

GLP-1 receptor agonists and their dental implications: A practical guide for dental practitioners (Review)

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Abstract. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have revolutionized the management of type 2 diabetes mellitus and obesity. Their rapidly increasing use has critical implications for dental practice, yet guidance for clinicians remains limited. The aim of the present review was to provide an evidence-based narrative summary of the oral and dental implications of GLP-1RA therapy and to provide practical chair-side recommendations for dental practitioners. For this purpose, a narrative literature review was performed to synthesize current evidence concerning the oral, periodontal and perioperative implications of GLP-1RAs in dental practice. The PubMed/MEDLINE, Scopus, Web of Science and Google Scholar databases were searched for publications from January, 2015 through June, 2026. As this was a narrative review, studies were selected according to their clinical relevance and contribution to the mechanistic and practical questions addressed rather than according to a predefined systematic review protocol. No PRISMA flow diagram or quantitative study-selection estimates were therefore applied. Particular emphasis was placed on distinguishing established clinical evidence from observational associations, preclinical findings and mechanistic hypotheses. Current evidence suggests that GLP-1RAs may contribute to xerostomia, dysgeusia, halitosis and dental erosion, largely secondary to adverse gastrointestinal effects and salivary alterations. Conversely, improved glycemic control may favor periodontal health and wound healing. Delayed gastric emptying has critical implications for dental procedures requiring sedation or general anesthesia and necessitates careful preoperative assessment and interdisciplinary communication. On the whole, as the

number of prescriptions of GLP-1RAs continues to expand, dental practitioners should routinely identify affected patients, recognize potential oral manifestations, implement preventive strategies and consider perioperative implications to optimize patient safety and clinical outcomes.

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1. Introduction

Glucagon-like peptide (GLP)-1 receptor agonists (GLP-1RAs) represent among the most critical pharmacological advances in the treatment of type 2 diabetes mellitus (T2DM) and obesity in the past decade. Although these drugs were initially designed as anti-hyperglycemic agents, they are increasingly being prescribed due to their prominent effects on weight loss, cardiovascular risk reduction and metabolic control (1,2). The availability of long-acting agents, such as semaglutide and the dual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 receptor agonist, tirzepatide, has led to unprecedented worldwide demand, beyond endocrinology, in primary care, obesity medicine and aesthetic practice (3,4).

As the use of GLP-1RAs is increasingly prescribed, dental practitioners will come across a greater number of patients on these medications. In the UK and numerous other countries, prescriptions for semaglutide and tirzepatide have markedly increased in recent years. This is a reflection of the escalating prevalence of obesity, as well as wider therapeutic indications (5). Thus, knowledge of GLP-1RA treatment is currently

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applicable to all members of the dental team. Crucially, a number of patients may not have received information regarding weight loss medications unless specifically requested, particularly if the treatment was obtained through private clinics or online providers. Therefore, the complete medical history of the patient should include direct questioning about injectable or oral anti-obesity medications.

From a dental perspective, GLP-1RA therapy may have both potentially beneficial and adverse implications. Improved metabolic control may support periodontal health and tissue repair, whereas treatment-related gastrointestinal and salivary changes may adversely affect susceptible patients (6-9). However, direct dental evidence remains limited, and the clinical significance of these effects has not yet been fully established. Perioperative considerations are particularly relevant when deep sedation or general anaesthesia is planned as GLP-1RA-related delayed gastric emptying may influence the risk of aspiration (10,11).

The aim of the narrative present review was therefore to provide a practical, evidence-based framework for assessing and managing patients receiving GLP-1RAs in dental practice. For the purpose of the present review, the PubMed/MEDLINE, Scopus, Web of Science and Google Scholar databases were searched for publications from January, 2015 through June, 2026 using combinations of Medical Subject Headings and free-text terms related to 'GLP-1 receptor agonists', 'semaglutide', 'tirzepatide', 'exenatide', 'liraglutide', 'dulaglutide', 'oral health', 'xerostomia', 'hyposalivation', 'dysgeusia', 'dental erosion', 'periodontitis', 'peri-implantitis', 'wound healing', 'sedation', 'gastric emptying', and 'perioperative management'. Reference lists of relevant publications and clinical guidance documents were additionally screened to identify pertinent studies. Eligible evidence included clinical studies, observational studies, systematic and narrative reviews, meta-analyses, clinical practice guidelines, pharmacovigilance studies, experimental studies and relevant case reports published in the English language.

