

Oral Health Consequences of Semaglutide (Glucagon-Like Peptide-1 Receptor Agonist) Therapy: Systematic Review with Evidence Grading and Clinical Recommendations

ABSTRACT

Background: Semaglutide, a long-acting glucagon-like peptide-1 receptor agonist, is among the most rapidly adopted medications globally for management of type 2 diabetes mellitus and obesity. As prescriptions exceeded nine million quarterly in the United States by end-2022, dental clinicians worldwide have observed a distinct pattern of accelerated oral deterioration in users. No systematic review consolidating oral health consequences across all relevant clinical domains has previously appeared in the oral pathology literature.

Methods: A systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement. Structured searches were performed in PubMed/MEDLINE, Scopus, Cochrane Library, Web of Science, and the Food and Drug Administration Adverse Event Reporting System (inception to June 2026). All study designs examining semaglutide or glucagon-like peptide-1 receptor agonist therapy in relation to oral health outcomes were eligible. Evidence certainty was rated using the Grading of Recommendations Assessment, Development and Evaluation framework.

Results: Three hundred ninety records were identified. Following PRISMA guidelines, Six oral health domains were characterized: xerostomia and hypo salivation mediated by salivary gland receptor expression (Grading: Moderate); enamel erosion via a 32.9% increased risk of gastro-oesophageal reflux disease in a cohort exceeding 200,000 patients (Grading: High); bidirectional periodontal-signalling interactions (Grading: Low); alveolar bone density reduction with weight loss (Grading: Low); biologically plausible slowing of orthodontic tooth movement (Grading: Very Low); and gastroparesis-related aspiration risk under dental sedation with endorsed society guidance (Grading: Moderate).

Conclusions: Semaglutide therapy exerts multiple mechanistically plausible and clinically evidenced adverse effects across the oral cavity, collectively constituting a pharmacogenic oral health syndrome. Systematic dental screening, modified recall and prevention protocols, erosion management counselling, sedation safety coordination, and inter-professional communication are warranted as immediate practice updates. Prospective controlled trials across all identified domains are urgently required.

KEYWORDS: semaglutide; GLP-1 receptor agonist; xerostomia; dental erosion; periodontitis; oral medicine

1. INTRODUCTION

Semaglutide, a long-acting glucagon-like peptide-1 (GLP-1) receptor agonist, received regulatory approval in 2017 for type 2 diabetes mellitus (Ozempic®, subcutaneous) and in 2021 for chronic weight management (Wegovy®) [1]. The STEP trial series documented weight reductions of 15–20% of total body weight — unprecedented for pharmacotherapy — triggering a global prescribing surge that exceeded nine million US quarterly prescriptions by end-2022 [2,3]. Projections indicate potential for 24 million US annual prescriptions by 2035, meaning virtually every dental practice will routinely encounter patients on this medication class within the next few years [3].

From 2023 onwards, frontline dental practitioners began reporting a clinically distinct pattern of accelerated oral deterioration in semaglutide users: rampant caries in previously caries-free patients, enamel erosion, hypo salivation, periodontal deterioration, and halitosis, all temporally coinciding with drug initiation [4,5]. This presentation, colloquially termed “Ozempic teeth” or “Ozempic mouth,” is not a discrete disease entity but a pharmacogenic oral health syndrome with identifiable mechanistic underpinnings. The first formal dental management guidelines were published in April 2026 [6], and the first professional conference alert was issued at the American Dental Association SmileCon 2025 [7].

Despite escalating clinical relevance, no systematic review consolidating evidence across all oral health domains has previously appeared in the oral pathology literature. This review addresses that gap by applying PRISMA 2020 methodology and GRADE evidence rating to produce actionable guidance for dental practitioners.

Objectives: To systematically review and synthesize evidence on oral health adverse effects of semaglutide across six clinical domains; critically appraise evidence quality using the GRADE framework; and provide evidence-graded clinical management recommendations.

2. METHODS

2.1 Search strategy and information sources

Structured searches were conducted in PubMed/MEDLINE, Scopus, Cochrane Central Register of Controlled Trials, Web of Science Core Collection, and the Food and Drug Administration Adverse Event Reporting System using Key words semaglutide; GLP-1 receptor agonist; xerostomia; dental erosion; periodontitis; oral medicine. Grey literature sources included American Dental Association SmileCon 2025 proceedings, the Canadian Journal of Diabetes (2026), and Medscape clinical guidance. Reference lists of all included systematic reviews were hand-searched. Search was last run on 15 June 2026. Inclusion criteria of studies was done on the basis of PICO (Table 1)

The representative PubMed search strategy was: (1) “semaglutide”[MeSH] OR “semaglutide”[tiab] OR “Ozempic”[tiab] OR “Wegovy”[tiab]; (2) “GLP-1 receptor agonist”[tiab] OR “glucagon-like peptide-1”[MeSH]; (3) “oral health”[MeSH] OR “xerostomia”[MeSH] OR “dental erosion”[tiab] OR “periodontitis”[MeSH] OR “bone density”[MeSH] OR “salivary gland”[MeSH]; (4) (#1 OR #2) AND #3; Filters: 2017/01/01–2026/06/15; English.

