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Oral and Dental Considerations in Diabetes Mellitus: An Integrative Review

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INTRODUCTION:

The human body is remarkably designed with the ability to preserve a stable internal environment through highly organized regulatory mechanisms. This state of equilibrium, known as *homeostasis*, is maintained by intricate feedback systems and internal signals, including hormones and chemical pathways, that enable adaptation to external challenges such as fluctuations in temperature, pH, and blood glucose levels. When this delicate balance is disrupted, it often results in disorders or diseases.

Diabetes mellitus (DM) is one such multifaceted metabolic disorder, marked by disturbances in carbohydrate, protein, and lipid metabolism. Its defining feature is persistent hyperglycemia, which arises from inadequate insulin secretion, resistance of target tissues to insulin action, or a combination of both [1].

As of 2024, approximately 9.5 million people worldwide are living with type 1 diabetes (T1D), reflecting an increase of 1.1 million (13%) compared to the 8.4 million estimated in 2021 [2]. A significant concern is the high proportion of affected youth under the age of 20, particularly in low- and middle-income countries. If mortality rates among individuals with T1D matched those of the general population, the global prevalence in 2024 would be closer to 13.9 million—46% higher than current estimates.

The increase in global prevalence—from 8.4 million in 2021 to 9.5 million in 2024—represents a 13% rise, largely attributable to a combination of higher incidence rates (partly due to improved diagnostics), population growth, aging, and declining mortality. In lower-income regions, prevalence has grown even more sharply, rising 20% from 1.8 million in 2021 to 2.1 million in 2024. Certain countries demonstrate even steeper trends; for example, data from Kazakhstan [3] and the Maldives [4] reveal substantially greater annual increases in incidence compared to regional model estimates.

On a global scale, diabetes prevalence continues to rise steadily at approximately 2.5% annually [5]. By 2024, the number of people living with diabetes is projected to surpass 570 million, with diabetes-related deaths expected to reach 1.59 million [6]. Among children and adolescents, mortality remains especially high, primarily due to missed diagnoses,

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misdiagnoses, or lack of access to professional medical care—often culminating in fatal diabetic ketoacidosis [7–12].

Demographic patterns also shape the burden of T1D. For instance, India reports the highest prevalence of T1D among youth under 20, and together with China, ranks among the top 10 countries with the greatest numbers of T1D cases across all ages. This occurs despite both nations reporting lower incidence rates than European countries such as Finland and Sweden [13].

Although the peak onset of T1D typically occurs in childhood, the majority of individuals are diagnosed in adulthood. The global median age for T1D is 36 years, and around 1.1 million patients are aged 60 years or older. Consequently, initiatives to improve T1D management must target both younger and older populations alike [14].

Pathogenesis and Classification

Diabetes mellitus (DM) represents a heterogeneous group of disorders that share the common feature of hyperglycemia but differ in their underlying mechanisms. In 2010 and 2013, Wilson et al. and Aguirre et al. refined the classification into four major categories: **type 1 DM, type 2 DM, gestational DM, and diabetes due to other specific causes**. Type 1 DM is characterized by autoimmune destruction of pancreatic β-cells, which results in near-complete loss of insulin secretion. In contrast, type 2 DM is associated either with insufficient insulin release or impaired tissue responsiveness to the hormone. Gestational DM, a transient but clinically significant form, emerges primarily during the second and third trimesters of pregnancy. The fourth category encompasses atypical forms of diabetes, including monogenic syndromes, genetic defects, exocrine pancreatic disease, or drug- and chemical-induced diabetes such as that associated with cyclosporine use [15,16].

Earlier, the **Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (1998)** also proposed an etiological approach that distinguished between **primary** and **secondary** diabetes. Primary DM incorporates type 1 and type 2, with type 1 subdivided into immune-mediated (type A) and idiopathic (type B) variants, and type 2 linked to insulin resistance or defective insulin secretion. Secondary diabetes, on the other hand, arises from diverse causes, including pancreatic pathologies, endocrine disturbances, critical illness, gestational diabetes, genetic syndromes, autoimmune endocrinopathies, or the use of specific medications [17].