2. Definition of GLP-1 receptor agonists

GLP-1 is an endogenous incretin hormone produced predominantly by enteroendocrine L-cells in the distal small intestine and colon. Released post-prandially, it contributes to glucose homeostasis by enhancing glucose-dependent insulin secretion, suppressing glucagon release, delaying gastric emptying and promoting satiety (12,13). As native GLP-1 is rapidly degraded by dipeptidyl peptidase-4 (DPP-4), its short half-life limits therapeutic use (14). DPP-4-resistant GLP-1 receptor agonists (GLP-1RAs) provide prolonged biological activity and include exenatide, liraglutide, dulaglutide, lixisenatide and semaglutide; tirzepatide is a dual GIP/GLP-1RA (2,15). These agents are available in daily or weekly injectable formulations, with oral semaglutide also available (16). Their principal effects include glucose-dependent insulin secretion, the suppression of glucagon, reduced hepatic glucose output, delayed gastric emptying and appetite suppression, resulting in improved glycaemic control and weight loss (13,17). Cardiovascular outcome trials have additionally demonstrated reductions in major adverse cardiovascular events in high-risk populations (18).

GLP-1-based therapies are approved primarily for patients with T2DM and to those with obesity or who are overweight with relevant comorbidities (3,19), with expanding investigation across cardiovascular, renal, hepatic and neurodegenerative conditions (20). Consequently, dental practitioners are increasingly likely to encounter patients receiving these therapies.

3. Oral and dental aspects in glucagon-like peptide-1 receptor agonist therapy

The use of GLP-1RAs is associated with a variety of oral, dental and clinical aspects. A schematic representation of these is presented in Fig. 1 and these are also briefly described below:

Changes in saliva and dry mouth. Xerostomia is a commonly reported oral complaint in patients receiving GLP-1RAs. Its mechanism is likely multifactorial, involving reduced fluid intake associated with appetite suppression, gastrointestinal fluid loss, altered autonomic regulation and the concomitant use of xerogenic medications (21). Reduced salivary flow may increase the risk of developing dental caries, mucosal discomfort, candidal infection, dysphagia and impaired denture retention (22). Clinicians should therefore enquire about oral dryness and related functional symptoms during routine examinations. The mechanisms underlying xerostomia in patients receiving GLP-1RAs remain incompletely defined and are likely multifactorial. An indirect mechanism is clinically plausible as GLP-1RAs commonly cause nausea, vomiting, reduced appetite and gastrointestinal discomfort, which may reduce fluid intake and contribute to dehydration and diminished salivary flow. The concomitant use of other xerogenic medications in patients with diabetes, obesity and cardiovascular comorbidities may further contribute to subjective oral dryness. A potential direct effect on salivary-gland function has also been proposed. Experimental evidence has revealed GLP-1 receptor expression in major salivary glands, providing biological plausibility for receptor-mediated modulation of glandular function. Salivary secretion is regulated by coordinated autonomic signaling involving intracellular calcium and cyclic AMP pathways; therefore, modulation of these pathways by sustained GLP-1 receptor stimulation could theoretically influence secretory activity (6,7). Recent mechanistic research has proposed that prolonged GLP-1 receptor activation may involve cAMP-dependent signaling, β -arrestin recruitment, receptor internalization and altered cellular responsiveness (6). However, these mechanisms remain hypothesis-generating, and direct human evidence demonstrating GLP-1RA-induced salivary-gland dysfunction is currently lacking. Clinically, this distinction is critical as xerostomia should not automatically be attributed to a direct pharmacological effect of the GLP-1RA. Hydration status, gastrointestinal symptoms, concomitant medications and underlying systemic disease should also be considered (7,8). A 2023 case series reported hyposalivation in three patients receiving semaglutide, providing important clinical information but not definitive evidence of causality (7).

Management should include preventive strategies, including the reinforcement of meticulous plaque control, frequent hydration, the use of sugar-free chewing gum, the use of salivary substitutes and the use of topical fluoride, where

