ, Chiba FY, Prieto AK, et al. Periapical lesions decrease insulin signal and cause insulin resistance. J Endod 2013;39:648–52.
63. Pereira RF, de Oliveira da Mota MS, de Lima Coutinho Mattera MS, Tsosura TV, Chiba FY, Garbin CA, et al. Periapical lesions decrease Akt serine phosphorylation and plasma membrane GLUT4 content in rat skeletal muscle. Clin Oral Investig 2016;20:1625–30.
64. Tavares BS, Tsosura TV, Mattera MS, Santelli JO, Belardi BE, Chiba FY, et al. Effects of melatonin on insulin signaling and inflammatory pathways of rats with apical periodontitis. Int Endod J 2021;54:926–40.
65. Nagendrababu V, Segura-Egea JJ, Fouad AF, Pulikkotil SJ, Dummer PM. Association between diabetes and the outcome of root canal treatment in adults:An umbrella review. Int Endod J 2020;53:455–66.
66. Rudranaik S, Nayak M, Babshet M. Periapical healing outcome following single visit endodontic treatment in patients with type 2 diabetes mellitus. J Clin Exp Dent 2016;8:e498–504.
67. Cabanillas-Balsera D, Martín-González J, Montero-Miralles P, Sánchez-Domínguez B, Jiménez-Sánchez MC, Segura-Egea JJ. Association between diabetes and nonretention of root filled teeth:A systematic review and meta-analysis. Int Endod J 2019;52:297–306.
68. de Lourdes Ochoa-González F, González-Curiel IE, Cervantes-Villagrana AR, Fernández-Ruiz JC, Castañeda-Delgado JE. Innate immunity alterations in type 2 diabetes mellitus:Understanding infection susceptibility. Curr Mol Med 2021;21:318–31.
69. Segura-Egea JJ, Martín-González J, Castellanos-Cosano L. Endodontic medicine:Connections between apical periodontitis and systemic diseases. Int Endod J 2015;48:933–51.
70. Alshargawi WK, Alghamdi FM, Alziyad SA, Ramadan FL, Alamoudi AI, Alotni S, et al. Impact of diabetes mellitus on endodontic treatment outcomes. Int J Community Med Public Health 2024;11:4123-6.
71. Gupta A, Aggarwal V, Mehta N, Abraham D, Singh A. Diabetes mellitus and the healing of periapical lesions in root filled teeth: a systematic review and meta-analysis. Int Endodont J. 2020;53(11):1472-84.
72. Persoon I, Özok A. Definitions and epidemiology of endodontic infections. Curr Oral Health Rep. 2017;4:278-85.
73. Rosenberg CS. Wound healing in the patient with diabetes mellitus. Nurs Clin North Am. 1990;25(1):247-61.
74. Little JW, Falace DA, Miller CS, Rhodes NL. Dental Management of Medically Compromised Patient. 6th ed. St. Louis: Mosby; 2002.
75. Ingle JI, Bakland LK, Baumgartner JC. Ingle's Endodontics. 6th ed. Hamilton: BC Decker Inc.; 2008. p. 763.
76. Azodo CC. Current trends in the management of diabetes mellitus: The dentist' perspective. J Postgrad Med 2009;11:113-29.
77. Mc Kenna SJ. Dental management of patients with diabetes. Dent Clin North Am 2006;50:591-606.
78. Chakravarthy PV. Diabetes mellitus: An endodontic perspective. Eur J Gen Dent 2013;2:241-5.
79. Razia A.G. Khammissa et al. Orthdontic Treatment Considerations in Patients with Diabetes. Afr. J. Biomed. Res.2025; 28:1577-84.
80. Almadih A, Al-Zayer M, Dabel S, Alkhalaf A, Al Mayyad A, Bardisi W, Alshammari S, Alsihati Z. Orthodontic Treatment Consideration in Diabetic Patients. J Clin Med Res. 2018 Feb;10(2):77-81.