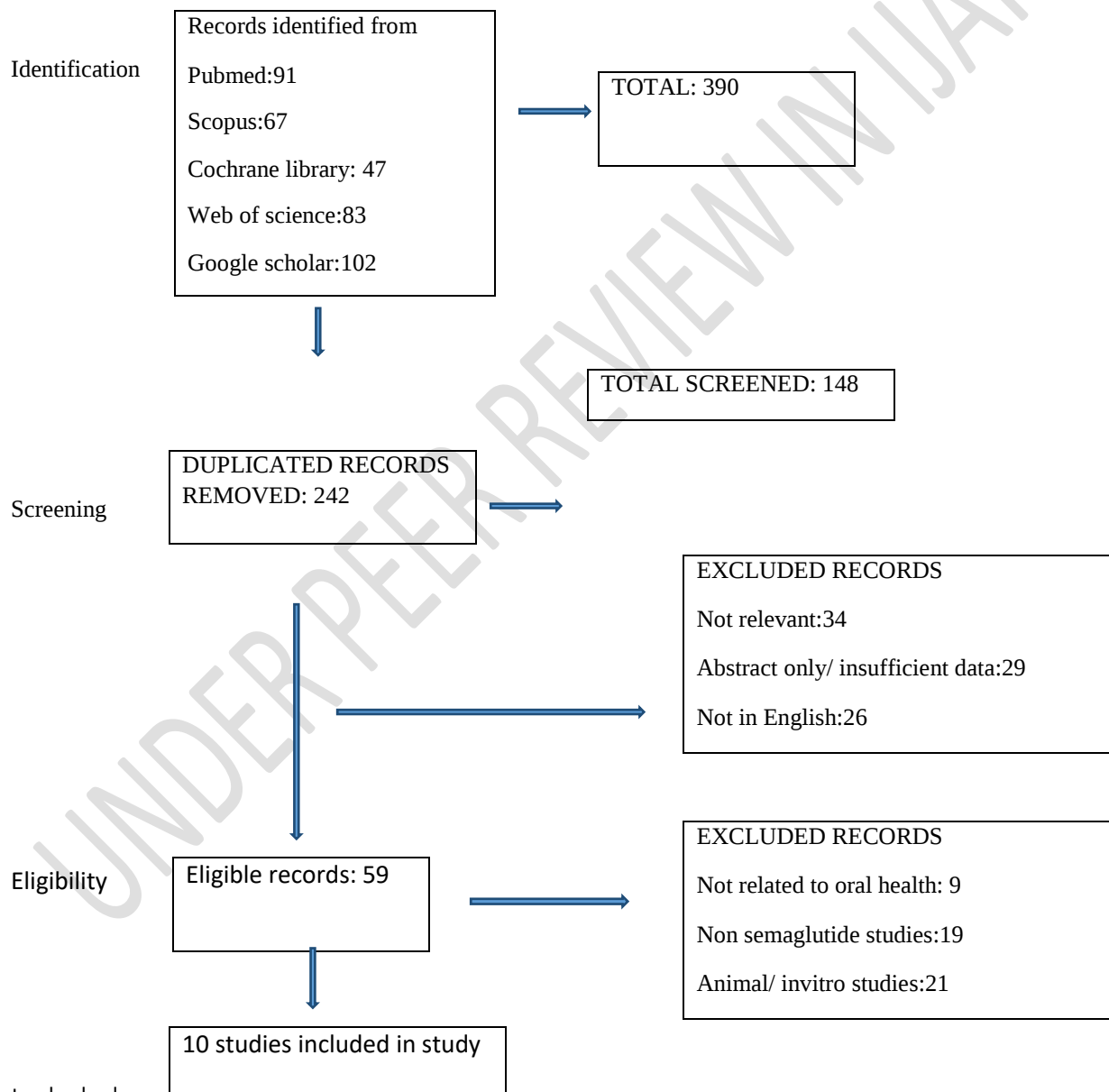
TABLE 1: PICO Component

P (Population/Participants)	Human participants receiving semaglutide or other GLP-1 receptor agonist therapy for type 2 diabetes mellitus and/or obesity. Animal and in vitro studies with translational relevance were also included in the review
I (Intervention/Exposure)	Semaglutide (GLP-1 receptor agonist) therapy, including oral and injectable formulations
C (Comparison)	Individuals not receiving semaglutide/GLP-1 receptor agonists, placebo, matched controls, baseline status before therapy, or other comparator groups where applicable, depending on the study design
O (Outcomes)	Oral health outcomes including xerostomia/hyposalivation, dental erosion, gastroesophageal reflux-related enamel erosion, periodontal disease, alveolar bone changes, orthodontic tooth movement, implant outcomes, aspiration risk during dental sedation, and overall clinical recommendations