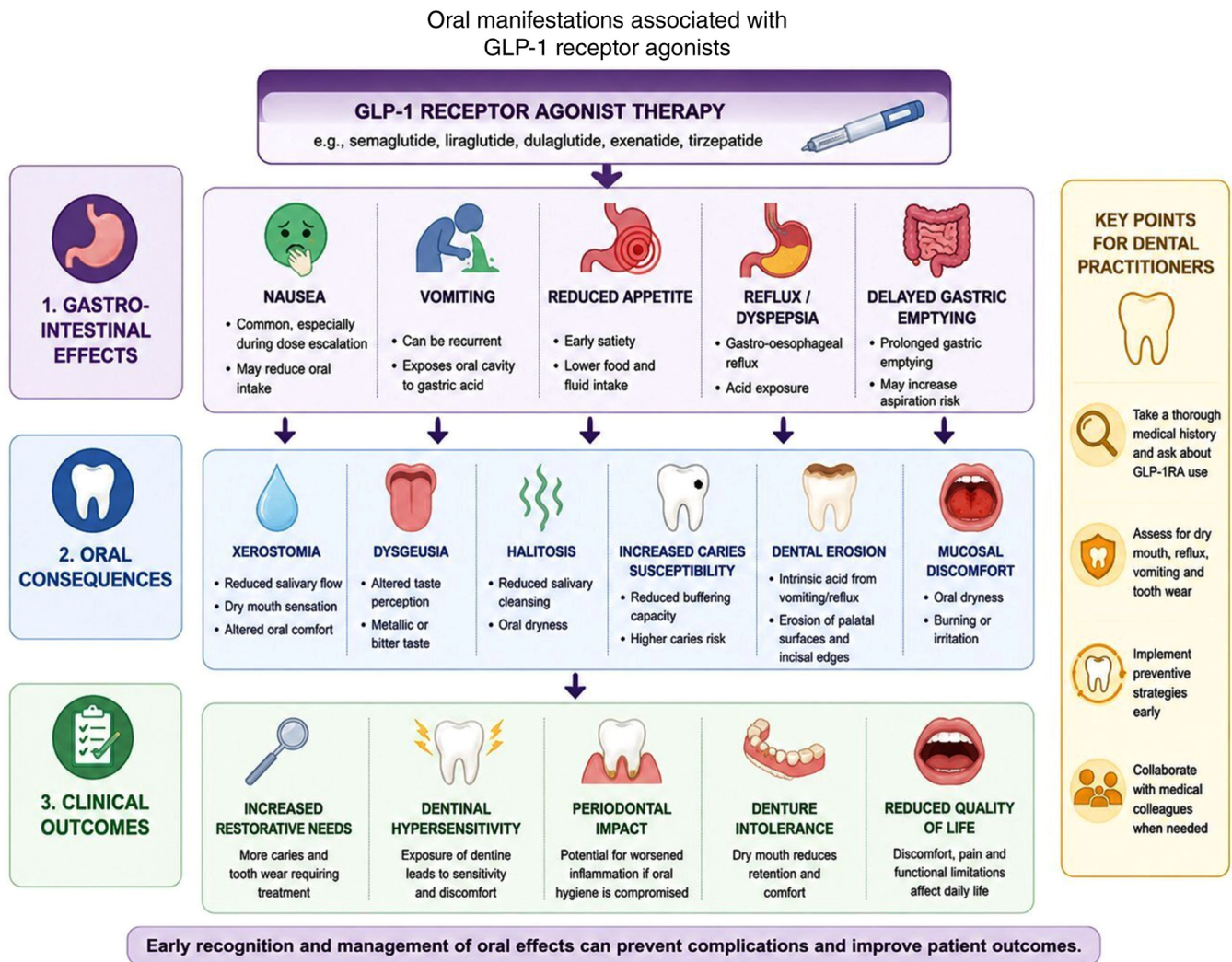


Figure 1. Schematic representation of the oral manifestations associated with GLP-1 receptor agonists (2,7,9,13,15,34,37-39).

indicated (9). Patients with persistent symptoms may require more frequent recall and better caries prevention.

Although GLP-1 receptor agonists share broadly similar pharmacological effects, their gastrointestinal adverse effects are not identical. A recent Bayesian network meta-analysis of randomized trials demonstrated differences among individual agents in nausea, vomiting, diarrhea and other gastrointestinal adverse events, with some agents exhibiting greater gastrointestinal intolerance than others (10). Real-world comparative data have similarly suggested differences in gastrointestinal adverse event patterns between individual GLP-1RAs (10,11). However, these findings cannot be directly extrapolated to adverse dental effects. Direct comparative evidence for xerostomia, dysgeusia, dental erosion or other oral manifestations remains very limited. Semaglutide-associated hyposalivation has been described in a small case series (7); however, causality and the underlying mechanism remain uncertain. Therefore, clinicians should avoid assuming that one GLP-1RA has consistently greater oral toxicity than another does. Dental assessments should instead consider the specific agent, dose, treatment duration, recent dose escalation, gastrointestinal symptoms, hydration status and individual oral findings (7,10,12).

Gastrointestinal effects and their oral consequences. Gastrointestinal adverse effects are the most common toxicity associated with the use of GLP-1RAs, particularly nausea and, in susceptible patients, vomiting during treatment initiation or dose escalation (2,11,13,14). Recurrent vomiting or gastro-oesophageal reflux may expose oral tissues to gastric acid, increasing the risk of dental erosion, dentin hypersensitivity and restorative deterioration (15). Erosive tooth wear typically affects the palatal surfaces of the maxillary anterior teeth and occlusal surfaces of posterior teeth and may progress insidiously; therefore, patients with persistent nausea, retching, vomiting or reflux should be assessed for erosive changes (15).