On a mechanistic level, diabetes arises through two principal pathways: **deficient insulin production** or **failure of insulin receptors to respond adequately**. In the latter case, tissues resist the action of insulin, prompting compensatory hyperinsulinemia that eventually proves insufficient. Environmental triggers, genetic predisposition, or a combination of both factors can disrupt insulin dynamics, thereby initiating a state of chronic insulin resistance [18].

Pathogenesis: Type 1 DM

The hallmark of type 1 DM is the autoimmune-mediated destruction of pancreatic β-cells. This immune assault is driven by macrophages along with CD4+ and CD8+ T lymphocytes,

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ultimately eliminating insulin-secreting cells [19]. Autoantibodies directed against insulin are often detectable before any therapeutic intervention, while glutamic acid decarboxylase (GAD) in β-cells is a frequent antigenic target of islet cell antibodies [20].

Loss of β-cell function disrupts metabolic balance, but abnormal α-cell activity compounds the dysfunction. Instead of suppressing glucagon in hyperglycemic states, α-cells release it in excess. This paradoxical rise in glucagon alongside high glucose levels intensifies hyperglycemia and undermines normal feedback regulation [21].

The consequences of insulin deficiency extend well beyond glycemic control. Accelerated lipolysis elevates circulating free fatty acids, which in turn blunt glucose uptake by peripheral tissues such as skeletal muscle [22]. Moreover, impaired insulin signaling alters gene expression essential for normal metabolic regulation. For example, glucokinase activity in the liver and GLUT-4 transporter expression in adipose tissue are both disrupted, leading to widespread disturbances in carbohydrate, lipid, and protein metabolism [22].

Pathogenesis: Type 2 DM

Type 2 diabetes mellitus (T2DM) does not arise from a single defect but from a **dual breakdown in insulin physiology**: impaired pancreatic insulin production on one side and tissue-level resistance to insulin on the other [17]. The relentless consequence of this dysfunction is impaired glucose tolerance, sustained hyperinsulinemia, and progressive β-cell exhaustion.

Within this spectrum lies **maturity-onset diabetes of the young (MODY)**—a monogenic form of T2DM. Although still under investigation, evidence points toward specific genetic defects, such as mutations in the glucokinase gene on chromosome 7p, as a central mechanism [23]. MODY is clinically defined as persistent hyperglycemia diagnosed before the age of 25, remaining manageable for more than five years without the need for insulin, and occurring in the absence of islet cell antibodies (ICA) [24]. This unique phenotype highlights the genetic complexity hidden beneath the broader category of T2DM.

Complications of Diabetes Mellitus

Diabetes is devastating not only because of chronic hyperglycemia but because of the **relentless complications it drives**. These can be broadly divided into two categories:

- **Microvascular complications**: retinopathy, nephropathy, and neuropathy, which result from structural and functional injury to capillaries and arterioles.
- **Macrovascular complications**: coronary artery disease (CAD), cerebrovascular disease, and peripheral arterial disease (PAD), all linked to accelerated atherosclerosis and endothelial dysfunction [25–28].

The destructive potential of these complications makes diabetes a systemic disease, not simply a metabolic disorder.

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1. **Diabetic Retinopathy (DR)**

Among microvascular complications, **diabetic retinopathy (DR)** stands out as the most prevalent and one of the most feared, being the leading cause of preventable blindness worldwide. It affects roughly **one-third of all diabetic patients**, cutting across both type 1 and type 2 DM [25].

Pathophysiological Mechanisms [25]:

- Persistent hyperglycemia triggers oxidative stress and cellular injury.
- Activation of the polyol pathway leads to accumulation of sorbitol, worsening vascular damage.
- Overexpression of vascular endothelial growth factor (VEGF) promotes pathological angiogenesis.

