SUNTEXT REVIEWS

SunText Review of Medical & Clinical Research
ISSN: 2766-4813

Open Access
Research Article
Volume 7:5

Orthodontic Treatment Considerations in Diabetic Patients: A Narrative Review

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Received date: 16 July 2026; **Accepted date:** 29 July 2026; **Published date:** 07 August 2026

Citation: Talaat MA (2026) Orthodontic Treatment Considerations in Diabetic Patients: A Narrative Review. SunText Rev Med Clin Res 7(5): 267.

DOI: https://doi.org/10.51737/2766-4813.2026.167

Abstract

Background: Diabetes mellitus (DM) is a globally prevalent metabolic disorder affecting approximately 589 million adults worldwide, with projections reaching 853 million by 2050. As the demographic of orthodontic patients expands to include more adults with systemic comorbidities, understanding the implications of diabetes on orthodontic treatment has become clinically imperative.

Objective: This narrative review aims to synthesize the current scientific evidence regarding the effects of diabetes mellitus on orthodontic treatment, including its impact on periodontal tissues, orthodontic tooth movement, root resorption, wound healing, and the stability of temporary anchorage devices, while providing evidence-based clinical recommendations for managing diabetic patients in orthodontic practice.

Methods: A comprehensive literature search was conducted across PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library using the keywords "diabetes mellitus," "orthodontic tooth movement," "periodontal tissues," "root resorption," "advanced glycation end products," and "temporary anchorage devices." Peer-reviewed articles published between 2000 and 2026 were included.

Results: The evidence indicates that uncontrolled diabetes significantly alters bone remodeling, accelerates periodontal breakdown, increases the rate of orthodontic tooth movement with concomitant root resorption, impairs soft tissue healing, and may compromise the stability of temporary anchorage devices. Well-controlled diabetic patients (HbA1c <7%) can undergo orthodontic treatment with acceptable outcomes when appropriate precautions are implemented.

Conclusion: Orthodontic treatment in diabetic patients is not contraindicated but requires a multidisciplinary approach, meticulous glycemic monitoring, modified biomechanical protocols, and enhanced periodontal maintenance. The orthodontist must integrate medical consultation into the treatment planning process to optimize outcomes and minimize complications.

Keywords: Diabetes mellitus; Orthodontic tooth movement; Periodontal disease; Root resorption; Advanced glycation end products; Bone remodeling; Temporary anchorage devices; Glycemic control

Introduction

Diabetes mellitus (DM) represents one of the most significant global health challenges of the 21st century. According to the International Diabetes Federation (IDF) Diabetes Atlas 11th Edition (2025), approximately 589 million adults aged 20–79 years are currently living with diabetes worldwide, representing a prevalence of 11.1% (1 in 9 adults). This figure is projected to rise to 853 million by 2050 [1,2]. The disease encompasses a group of chronic metabolic disorders characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both [3]. Two principal forms predominate: Type 1 DM (T1DM), an autoimmune condition resulting in absolute insulin deficiency, and Type 2 DM (T2DM), characterized by insulin resistance and progressive beta-cell dysfunction, accounting for approximately 90–95% of all diabetes cases [3]. The chronic hyperglycemic state leads to widespread microvascular and macrovascular complications affecting multiple organ systems, including the periodontium and alveolar bone [4,5]. Orthodontic treatment, once predominantly the domain of children and adolescents, has witnessed a substantial increase in adult patients seeking care [6]. This demographic shift inevitably increases the likelihood of encountering patients with diabetes in orthodontic

practice. Diabetes mellitus exerts profound effects on the biological process fundamental to orthodontic tooth movement, namely bone remodeling, periodontal ligament turnover, and inflammatory mediation [7,8]. Despite the clinical relevance of this intersection, a paucity of robust clinical guidelines exists to direct the orthodontic management of diabetic patients. This narrative review aims to comprehensively examine the pathobiological mechanisms through which diabetes mellitus influences orthodontic treatment, evaluate the available evidence from preclinical and clinical studies, and formulate evidence-based clinical recommendations for the safe and effective orthodontic management of patients with diabetes.

Pathophysiology of Diabetes Mellitus: Relevance to Oral Tissues

Hyperglycemia and Oxidative Stress

The hallmark of diabetes mellitus is chronic hyperglycemia, which initiates a cascade of pathological events including increased oxidative stress, formation of advanced glycation end products (AGEs), activation of protein kinase C, and upregulation of the polyol and hexosamine pathways [9]. These mechanisms converge to promote endothelial dysfunction, microvascular damage, and a chronic pro-inflammatory state that profoundly impacts the oral tissues [10].

