



GLP-1 Inhibitor (Tirzepatide / Mounjaro® 2.5 mg) and Oral Health: Dental Considerations for Clinical Practice

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ABSTRACT

The increasing use of glucagon-like peptide-1 (GLP-1)-based therapies has introduced important interdisciplinary considerations for dental professionals. Tirzepatide (Mounjaro®), a dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptor agonist, is commonly initiated at 2.5 mg weekly to improve tolerability prior to dose escalation (5–7,14). Although primarily prescribed for type 2 diabetes mellitus (T2DM) and metabolic control, its systemic effects—including improved glycemic regulation, delayed gastric emptying, appetite modulation, and gastrointestinal symptoms—may influence oral health outcomes and dental procedural planning (7–9). This narrative review synthesizes current pharmacologic and oral-systemic literature to evaluate periodontal implications, xerostomia and caries risk, dental erosion, sedation safety, and the role of dental hygienists in patient management. While metabolic stabilization may positively impact periodontal inflammation and wound healing (1–4), gastrointestinal symptoms and hydration changes may increase erosion and caries susceptibility (7,8,10–13). Individualized risk assessment and interdisciplinary communication are essential. Further prospective research is needed to clarify direct oral outcomes associated with GLP-1-based therapies.

Keywords: GLP-1 inhibitor, tirzepatide, Mounjaro®, oral health, periodontal disease, dental erosion, sedation dentistry, dental hygiene, oral-systemic health

I. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is strongly associated with periodontal inflammation, impaired wound healing, and increased oral disease burden (1–4). Advances in metabolic pharmacotherapy have significantly improved glycemic management, particularly through GLP-1 receptor agonists (9,14).

Tirzepatide, developed by Eli Lilly and Company and approved by the U.S. Food and Drug Administration, is a dual GIP/GLP-1 receptor

agonist administered once weekly (6,14). The 2.5 mg dose serves as the initiation phase prior to escalation (7).

As GLP-1 inhibitor (Tirzepatide / Mounjaro® 2.5 mg) therapy becomes increasingly common, dental professionals must understand its systemic mechanisms and their indirect oral implications.

II. METHODS

A narrative review was conducted using peer-reviewed literature (2005–2026) from PubMed/MEDLINE and Scopus. Search terms included:

- “GLP-1 receptor agonists”
- “tirzepatide”
- “diabetes and periodontal disease”
- “gastric reflux and dental erosion”
- “GLP-1 and anesthesia”
- “xerostomia and caries risk”

Pharmacologic data (5–9,14), diabetes-periodontal literature (1–4), gastrointestinal physiology (8,9), and oral pathophysiology sources (10–13) were thematically synthesized to provide clinically relevant recommendations.

III. NARRATIVE SYNTHESIS: CLINICAL CONSIDERATIONS

3.1 Glycemic Control and Periodontal Health

The bidirectional relationship between diabetes and periodontal disease is well established (1–4). Hyperglycemia increases advanced glycation end products (AGEs), oxidative stress, and inflammatory cytokine production, contributing to periodontal tissue breakdown (2,3).

Clinical trials demonstrate that tirzepatide significantly reduces HbA1c and improves metabolic stability (5,6).

Improved glycemic control may contribute to:

- Reduced gingival inflammation (1–4)
- Decreased bleeding on probing (1–4)
- Improved response to scaling and root planing (1–4)



- Enhanced post-surgical healing (1–4)
- Greater long-term periodontal stability (1–4)

Although direct periodontal trials in tirzepatide users remain limited, metabolic stabilization is expected to positively influence inflammatory burden (1–4).

3.2 Gastrointestinal Effects and Dental Erosion

Tirzepatide commonly produces nausea and occasional vomiting, particularly during initiation (7,8). GLP-1 receptor agonists delay gastric emptying as part of their mechanism of action (8,9). Gastric acid exposure is a well-established cause of dental erosion (10,11).

Repeated exposure may lead to:

- Enamel demineralization (10,11)
- Increased dentin sensitivity (10,11)
- Palatal erosion of maxillary anterior teeth (10,11)

Preventive strategies include:

- Rinsing with water or sodium bicarbonate after vomiting (10,11)
- Avoiding brushing for at least 30 minutes post-emesis (10,11)
- Topical fluoride application (10–13)
- Use of remineralization agents (10–13)

3.3 Xerostomia, Hydration, and Caries Risk

Although xerostomia is not a primary labeled adverse reaction, reduced appetite and decreased fluid intake may contribute to mild dehydration and lower salivary flow (7,12). Saliva plays a critical role in:

- Acid buffering (12)
- Enamel remineralization (12)
- Biofilm control (12)
- Reduced salivary flow increases caries risk (12,13).

Preventive management may include:

- Hydration counseling (12)
- Xylitol-based products (12,13)
- Fluoride varnish application (12,13)
- Individualized caries risk assessment (12,13)
- Modified recall intervals (12,13)

3.4 Sedation and Delayed Gastric Emptying

GLP-1 inhibitors delay gastric emptying as part of their mechanism of action (8,9). This effect may result in residual gastric contents despite standard fasting protocols (8).

During moderate or deep sedation:

- Protective airway reflexes are diminished
- Regurgitation risk increases
- Aspiration becomes a concern (8,9)

Patients at higher sedation risk may include those who recently initiated tirzepatide (2.5 mg phase), recently escalated dose, report active nausea or vomiting, or have underlying diabetic gastroparesis (7,8).

Routine dental procedures without sedation are generally unaffected. However, for IV sedation or general anesthesia, clinicians should assess active GI symptoms, document last injection timing, and consider postponing elective sedation in symptomatic patients (8,9).

IV. THE ROLE OF THE DENTAL HYGIENIST

Dental hygienists play a central role in identifying and managing patients receiving GLP-1 therapy. Hygienists should monitor for early enamel erosion (10,11), salivary changes (12), increased caries activity (12,13), and gingival inflammation (1–4). Preventive interventions such as fluoride varnish, remineralization products, dietary counseling, and salivary stimulation are essential components of care (10–13).

Clear documentation and communication with the dentist are essential, particularly before sedation-based procedures.

V. DISCUSSION

GLP-1 inhibitor (Tirzepatide / Mounjaro® 2.5 mg) therapy represents an important intersection between metabolic medicine and dentistry. Improved glycemic control may positively influence periodontal inflammation and wound healing, consistent with established diabetes literature (1–4,5,6). However, gastrointestinal side effects and hydration changes introduce secondary risks for dental erosion and caries (7,8,10–13). Sedation safety considerations further emphasize the need for comprehensive medical review and risk-based assessment (8,9).

Direct prospective research evaluating oral outcomes in GLP-1 users remains limited.

VI. CONCLUSION

GLP-1 inhibitor (Tirzepatide / Mounjaro® 2.5 mg) therapy has meaningful indirect implications for oral health and dental procedural safety. While metabolic stabilization may support periodontal improvement (1–4), gastrointestinal effects and delayed gastric emptying require preventive awareness and individualized sedation assessment (8,9). Dental professionals—and particularly dental hygienists—play a critical role