GLP-1RA-associated delayed gastric emptying may contribute to reflux symptoms in susceptible individuals (16,17). Reflux may also produce unpleasant taste, halitosis or oral burning. Management should focus on preventive counselling, dietary and reflux-related advice, monitoring for erosive tooth wear, and medical referral when symptoms are severe or persistent. Following vomiting, patients should avoid immediate toothbrushing as acid-softened enamel is more vulnerable to abrasion; rinsing with water, milk or a bicarbonate-containing rinse followed by delayed brushing with fluoridated toothpaste is recommended. Topical fluoride

varnish and remineralizing agents may be considered for early erosive lesions (15,18).

Dysgeusia (taste alteration) and altered oral sensations. Some patients treated with GLP-IRAs report dysgeusia or an altered taste perception; however, the prevalence estimates are poorly defined (19). Disturbances in taste can affect food preferences, oral hygiene habits and nutritional intake. Although symptoms are usually transient, if the disturbance persists, other causes should be excluded, e.g., nutritional deficiencies, polypharmacy and systemic disease. While the direct effects of GLP-IRAs on oral sensory pathways are speculative, it is crucial that clinicians are aware of these symptoms and provide appropriate reassurance and supportive care. Overall, a number of the oral manifestations associated with GLP-IRA therapy are relatively mild; however, their cumulative effects may have a marked effect on oral health, treatment outcomes and patient comfort. The critical roles of GLP-IRAs in early detection and the application of preventive strategies are incumbent upon those of dental practitioners.

4. GLP-1 receptor agonists and periodontal health: Drawbacks and benefits

A compelling aspect of GLP-IRA therapy is the potential beneficial effects of these drugs on periodontal tissues. The link between periodontitis and diabetes mellitus is well-established, is bidirectional in nature and is characterized by chronic systemic inflammation, impaired immune function and altered tissue repair (20). A reduction in periodontal inflammation and improved treatment outcomes have been associated with improved glycemic control (21); thus, the optimization of metabolic status may be an indirect periodontal benefit of GLP-IRAs. Experimental research has demonstrated that GLP-1 receptors in several cell types are relevant for periodontal health, such as osteoblasts, endothelial cells and immune cells (22). The activation of these receptors appears to modulate inflammatory pathways by downregulating the expression of pro-inflammatory cytokines, such as tumor necrosis factor- α (TNF- α), interleukin (IL)-1b and IL-6, and upregulating anti-inflammatory responses (23). Animal studies have also demonstrated that GLP-1 signaling can prevent alveolar bone loss and inhibit osteoclastogenesis via the modulation of receptor activator of nuclear factor κ B ligand (RANKL)-mediated pathways (24,25).

The potential periodontal benefits of GLP-IRA therapy should, however, be interpreted with caution. This biological rationale is supported by experimental evidence indicating that GLP-1 signaling can influence inflammatory pathways, oxidative stress, osteoclastogenesis and bone metabolism (26). Nevertheless, experimental effects cannot be assumed to translate directly into clinically meaningful periodontal improvement. Human evidence remains limited and is predominantly observational. Associations between GLP-IRA use and improved periodontal parameters may reflect improved glycemic control, weight reduction, changes in systemic inflammation or differences in healthcare behavior, rather than a direct pharmacological effect on periodontal tissues. Confounding by indication is particularly relevant as patients receiving GLP-IRAs may differ substantially from patients in

other groups in terms of diabetes severity, obesity, healthcare utilization and concurrent medications. Therefore, GLP-IRAs should not currently be considered a periodontal treatment or a substitute for plaque control, professional periodontal therapy, smoking cessation or supportive periodontal care. The available evidence is better interpreted as hypothesis-generating and supportive of further investigation. Well-designed prospective periodontal trials, ideally incorporating glycemic status, inflammatory biomarkers, microbiological outcomes and standardized periodontal endpoints are required before a direct therapeutic role can be established (27).

Implications for oral surgery and wound healing. Patients receiving GLP-IRAs frequently undergo routine dentoalveolar procedures, such as extractions, periodontal surgery and implant placement. This raises questions about the possible effects on wound healing and post-operative recovery. Delayed wound healing, post-operative infection and impaired periodontal regeneration are known to be associated with poor glycemic control (20). Theoretically, GLP-IRAs could improve glucose homeostasis, which can improve tissue repair and decrease postoperative complications (28). This concept is supported by experimental data demonstrating increased angiogenesis, reduced oxidative stress and the modulation of inflammatory responses in several tissue models upon GLP-1 receptor stimulation (22).