Clinical Presentation and Diagnosis [25]:

- *Non-proliferative diabetic retinopathy (NPDR)* manifests with microaneurysms, intraretinal hemorrhages, and cotton-wool spots, signaling early ischemic damage.
- *Proliferative diabetic retinopathy (PDR)* is defined by retinal neovascularization, vitreous hemorrhage, and tractional retinal detachment—often progressing to irreversible vision loss if untreated. Diagnosis relies on **fundoscopy and optical coherence tomography (OCT)**, with fluorescein angiography offering detailed evaluation of retinal perfusion defects.

Current and Emerging Therapies [25]:

- Intravitreal anti-VEGF agents remain the cornerstone of treatment.
- Corticosteroid injections serve as adjuncts in selected cases.
- Novel systemic agents such as SGLT2 inhibitors show promise in reducing disease progression.

2. **Diabetic Neuropathy (DN)**

Diabetic nephropathy (DN) stands as one of the **most devastating complications of diabetes**, responsible for the majority of chronic kidney disease (CKD) and a leading cause of end-stage renal disease (ESRD). It affects nearly **30–40% of diabetic patients**, presenting with persistent albuminuria, a progressive decline in glomerular filtration rate (GFR), and the eventual development of glomerulosclerosis [29].

Pathophysiological Mechanisms [30,31]:

- Glomerular hyperfiltration leading to increased intraglomerular pressure.
- Renal hypertrophy coupled with inflammatory cascades.
- Accumulation of advanced glycation end-products (AGEs), which accelerate tissue damage.

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- Dysregulation of the renin–angiotensin–aldosterone system (RAAS), amplifying renal injury.

Clinical Presentation and Diagnosis [25]:

- *Microalbuminuria* (30–300 mg/day): the earliest indicator of glomerular dysfunction.
- *Macroalbuminuria* (>300 mg/day): hallmark of advanced disease.
- A steady reduction in GFR, progressing toward renal failure.

Therapeutic Strategies [25,32,33]:

- **RAAS inhibition** (ACE inhibitors or ARBs) as the cornerstone of renoprotection.
- **SGLT2 inhibitors**, which demonstrate powerful nephroprotective effects beyond glycemic control.
- **GLP-1 receptor agonists**, offering both cardiovascular and renal benefits.

3. Diabetic Neuropathy

Diabetic neuropathy is another **high-burden complication**, affecting up to **half of all individuals with diabetes**. It manifests in multiple forms, reflecting widespread damage to both peripheral and autonomic nerves [25].

Clinical Manifestations:

- *Peripheral neuropathy*: The most common presentation, characterized by distal symmetric sensory loss, numbness, burning pain, and tingling that begin in the feet and progress proximally.
- *Autonomic neuropathy*: A more insidious form, leading to dysfunction of the autonomic nervous system. Patients may experience gastroparesis, orthostatic hypotension, bladder dysfunction, and even silent myocardial ischemia.

Treatment Approaches [25,34,35]:

- **Pain management**: First-line pharmacologic options include anticonvulsants such as gabapentin and pregabalin, and antidepressants such as duloxetine, which help relieve neuropathic pain.
- **Emerging therapies**: New evidence supports the role of GLP-1 receptor agonists and SGLT2 inhibitors in mitigating neuropathic damage, offering hope for disease modification rather than just symptomatic relief.

4. Cardiovascular Diseases (CVD)

Cardiovascular disease (CVD) remains the **most lethal consequence of diabetes**, encompassing **coronary artery disease, myocardial infarction, and heart failure**. Driven by insulin resistance, dyslipidemia, chronic low-grade inflammation, and endothelial dysfunction, diabetic patients face a **two- to four-fold higher risk** of developing CVD compared to non-diabetic populations [36,37].

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Pathophysiological Mechanisms [25]:

- Insulin resistance with associated lipid abnormalities.
- Persistent inflammation accelerating atherosclerotic plaque formation.
- Oxidative stress and endothelial dysfunction impairing vascular integrity.
- Advanced glycation end-products (AGEs) and activation of their receptors (RAGE), fueling vascular damage.