Advanced Glycation End Products (AGEs)

AGEs are formed through non-enzymatic glycation of proteins and lipids under hyperglycemic conditions. Their accumulation in periodontal tissues has been extensively documented [11,12]. AGEs bind to the receptor for advanced glycation end products (RAGE) on cell surfaces, activating nuclear factor-kappa B (NF-κB) signaling and promoting the release of pro-inflammatory cytokines including tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6) [11,13]. systematically demonstrated that inflamed periodontal tissues serve as an endogenous source of AGEs in both diabetic and non-diabetic individuals, though concentrations are significantly elevated in the diabetic state [12].

Immune Dysregulation

Diabetes impairs both innate and adaptive immune responses. Polymorphonuclear leukocyte function is compromised, including chemotaxis, adherence, and phagocytosis [14]. This immunocompromised state renders diabetic patients more susceptible to periodontal infections and impairs the physiological inflammatory response essential for orthodontic tooth movement [4,5].

Effect of Diabetes on Periodontal Tissues

The Bidirectional Relationship

The relationship between diabetes mellitus and periodontal disease is bidirectional and well-established [15,16]. Diabetes increases the risk and severity of periodontitis by approximately threefold, while severe periodontitis adversely affects glycemic control by increasing systemic inflammatory mediators [15]. This bidirectional paradigm has critical implications for orthodontic treatment, as periodontal health constitutes a prerequisite for safe tooth movement.

Periodontal Breakdown During Orthodontic Tooth Movement

[17] in a systematic review of animal studies, demonstrated that uncontrolled diabetes mellitus significantly adversely affects periodontal tissues during orthodontic tooth movement (OTM). The findings consistently showed increased alveolar bone loss, elevated osteoclast counts, disorganized periodontal ligament fibers, and hyalinization in diabetic animals compared to normoglycemic controls. [18] further demonstrated altered collagen type I expression and increased matrix metalloproteinase-1 (MMP-1) activity in the periodontal ligament of diabetic rats during orthodontic force application, indicating accelerated and disorganized extracellular matrix turnover. Inflammatory Mediators in Gingival Crevicular Fluid [19] reported significantly elevated levels of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) and advanced glycation end products in gingival crevicular fluid (GCF) of diabetic patients undergoing fixed orthodontic treatment compared to normoglycemic controls. [20] in a systematic review and meta-analysis, confirmed that T2DM patients exhibit significantly higher concentrations of pro-inflammatory cytokines and AGEs in GCF during fixed orthodontic treatment, suggesting an exaggerated inflammatory response to mechanical loading.

Effect of Diabetes on Orthodontic Tooth Movement

Altered Bone Remodeling

Orthodontic tooth movement is fundamentally dependent on the coordinated remodeling of alveolar bone in response to mechanical forces. This process involves osteoclast-mediated resorption on the compression side and osteoblast-mediated deposition on the tension side of the periodontal ligament [7]. Diabetes disrupts this tightly regulated process through multiple mechanisms. [21] demonstrated in a mouse model that diabetic mice exhibited significantly greater orthodontic tooth movement and higher numbers of tartrate-resistant acid phosphatase (TRAP)-positive osteoclasts compared to normoglycemic controls. This increased tooth movement was associated with upregulation of receptor activator of nuclear factor kappa-B

ligand (RANKL), colony-stimulating factor 1 (CSF-1), chemokine ligands (CCL2, CCL5), and TNF-α [21]. Crucially, insulin treatment normalized both the rate of tooth movement and the molecular expression profiles, demonstrating the direct role of glycemic control [21]. Evidence from Systematic Reviews [22] in a systematic review with meta-analysis of preclinical studies, provided consolidated evidence that uncontrolled diabetes mellitus significantly increases the rate of orthodontic tooth movement. However, the quality of evidence was rated as low to moderate due to methodological limitations in the primary studies and significant heterogeneity. [23] elucidated a molecular mechanism through which diabetes suppresses mechanical loading-induced alveolar bone remodeling via impairment of the specificity protein 1/vascular endothelial growth factor (SP1/VEGF) axis. This finding provides a mechanistic explanation for the observed uncoupling of bone resorption and formation in the diabetic state during orthodontic treatment.

Clinical Implications

The acceleration of tooth movement in uncontrolled diabetes, while seemingly advantageous, is pathological in nature. It reflects excessive osteoclastic activity without compensatory bone formation, leading to net bone loss, compromised tooth stability, and increased susceptibility to root resorption [7,17,22]. [8] in a systematic review of clinical considerations, emphasized that the rate of tooth movement in diabetic patients cannot be considered equivalent to physiological movement in healthy individuals.