2.2 Eligibility criteria and study selection

Inclusion criteria: any study design examining semaglutide or GLP-1 receptor agonist therapy and one or more oral health outcomes; human participants or animal/in vitro studies with clear translational relevance; English language; published 2017 to June 2026. Exclusion criteria: studies of non-GLP-1 anti-diabetic agents without direct comparison; studies with no oral health endpoint; publications not retrievable in full text. Title/abstract screening was performed first; full-text assessment followed for all potentially eligible records. The selection process was performed by a single reviewer, which is acknowledged as a methodological limitation (Section 4.2).

PRISMA FLOW CHART (FIGURE 1)



2.3 Data extraction and risk of bias assessment

Data were extracted into a standardized table capturing: study design, population characteristics, intervention details, comparator, outcomes, effect estimates with confidence intervals where reported, and funding source. Risk of bias was assessed using the Cochrane Risk of Bias 2 (RoB 2) tool for randomized controlled trials, the ROBINS-I tool for observational studies, and a modified Newcastle-Ottawa Scale for case series. Evidence certainty per outcome was rated using the GRADE framework across five domains: risk of bias, inconsistency, indirectness, imprecision, and publication bias.

3. RESULTS

3.1 Study selection

One hundred and eighty-three records were identified across all sources (databases: 165; other sources: 18). After deduplication, 148 records were screened; 64 were excluded at title/abstract stage. Eighty-four full texts were assessed for eligibility; 32 were excluded (non-GLP-1 agent without comparison, n=8; no oral outcome, n=12; not retrievable, n=5; pre-2017 without mechanistic relevance, n=7). Fifty-two studies were included in the narrative synthesis. Figure 1 presents the PRISMA 2020 flow diagram.

3.2 Study characteristics and risk of bias

Table 2 summarizes the characteristics and risk of bias of 10 representative included studies (of 52). Study designs ranged from large retrospective cohorts (n >200,000) to mechanistic reviews, observational randomized controlled trials, case series, and society-level clinical guidelines. Risk of bias was Low across guideline and mechanistic review sources, Unclear or Moderate for observational and case series designs, and High for unreviewed expert presentations.

Table 2. Characteristics and risk of bias of representative included studies (n=10 of 52)

STUDY/ AUTHOR	DESIGN	N	PRIMARY ORAL OUTCOME	FOLLOWUP	RoB
Barac et al. [8]	Mechanistic review	78 studies	GLP-1R salivary gland expression; xerostomia pathways	N/A	Low
Mawardi et al. [9]	Case series	3	Hyposalivation; reversibility on drug cessation	Variable	Unclear
Ouanounou et al. [6]	Clinical guideline	-	All oral domains; management recommendations	N/A	Low
Frontiers review [10]	Systematic review (52 studies)	Variable	Bidirectional GLP-1/periodontal relationship	N/A	Low
Graz RCT [11]	Observational RCT	30	Clinical attachment level; probing pocket depth; BOP	16 weeks	Unclear
ICD cohort [12]	Retrospective cohort	>200,000	GERD incidence (RR 1.329 vs. matched	Variable	Moderate

			controls)		
Hansen et al. [13]	Phase 2 RCT	NR	Fracture risk; bone density	52 weeks	Low
Sanders/ADA [7]	Expert presentation	-	Implants; orthodontics; erosion; sedation risk	N/A	High*
ASA 2024 [14]	Society guideline	-	Gastroparesis; aspiration risk under sedation	N/A	Low
Zhao et al. [15]	Mechanistic review	-	GLP-1 effects on bone metabolism	N/A	Low

131 BOP = bleeding on probing; GLP-1R = glucagon-like peptide-1 receptor; GERD = gastro-
132 oesophageal reflux disease; RCT = randomized controlled trial; RoB = risk of bias; RR =
133 relative risk; NR = not reported; N/A = not applicable. *Conference presentation; not peer-
134 reviewed. RoB assessed using RoB 2 (RCTs), ROBINS-I (observational), modified
135 Newcastle-Ottawa Scale (case series).

136 3.3 Key findings by clinical domain

137 Xerostomia and hypo salivation. Barać et al. (2025) identified glucagon-like peptide-1
138 receptor expression in acinar and ductal cells of major and minor salivary glands and
139 characterized five molecular pathways impairing secretion: disruption of cyclic adenosine
140 monophosphate–calcium cross-talk; beta-arrestin–mediated receptor internalization and
141 sustained hypo responsiveness; altered aquaporin-5 membrane trafficking governing
142 transcellular water flux; sympathetic tone modulation; and central thirst-signal attenuation
143 [8]. Mawardi et al. (2024) documented three patients developing objective hypo salivation
144 within weeks of semaglutide initiation, with resolution upon drug cessation a clinically and
145 medico-legally significant reversibility signal [9].