Therapeutic Approaches [25,38]:
The **2022 Guidelines for the Prevention of Atherosclerotic Cardiovascular Diseases**, published by the Japan Atherosclerosis Society, emphasized aggressive risk factor control to prevent cardiovascular outcomes [38]. Management strategies include:

- **Statin therapy** as the cornerstone of lipid regulation.
- **Antiplatelet agents** to reduce thrombotic events.
- **SGLT2 inhibitors and GLP-1 receptor agonists**, which extend benefits beyond glycemic control to cardiovascular protection.

5**. Cerebrovascular Disease [25]**

Diabetes dramatically heightens the risk of **stroke**, both ischemic and hemorrhagic, due to multifactorial vascular injury [25].

Pathophysiological Mechanisms:

- Hyperglycemia-induced endothelial dysfunction.
- Enhanced platelet activation and thrombogenesis.
- Increased vascular fragility, predisposing to hemorrhagic events.

Clinical Management:

- **Anticoagulant therapy** for secondary prevention.
- Rigorous **blood pressure control**, crucial for mitigating recurrent cerebrovascular events.

6. Peripheral Arterial Disease (PAD) [25]

Diabetes confers up to a **four-fold increase in the risk of peripheral arterial disease (PAD)**, a condition marked by systemic atherosclerosis and reduced perfusion of the extremities [25].

Pathophysiological Mechanisms:

- Compromise of microvascular networks.
- Chronic inflammatory activation contributing to arterial narrowing.

Power System Technology
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Diagnosis and Advanced Therapies:

- The **ankle-brachial index (ABI)** remains a simple, reliable, non-invasive tool for PAD detection.
- **Revascularization procedures** restore limb perfusion in advanced cases.
- **Emerging pharmacotherapies**, including SGLT2 inhibitors and novel anti-inflammatory agents, hold promise in improving outcomes.

Diagnosis

According to the **American Diabetes Association (ADA)**, the diagnosis of diabetes mellitus (DM) requires the presence of at least one of the following clinical or biochemical criteria [39]:

1. **Fasting plasma glucose ≥126 mg/dl (≥7 mmol/L)**
2. **Classic symptoms of diabetes**—polyuria, polyphagia, polydipsia, visual disturbances, recurrent infections such as thrush, unexplained fatigue, and weight loss—accompanied by unequivocal hyperglycemia (*random plasma glucose ≥200 mg/dl [11 mmol/L]*)
3. **Abnormal Oral Glucose Tolerance Test (OGTT):** Following an overnight fast, the patient ingests 75 g of glucose dissolved in 300 ml of water. Plasma glucose is measured at 30-minute intervals for 2 hours. A 2-hour plasma glucose level ≥200 mg/dl (11 mmol/L) confirms the diagnosis.
4. **Hemoglobin A1c (HbA1c) ≥6.5%:** HbA1c reflects the average plasma glucose concentration over 2–3 months, providing both diagnostic and long-term monitoring value.

Orofacial Manifestations of Diabetes

Diabetes exerts profound effects on oral tissues, with manifestations ranging across multiple sites [40]:

1. **Dento-alveolar structures:** Marked susceptibility to gingivitis, periodontitis, and progressive alveolar bone resorption.
2. **Salivary glands:** Enlargement (sialosis) and functional impairment leading to xerostomia.
3. **Oral mucosa:** Presence of lichenoid lesions, candidiasis, and tongue depapillation.
4. **Orofacial sensations:** Burning mouth syndrome, altered taste perception, and circumoral paraesthesia.
5. **Jaw bones and paranasal sinuses:** Elevated risk of osteomyelitis and invasive fungal infections such as mucormycosis.
6. **Cranial nerves:** Isolated mononeuropathies, especially affecting the oculomotor nerve.
7. **Halitosis:** Often associated with diabetic ketoacidosis.