Preclinical studies have demonstrated that GLP-1 signaling promotes fibroblast proliferation, enhances collagen deposition and improves vascular function, all of which are important for oral wound healing (23). These anti-inflammatory effects may also benefit healing following periodontal or implant surgery. However, there is limited evidence from studies on oral surgery.

There is no current evidence available to support the routine discontinuation of GLP-IRA therapy prior to minor dental procedures under local anesthesia alone. Simple extractions, scaling and root surface debridement, restorative treatment and periodontal maintenance can usually be accomplished without any alteration of medication regimens as long as the medical condition of the patient is stable (29). However, clinicians should be aware of the possibility of gastrointestinal adverse effects that may affect post-operative care. Patients with marked nausea or vomiting may have difficulty tolerating post-operative analgesics, maintaining adequate nutrition or following oral hygiene instructions. Post-operative advice may also need to be reinforced.

The proposed effects of GLP-IRAs on wound healing remain biologically plausible, yet clinically uncertain in dentistry. Improved glycemic control could indirectly support tissue repair in patients with diabetes, while experimental studies suggest additional effects on inflammation, angiogenesis, oxidative stress and tissue remodeling. However, these mechanisms should not be interpreted as evidence that GLP-IRAs independently accelerate oral or periodontal wound healing. Recent non-dental surgical studies (30,31) have produced heterogeneous findings, with some retrospective analyses reporting favorable or neutral postoperative outcomes and others identifying possible wound-healing concerns. These studies are difficult to extrapolate to periodontal surgery or implant surgery due to differences in procedure type, diabetes severity, nutritional status, obesity,

comorbidities and peri-operative management. Thus, there is currently insufficient evidence to recommend GLP-1RA therapy specifically to enhance oral wound healing (30).

Dental implant therapy considerations. The association between GLP-1RA therapy and dental implant outcomes remains inadequately characterized. Improved glycemic control may theoretically reduce some of the biological risks associated with diabetes, while experimental research suggests that GLP-1 signaling may influence bone turnover, inflammation and osseointegration (27). However, these mechanisms do not establish a clinical benefit for implant therapy. The available human evidence concerning peri-implant health is limited and predominantly observational. Differences in diabetes control, periodontal status, smoking status, obesity status, implant characteristics and concurrent medications introduce substantial potential confounding effects. Consequently, GLP-1RA therapy should neither be considered an indication for implant treatment nor viewed as an independent predictor of implant success or failure. For current clinical practice, implant risk assessments should continue to prioritize glycemic control, periodontal stability, smoking status, oral hygiene, bone quality, systemic comorbidities and adherence to maintenance therapy. The potential effects of GLP-1RAs on osseointegration and peri-implant inflammation remain key research questions rather than established clinical indications (27). A summary of the strength of current evidence for oral and dental effects of GLP-1RAs is presented in Table I.

Sedation and perioperative issues: *The critical issue for dentists.* Delayed gastric emptying is clinically relevant when dental treatment involves deep sedation or general anesthesia as residual gastric contents may increase the potential for regurgitation and pulmonary aspiration. However, the magnitude of this risk varies among patients, and perioperative recommendations have evolved rapidly. Current multisociety guidance generally favors individualized risk assessment rather than the routine discontinuation of GLP-1RA therapy for each patient. Prior to sedation or general anesthesia, the dental team should document the GLP-1RA agent, indication, dose, frequency, timing of the most recent dose, recent dose escalation and the presence of gastrointestinal symptoms, such as nausea, vomiting, abdominal pain, bloating or severe reflux. Patients in an active dose-escalation phase or those with substantial gastrointestinal symptoms may represent a higher-risk group and require additional assessment or modification of the planned procedure. There is no basis for applying a universal prolonged fasting period or medication-withholding rule to every patient receiving a GLP-1RA. Instead, the dental practitioner should follow the current institutional, national and anesthesia-provider guidance applicable to the planned level of sedation. For patients considered to have an increased risk of aspiration, risk-mitigation measures may include modification of the procedure, consideration of a liquid-only diet for an appropriate period, adjustment of the anesthetic technique, or postponement when clinically indicated (17,29). Decisions concerning temporary interruption of GLP-1RA therapy should be made collaboratively with the prescribing clinician and anesthesia team, particularly in patients receiving medication for diabetes, as interruption may adversely affect glycemic control. For procedures

performed under local anesthesia without sedation, delayed gastric emptying is generally not a major procedural concern. Nevertheless, gastrointestinal symptoms, hydration and overall medical stability should still be considered (29,32,33).