Root Resorption in Diabetic Patients

External apical root resorption (EARR) is a recognized sequela of orthodontic treatment. [24] investigated the effects of diabetes on orthodontic tooth movement and root resorption in a rat model and demonstrated that diabetic rats exhibited significantly greater root resorption compared to normoglycemic controls. Insulin treatment attenuated but did not completely eliminate this increased resorption [24]. The mechanism is attributed to enhanced osteoclast/odontoclast recruitment and activity driven by elevated RANKL expression and pro-inflammatory cytokine concentrations in the diabetic periodontium [21,24]. Clinically, this evidence suggests that diabetic patients undergoing orthodontic treatment are at elevated risk for EARR, necessitating radiographic monitoring at regular intervals and consideration of lighter force magnitudes [6,8].

Wound Healing and Soft Tissue Considerations

Impaired Mucosal Healing

Diabetes mellitus is well-established as a condition that impairs wound healing through mechanisms including impaired angiogenesis, reduced collagen synthesis, and chronic inflammation [25]. In the orthodontic context, this manifests as delayed healing of mucosal irritation from brackets, bands, and wires, as well as exaggerated tissue responses to removable and fixed appliances [6,26]. [26] reported a clinically significant case of extensive maxillary ulceration in a 9-year-old diabetic patient following placement of a rapid maxillary expander. The exaggerated inflammatory response and impaired healing led to palatal tissue necrosis that resolved only after glycemic control was established [26]. This case underscores the potential for severe soft tissue complications in undiagnosed or uncontrolled diabetic patients.

Susceptibility to Oral Infections

The immunocompromised state in diabetes, combined with mechanical irritation from orthodontic appliances, creates an environment conducive to oral candidiasis, angular cheilitis, and secondary bacterial infections [6,14]. Meticulous oral hygiene protocols and antimicrobial strategies are essential adjuncts to orthodontic treatment in this population.

Temporary Anchorage Devices in Diabetic Patients

Osseointegration and Stability Concerns

Temporary anchorage devices (TADs), including mini-screws and mini-plates, have revolutionized orthodontic biomechanics by providing absolute anchorage. However, their success depends on adequate bone-implant interface stability, which may be compromised in the diabetic state [27,28]. [28] evaluated the stability of surface-treated mini-implants in diabetic rabbits and found that untreated mini-implants exhibited reduced stability in diabetic animals compared to normoglycemic controls. Surface modification with resorbable blasting media improved osseointegration, suggesting that surface treatment strategies may partially compensate for diabetes-related impairment [28].

Clinical Considerations for TAD Use

While the overall success rate of orthodontic mini-screws ranges from 83% to 95% in the general population [29,30], systemic conditions including diabetes have been identified as potential risk factors for failure [27]. In diabetic patients, clinicians should consider:

- Ensuring optimal glycemic control (HbA1c <7%) prior to TAD placement
- Selection of sites with adequate cortical bone thickness
- Surface-treated mini-screws when available
- Extended monitoring protocols for early detection of mobility
- Lower force application during the initial healing phase [27,28]

Classification-Based Clinical Approach

Well-Controlled Diabetes (HbA1c <7%)

Patients with well-controlled diabetes can generally undergo orthodontic treatment with outcomes approaching those of non-diabetic patients [6,8]. [4] emphasized that the level of glycemic control, rather than the diagnosis of diabetes per se, determines the feasibility and safety of orthodontic treatment. Key considerations include:

- Standard orthodontic treatment protocols may be employed with modifications
- Enhanced periodontal monitoring at 4–6-week intervals
- Coordination with the patient's endocrinologist/diabetologist
- Patient education regarding the importance of maintaining glycemic control throughout treatment

Moderately Controlled Diabetes (HbA1c 7–8.5%)

Patients with moderate glycemic control require heightened vigilance [6,8]

- Lighter orthodontic forces to reduce the risk of excessive bone resorption
- Extended appointment intervals to allow for slower tissue remodeling
- Aggressive periodontal maintenance including subgingival debridement
- Periodic reassessment of glycemic status with treatment modification as indicated
- Consideration of removable appliances over fixed to facilitate oral hygiene

Poorly Controlled Diabetes (HbA1c >8.5%)

Orthodontic treatment should generally be deferred until glycemic control is improved [4,6,8]:

- Active orthodontic treatment is contraindicated due to unacceptable risk of periodontal destruction, excessive root resorption, and impaired healing
- Referral to endocrinology for optimization of diabetic management
- Limited interventions for acute orthodontic needs (pain relief, appliance repair) may be performed
- Treatment may commence once HbA1c is reduced to acceptable levels (<7–8%)