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Dental Considerations in Prosthodontics

Prosthetic rehabilitation in diabetic patients requires heightened vigilance, as systemic metabolic changes directly influence oral tissue health.

a. Salivary hypofunction [41,42,43]: Diabetes mellitus induces both qualitative and quantitative alterations in the major salivary glands. Replacement of functional glandular parenchyma with adipose tissue reduces salivary output [42], causing xerostomia. Clinically, this translates into difficulties in denture retention, persistent mucosal irritation, and a burning sensation in the oral cavity [43].

b. Candida infections and denture stomatitis [44]: Diabetic patients are predisposed to oral candidiasis due to xerostomia, increased salivary glucose levels, and impaired immune regulation. Clinically, this may appear as median rhomboid glossitis, atrophic glossitis, denture stomatitis, or angular cheilitis. Such infections not only compromise oral comfort but also complicate prosthodontic outcomes.

Additional Prosthodontic Considerations in Diabetic Patients

c. Impaired Wound Healing [41,43]: Diabetic tissues demonstrate profound healing deficiencies due to microvascular angiopathy, reduced oxygen delivery, diminished collagen synthesis, and elevated collagenase activity. As a result, surgical wounds close slowly and are prone to complications. This mandates **strict glycemic control before undertaking pre-prosthetic surgeries or implant placement**, with elective procedures deferred until metabolic stability is achieved.

e. Burning Mouth Syndrome [45]: Patients frequently report burning sensations, dysgeusia, and dysphagia. These symptoms are attributed to altered salivary flow, diminished buffering capacity, and peripheral neuropathic changes. Burning mouth syndrome in diabetes is thus a multifactorial condition reflecting both systemic and local dysfunctions.

f. Increased Caries Susceptibility [41,43]: Diabetes shifts the oral microenvironment toward cariogenicity. Reduced salivary output, lowered pH, and enhanced pathogenic bacterial activity collectively accelerate dental caries and worsen periodontal breakdown. This underlines the importance of **preventive strategies and meticulous plaque control** in prosthodontic rehabilitation.

g. Residual Ridge Resorption [43]: Microvascular compromise impairs blood flow to alveolar bone and mucosa, intensifying residual ridge resorption. Diabetic patients therefore experience **accelerated ridge atrophy**, complicating denture fit and stability over time.

h. Antibiotic Prophylaxis [43]: For invasive dental interventions such as implant placement or pre-prosthetic surgery, **antibiotic coverage (Penicillin V or Erythromycin)** is strongly recommended to reduce infection risk, given the compromised host response in diabetics.

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i. **Anesthetic Considerations** [43,46]: Epinephrine exacerbates hyperglycemia by mobilizing glycogen stores into glucose. Thus, excessive epinephrine in local anesthesia or gingival retraction cords must be avoided. **Aluminum- or zinc chloride–based retraction cords** are preferred alternatives, minimizing systemic metabolic disturbance.

Prosthetic Therapy in Diabetic Patients

1. **Comprehensive Medical History** [41]: Every patient must undergo a thorough medical assessment prior to prosthodontic intervention. This includes documenting fasting blood glucose, HbA1c, prescribed medications, dosage, and timing. For patients over 45 years of age, **glucose screening at the initial appointment is mandatory**.

2. **Dietary Preparation** [41]: Patients should never present for prosthetic procedures in a fasting state. It must be ensured that they have consumed their usual breakfast and medications, as prosthetic appointments can be prolonged, increasing the risk of hypoglycemia.

3. **Appointment Scheduling** [41,47]: Morning appointments are optimal since **endogenous cortisol levels are highest early in the day**, providing a stabilizing effect on blood glucose. Patients should also be advised to seek medical clearance from their physician before undergoing significant dental procedures.

1. **Complete Denture** Diabetes-induced xerostomia compromises the resilience of oral mucosa, reducing denture retention. To address this, **salivary reservoir dentures** may be fabricated, allowing slow and continuous release of salivary substitutes. However, their effectiveness requires meticulous design and patient compliance for regular refilling, with high precision needed to ensure proper lid fit [48].