5. Chair-side assessment: Practical guide for general dental practitioners

A focused medical and oral assessment should identify treatment-related symptoms and factors relevant to dental risk. The medical history should document the GLP-1RA agent, indication, dose, frequency, duration and any recent dose escalation. The oral assessment should record dry-mouth symptoms, altered taste, reflux or vomiting, hydration status and relevant dietary changes. Examinations should include the assessment of salivary function when indicated, mucosal condition, caries activity, erosive tooth wear, periodontal status and denture tolerance. For patients scheduled for deep sedation or general anaesthesia, the timing of the most recent dose, any recent dose escalation and clinically significant gastrointestinal symptoms should also be documented to support individualized perioperative risk assessment. The chair-side checklist should therefore focus on the following: i) Current GLP-1RA use and indications; ii) dose, frequency and timing of the last dose; iii) recent dose escalation; iv) nausea, vomiting, reflux or other critical gastrointestinal symptoms; v) dry mouth or altered taste; vi) tooth sensitivity or erosive wear; vii) planned sedation or general anesthesia. This focused assessment allows preventive and perioperative management to be tailored to the individual patient.

6. Preventive care and maintenance

Preventive dental care should be individualized according to the oral and systemic risk profile of the patient. Patients experiencing dry mouth should maintain adequate hydration, limit acidic beverages, and use sugar-free gum or saliva substitutes to support oral comfort and residual salivary function (34,35). Daily use of fluoridated toothpaste containing 1,350-1,500 ppm fluoride is recommended, with prescription-strength fluoride preparations or professionally applied fluoride varnish considered for patients at high caries risk. For patients with recurrent vomiting or reflux, dietary counselling and preventive measures are appropriate. Rinsing with water or a bicarbonate-containing mouthwash after vomiting and postponing tooth brushing for ~30 min may help minimize erosive tooth wear (18). Early erosive changes should be monitored, with minimally invasive restorative management considered when indicated. In patients with diabetes and periodontal disease, GLP-1RA therapy should complement rather than replace established periodontal treatment and supportive periodontal therapy. Recall intervals should be individualized according to periodontal and caries risk (30,31). A summary of practical chair-side assessment and the management of patients receiving GLP-1RAs is provided in Table II.

Practical advice for situation in everyday clinical practice. Routine dental procedures performed under local anesthesia can be completed without altering GLP-1RA therapy when the

Table I. Strength of current evidence for oral and dental effects of GLP-1 receptor agonists.

Oral/dental outcome	Current evidence	Predominant evidence type	Evidence strength	Clinical interpretation	(Refs.)
Xerostomia/hyposalivation	Emerging association, particularly with semaglutide; mechanism remains uncertain	Case reports/series, pharmacovigilance, mechanistic evidence	Low	Monitor for dry mouth and salivary dysfunction; avoid assuming a direct class effect	(6,7)
Reduced salivary flow and dehydration-related oral effects	Biologically plausible through reduced intake, nausea/vomiting and dehydration	Indirect clinical evidence + physiological/mechanistic evidence	Low	Hydration and preventive salivary management are reasonable	(7,8,34)
Nausea and vomiting	Well-established systemic adverse effects of GLP-1RAs	RCTs and large clinical trials	High	Clinically relevant because recurrent vomiting may secondarily affect oral health	(10)
Gastro-esophageal reflux/gastric exposure	GI effects and delayed gastric emptying are established; direct dental consequences are mainly inferred	RCTs/systematic evidence for GI effects; indirect dental evidence	Moderate for GI effects; Low for dental consequences	Monitor patients with reflux/vomiting for erosive tooth wear	(11,16,17)
Dental erosion	Plausible secondary consequence of recurrent vomiting/reflux; no evidence for direct drug-tooth interaction	Mechanistic inference, general dental evidence, clinical reports	Low	Preventive erosion management is appropriate; do not describe as direct GLP-1RA toxicity	(11,18)
Dysgeusia/altered taste	Reported in association with GLP-1RA treatment, but agent-specific oral evidence is limited	Clinical adverse-event data, pharmacovigilance, case-level evidence	Low	Consider medication and systemic/GI causes when evaluating persistent taste alteration	(40)
Halitosis	Possible secondary association through xerostomia, reflux, vomiting and dietary changes	Mechanistic/indirect evidence	Low	Assess oral hygiene, periodontal status, salivary function and reflux	(8,11,19)
Periodontal inflammation	Potential beneficial association has been reported, but causality is uncertain	Observational human studies + experimental evidence	Low	GLP-1RAs should not be considered periodontal therapy	(24-27)
Periodontal clinical parameters	Some studies suggest improved periodontal indices in GLP-1RA users	Retrospective/observational studies	Low	Findings are hypothesis-generating and potentially confounded by improved metabolic control	(24,26,27)
Periodontal inflammatory pathways	Anti-inflammatory and immunomodulatory effects are biologically plausible	<i>In vitro</i> /animal/mechanistic studies	Low-moderate mechanistic evidence	Supports biological plausibility but not clinical efficacy	(24,25,27)
Oral wound healing	Potential effects on inflammation, angiogenesis and tissue repair; human dental evidence scarce	Preclinical studies + indirect surgical evidence	Low	No basis for prescribing GLP-1RAs to improve oral wound healing	(30,41)