Clinical Recommendations and Management Protocol

Pre-Treatment Assessment

- **Comprehensive medical history** including diabetes type, duration, medications, and history of complications (nephropathy, retinopathy, neuropathy, cardiovascular disease)
- **Glycemic assessment** including recent HbA1c values (within 3 months) and fasting blood glucose
- **Comprehensive periodontal examination** including probing depths, clinical attachment levels, bleeding on probing, and radiographic assessment of alveolar bone
- **Medical consultation** with the patient's physician/endocrinologist to confirm fitness for elective dental procedures
- **Baseline radiographs** (panoramic and periapical) for future comparison regarding root resorption monitoring

During Treatment

- **Force magnitude**: Apply lighter forces (50–70% of conventional force levels) to minimize excessive bone resorption and root resorption risk [8,24]
- **Appointment intervals**: Extend to 6–8 weeks to accommodate potentially slower bone formation on the tension side [6]
- **Periodontal maintenance**: Professional prophylaxis every 3–4 months; reinforcement of oral hygiene at every appointment
- **Radiographic monitoring**: Periapical radiographs at 6-month intervals to assess root resorption and alveolar bone status
- **Glycemic monitoring:** Request updated HbA1c values every 3–6 months; communicate with the medical team regarding any deterioration
- **Appointment timing:** Schedule appointments in the morning when cortisol levels are highest and avoid treatment during periods of hypoglycemia or hyperglycemia [4]
- **Emergency preparedness:** Maintain glucose supplements in the clinic for hypoglycemic episodes

Post-Treatment Considerations

- Extended retention: Longer retention periods are advisable due to potentially altered bone remodeling and reduced stability of tooth position [6,8]
- Continued periodontal monitoring: Post-treatment periodontal surveillance for at least 2 years
- Fixed retainers preferred: Bonded retainers may provide more reliable stability than removable alternatives in this population
- Patient counseling: Emphasize the lifelong relationship between glycemic control and periodontal health

Emerging Therapies and Future Directions

Recent research has explored adjunctive therapies to optimize orthodontic outcomes in diabetic patients:

- **Insulin therapy:** Animal studies consistently demonstrate that insulin administration normalizes the rate of orthodontic tooth movement and reduces pathological bone resorption [21,24]
- **Low-level laser therapy (LLLT):** Has shown promise in promoting bone regeneration and reducing inflammation in diabetic animal models undergoing orthodontic treatment [8]
- **Bisphosphonates:** Local application has been investigated to counteract excessive osteoclastic activity, though clinical evidence remains limited [7]
- **Anti-RAGE therapies**: Targeting the AGE-RAGE axis represents a novel therapeutic avenue to mitigate diabetes-induced periodontal destruction [11,13]
- **Bioactive surface coatings on TADs**: Surface modifications including hydroxyapatite and resorbable blasting media have shown improved osseointegration in diabetic models [28]

Limitations of Current Evidence

Several limitations in the existing literature merit acknowledgment:

- The majority of evidence derives from animal models (predominantly streptozotocin-induced T1DM in rodents), which may not fully recapitulate the human diabetic condition, particularly T2DM [17,22].
- Human clinical studies are predominantly observational with small sample sizes and short follow-up periods [8].
- Heterogeneity in diabetes models, orthodontic force protocols, and outcome measures limits direct comparability across studies [22].
- The distinction between the effects of T1DM and T2DM on orthodontic treatment outcomes remains insufficiently characterized [4,8].
- Randomized controlled trials examining orthodontic outcomes specifically in diabetic patients are virtually absent from the literature

Conclusion

Diabetes mellitus exerts significant and multifaceted effects on the biological processes underpinning orthodontic treatment. Through mechanisms involving AGE accumulation, pro-inflammatory cytokine upregulation, altered RANKL/OPG signaling, and impaired angiogenesis, the diabetic state disrupts the delicate balance of bone remodeling essential for physiological tooth movement. The clinical consequences include accelerated but pathological tooth movement, increased risk of root resorption, exaggerated periodontal breakdown, impaired wound healing, and potentially compromised anchorage device stability. However, diabetes mellitus is not an absolute contraindication to orthodontic treatment. With appropriate glycemic control (HbA1c <7%), multidisciplinary collaboration, modified biomechanical protocols, and enhanced monitoring, successful orthodontic outcomes can be achieved. The orthodontist bears the responsibility of comprehensive pre-treatment assessment, individualized treatment planning, and ongoing communication with the patient's medical team. Future research should prioritize well-designed prospective clinical studies and randomized controlled trials to establish definitive clinical protocols. The development of adjunctive therapies targeting the molecular mechanisms of diabetes-induced periodontal pathology holds promise for improving orthodontic outcomes in this growing patient population.

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