Frequent **relining and rebasing** is often necessary due to accelerated ridge resorption, though broader denture base coverage can mitigate this [43]. Additional strategies include reducing the number of artificial teeth, narrowing buccolingual tooth width, and optimizing occlusal tooth design [49]. For impression making, **mucostatic, minimal pressure, or neutral zone techniques** are recommended, as they accommodate altered soft tissue support in diabetics [43].

Liquid-supported dentures represent an advanced option, combining rigidity during function with adaptability to mucosa at rest. These prostheses enhance stability, preserve the residual ridge, and reduce mucosal soreness, offering long-term comfort and tolerance comparable to soft liners and tissue conditioners [50].

2. **Removable Partial Denture (RPD)** Every component of an RPD should be designed with tissue preservation in mind. Patients must receive **strict oral and prosthesis hygiene instructions** to prevent infection and reduce local complications [41].

Power System Technology
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3. Fixed Partial Denture (FPD)
For FPDs, **supragingival finish lines** are preferred to protect vulnerable soft tissues. A **chamfer margin** reduces stress on weakened teeth and adheres to Ante's law. Hygienic pontics are strongly advised due to their ease of cleaning. During tooth preparation, care must be taken to **avoid soft tissue trauma**, given impaired healing in diabetic patients [41].

4. Implants and Implant-Supported Dentures
Implant therapy is contraindicated in uncontrolled diabetes. In well-controlled patients, systemic glycemic levels, periodontal health, and bone quality must all be considered. Challenges include **poor wound healing, impaired osseointegration, and higher infection rates**. With careful patient selection, adequate antibiotic prophylaxis, and sustained glycemic control, survival rates of implants in diabetic patients can approach those in non-diabetic populations [41,42].

Endodontic Considerations in Diabetes

Apical Periodontitis (AP) is a global health concern influenced by systemic factors such as diabetes mellitus. Worldwide, AP prevalence reaches 52%, and pooled data show **T2DM patients have a 75% prevalence of at least one AP-affected tooth**, compared to 62% in non-diabetics [51–54].

a. Impact of Glycemic Control on AP

The relationship between glycemic control and AP remains debated. Some studies report **no significant differences in healing outcomes** between well- and poorly-controlled diabetic patients [55]. Others, however, document a **higher prevalence of AP and poorer healing** in long-term diabetics [56–59]. Poor glycemic control (HbA1c ≥6.5%) has been linked to increased AP prevalence [60], although conflicting data suggest otherwise [55]. Nevertheless, RCT outcomes in T2DM—particularly those with elevated HbA1c—are generally **less favorable, with more persistent lesions and reduced healing rates** [45,7,9,50]. Longer disease duration is also associated with greater AP prevalence and increased risk of root-filled teeth presenting with persistent periapical lesions [48,49].

b. Effect of AP on Diabetes Mellitus

The relationship is **bidirectional**. Persistent AP infection sustains systemic inflammation, undermining glycemic control and increasing insulin requirements [61]. Animal models confirm that AP can **impair insulin signaling** [62], driving insulin resistance and metabolic deterioration [63,64].

c. Diabetes and Endodontic Outcomes

Diabetes negatively influences endodontic prognosis. Patients show a **higher prevalence of residual periapical lesions** after RCT [65], slower healing of periapical tissues [60,66], and increased rates of root-filled tooth extraction [67].

The biological mechanisms underlying these outcomes include:

- **Impaired innate immunity**, reducing host defense against infection [68].

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- **Chronic hyperglycemia**, altering periapical tissue repair [56].
- **Advanced glycation end-products (AGEs)**, which disrupt normal tissue remodeling [69].

ENDODONTIC THERAPY IN DIABETICS

a. **Healing after endodontic treatment**

Hyperglycemia and the systemic complications of diabetescan 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].

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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].

I) 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

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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)

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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

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