Table I. Continued.

Oral/dental outcome	Current evidence	Predominant evidence type	Evidence strength	Clinical interpretation	(Refs.)
Bone healing/osteogenesis	Experimental evidence suggests effects on bone metabolism	Animal/ <i>in vitro</i> studies	Low-moderate preclinical evidence	Translational relevance remains uncertain	(24,25,27)
Dental implant osseointegration	Promising preclinical findings; very limited human evidence	Animal studies + limited observational clinical evidence	Low	GLP-1RA use should not currently be regarded as an independent predictor of implant success	(24,27)
Peri-implant inflammation/marginal bone loss	Preliminary evidence suggests possible benefit	Limited observational evidence + preclinical studies	Very low-low	Requires prospective clinical studies	(24,27)
Direct effects on enamel/dentin/pulp	No convincing evidence of direct pharmacological damage	Current clinical/review evidence	Very low/insufficient	Avoid attributing dental hard-tissue damage directly to GLP-1RAs	(6,18,24)
Aspiration risk during deep sedation/general anesthesia	Delayed gastric emptying is established, but magnitude of aspiration risk is uncertain and patient-dependent	Pharmacological evidence, observational perioperative studies, consensus guidance	Moderate for delayed gastric emptying; low/uncertain for actual aspiration risk	Individualized perioperative risk assessment is needed; avoid blanket medication withholding	(17,29,32)

Evidence strength reflects the nature, quantity and consistency of the available evidence and is intended as a qualitative appraisal rather than a formal GRADE assessment. 'High' indicates consistent evidence from randomized controlled trials or large, well-established clinical datasets; 'moderate' indicates consistent observational or indirect clinical evidence; 'low' indicates predominantly observational, mechanistic, preclinical or case-level evidence; and 'very low/insufficient' indicates sparse, inconsistent or indirect evidence. Evidence strength should not be interpreted as an estimate of treatment efficacy. GLP-1RAs, glucagon-like peptide-1 receptor agonists; RCTs, randomized controlled trials; GI, gastrointestinal.

Table II. Practical chair-side assessment and management of patients receiving GLP-1 receptor agonists.

Clinical situation	Chair-side assessment	Practical management	(Refs.)
Xerostomia/hyposalivation	Symptoms, caries activity, mucosal dryness, salivary flow when indicated	Frequent water intake, sugar-free gum, saliva substitutes, fluoride toothpaste; consider professional fluoride varnish for high caries risk	(7,34,35)
Nausea/vomiting	Frequency, recent dose escalation, hydration and dietary tolerance	Medical review if persistent; rinse after vomiting; delay brushing; monitor erosive tooth wear	(10,18)
Reflux	Reflux symptoms, erosion pattern, medication history	Dietary counseling, erosion monitoring and medical referral when persistent/severe	(11,18)
Dysgeusia	Onset, medication timing, nutritional status, polypharmacy	Reassurance when transient; investigate persistent symptoms	(6)
Routine dental treatment under local anesthesia	GLP-1RA, indication and systemic stability	Usually, proceed without medication interruption	(29,32)
Sedation/deep sedation/GA	Drug, dose, last administration, dose escalation, GI symptoms and aspiration risk	Individualized risk assessment and coordination with anesthesia/sedation provider; follow current institutional/national guidance	(17,29,32)
High aspiration-risk patient	Active nausea/vomiting, significant GI symptoms, dose-escalation phase or other risk factors	Consider postponement or additional risk-mitigation strategy in consultation with anesthesia team	(17,29,32)

Chair-side framework for identifying GLP-1RA-related oral and gastrointestinal symptoms, assessing caries, erosive, periodontal and salivary risk, and implementing individualized preventive and treatment measures. Particular attention should be given to recent dose escalation, persistent gastrointestinal symptoms, hydration status, and planned sedation or general anesthesia. Perioperative medication modification should be determined collaboratively with the prescribing clinician and anesthesia team rather than by the dentist independently. GLP-1RAs, glucagon-like peptide-1 receptor agonists; GA, general anesthesia; GI, gastrointestinal.

patient is medically stable and has no critical gastrointestinal symptoms or other factors increasing the risk of aspiration. Patients with severe or poorly controlled nausea, vomiting, regurgitation, or reflux should undergo medical assessment before elective dental treatment. Elective procedures may be deferred until symptoms are adequately controlled, particularly when sedation is being considered (11,12). For procedures requiring deep sedation or general anesthesia, the dental team should follow the individualized perioperative risk assessment described above and coordinate with the sedation provider and prescribing clinician when indicated. Dentists should not independently discontinue GLP-1RA therapy; decisions regarding withholding or modifying treatment should be made collaboratively with the prescribing clinician and anesthesia team (11,12).

7. Key clinical message and limitations of current evidence

Key clinical message. The key clinical message provided by the current evidence is the following: i) GLP-1RA-related oral effects are predominantly indirect, and direct evidence for specific oral adverse effects remains limited. ii) Preventive dental care should be individualized according to caries, erosion, periodontal and salivary risk. iii) Proposed periodontal, wound-healing and implant benefits remain preliminary and should not alter established periodontal or implant treatment protocols. iv) For deep sedation or general anesthesia, the risk of aspiration should be assessed individually according

to treatment phase, gastrointestinal symptoms and current perioperative guidance.

Limitations of the current evidence. Several limitations should be considered when interpreting the available evidence on GLP-1RAs and oral health. First, direct dental evidence remains relatively limited compared with the extensive systemic literature on these medications. A large amount of the available oral evidence consists of case reports, small observational studies, pharmacovigilance analyses or extrapolation from gastrointestinal and systemic research (7,24,36). Second, patients receiving GLP-1RAs commonly have diabetes, obesity, cardiovascular disease or multiple concomitant medications, rendering it difficult to separate medication-specific effects from underlying disease and treatment-related confounding effects. Third, differences among individual GLP-1RAs, doses, treatment duration and routes of administration limit direct comparisons between studies. Fourth, experimental evidence demonstrating anti-inflammatory, bone-modulating or tissue-repair effects cannot be assumed to predict clinical periodontal, implant or wound-healing outcomes. Finally, perioperative recommendations have evolved rapidly as new evidence has emerged, and differences between professional guidance documents mean that recommendations should be interpreted in the context of local anesthesia, sedation and surgical protocols. These limitations highlight the need for prospective dental studies using standardized measures of salivary function,

dental erosion, periodontal inflammation, peri-implant bone levels and postoperative healing. Future research is warranted to also investigate whether specific GLP-1RAs differ in their oral effects and whether any observed periodontal benefits persist after adjustment for glycemic control, weight reduction and other metabolic factors.

8. Conclusion and future perspectives

GLP-1RAs are being increasingly used in clinical practice; however, evidence of their effects on the oral cavity remains limited. The majority of the available data are from observational studies or case reports or are extrapolated from the medical literature. High-quality prospective studies are warranted to investigate xerostomia, dental erosion, periodontal outcomes, and implant survival among users of GLP-1RAs. The potential anti-inflammatory and bone-modulating effects of GLP-1RAs open an interesting field for future periodontal studies. Further characterization of the direct effects of GLP-1 signaling on periodontal tissues may reveal novel therapeutic applications beyond glycemic control. Further research is also required to establish optimal perioperative management strategies for patients receiving GLP-1RAs undergoing dental procedures under sedation or general anesthesia.

In conclusion, GLP-1RA therapy is increasingly relevant to dental practice; however, the direct evidence linking these agents to oral outcomes remains limited. Dental care should therefore be guided by individualized assessment of oral risk, metabolic status and treatment-related symptoms rather than by assumptions about drug-specific toxicity. Emerging evidence for periodontal, wound-healing and implant-related benefits is biologically plausible, but remains preliminary and should not replace established preventive, periodontal or implant protocols. For procedures involving deep sedation or general anesthesia, perioperative management should follow current guidance and individualized aspiration-risk assessment, with interdisciplinary communication when indicated.

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Authors' contributions

DK, SUN and MS conceived and designed the study. DK was involved in the collection and processing of data from the literature. DK, SUN, BT and MS were involved in the analysis and interpretation of data from the literature, and in the literature search, as well as in the writing of the manuscript. All authors have read and approved the final manuscript. Data authentication is not applicable.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

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Competing interests

The authors declare that they have no competing interests.

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