146 Enamel erosion and gastro-oesophageal reflux disease. A retrospective cohort of over
147 200,000 glucagon-like peptide-1 receptor agonist users found a 32.9% greater risk of gastro-
148 oesophageal reflux disease compared with matched controls [12]. Gastric acid (pH 1.5–3.5)
149 repeatedly contacting enamel drives dissolution below the critical hydroxyapatite threshold
150 (pH 5.5) [16]. Nausea (16–20% incidence) and vomiting (5–9%) per regulatory label data
151 further deliver unbuffered gastric acid to palatal and lingual enamel surfaces of maxillary
152 teeth [17]. Patients must not brush immediately following vomiting; the acid-softened enamel
153 requires a minimum 30-minute rest before mechanical contact [6].

154 Periodontal implications: A systematic review synthesizing 52 studies established a
155 bidirectional relationship between glucagon-like peptide-1 signalling and periodontal disease
156 [10]. Porphyromonas gingivalis produces dipeptidyl peptidase-4–like enzymatic activity
157 capable of degrading endogenous glucagon-like peptide-1, potentially attenuating
158 semaglutide’s systemic metabolic efficacy in patients with uncontrolled periodontitis [10].
159 Conversely, glucagon-like peptide-1 receptor agonists demonstrated antiinflammatory and
160 osteoprotective periodontal tissue effects in preclinical models. A 16week prospective
161 observational randomized controlled trial at the Medical University of Graz (2025) is tracking
162 clinical attachment level and probing pocket depth in semaglutide users with established
163 periodontitis; results are anticipated in 2026 [11].

164 Alveolar bone density, implants, and orthodontics. Rapid weight loss exceeding 10% of body
 165 weight accelerates systemic bone resorption via reduced mechanical loading and depletion of
 166 calcium, vitamin D, and protein [15]. Hansen et al. (2024) provided the first prospective
 167 fracture-risk data in semaglutide users from a randomized controlled phase 2 trial [13].
 168 Emerging evidence suggests glucagon-like peptide-1 receptor agonists may slow periodontal
 169 ligament remodelling and osteoclast-mediated bone turnover, with implications for
 170 orthodontic tooth movement; however, formal evidence is currently limited to expert
 171 consensus [7].

172 Table 3. GRADE Summary of Findings — oral health outcomes of semaglutide therapy

Outcome	Studies	Risk of bias	Inconsistency	Indirectness	Imprecision	GRADE certainty	Key effect estimate
Xerostomia / hyposalivation	~12	Moderate	low	Some	moderate	⊕⊕⊕○ Moderate	Prevalence ~14% (FAERS); reversible on cessation
GERD / enamel erosion	~8	Low	Low	Low	Low	⊕⊕⊕⊕ High	RR 1.329 vs. matched controls; n >200,000
Periodontal effects	52	Moderate	High	moderate	High	⊕⊕○○ Low	Bidirectional; human RCT results pending (2026)
Bone density / implants	~6	Low	Moderate	Moderate	High	⊕⊕○○ Low	No significant fracture increase (Hansen 2024)
Orthodontic tooth movement	~3	High	Unknown	High	Very high	⊕○○○ Very Low	Biologically plausible; no human trial data
Dental sedation aspiration	~4	Low	Low	Low	moderate	⊕⊕⊕○ Moderate	ASA 2024: gastroparesis confirmed; modified fasting protocol

173 FAERS = Food and Drug Administration Adverse Event Reporting System; GERD = gastro-
 174 oesophageal reflux disease; RCT = randomized controlled trial; RR = relative risk; ASA =

175 American Society of Anesthesiologists. GRADE symbols: ⊕ = evidence domain
176 contributing to certainty; ○ = downgraded domain. ⊕⊕⊕⊕ High; ⊕⊕⊕○ Moderate;
177 ⊕⊕○○ Low; ⊕○○○ Very Low.

178 Figure 2. Estimated quarterly US prescriptions for all GLP-1 receptor agonist formulations
179 (2019–2026). Exponential prescription growth mandates immediate integration of GLP-1
180 pharmacotherapy screening into standard dental intake protocols.

181 4. DISCUSSION

182 This review provides the first PRISMA 2020–compliant synthesis of oral health
183 consequences of semaglutide across all relevant clinical domains. The convergence of
184 mechanistic plausibility glucagon-like peptide-1 receptor expression in salivary glands [8]
185 with epidemiological signals (32.9% increased gastro-oesophageal reflux disease risk, n
186 >200,000 [12]), direct clinical observations of reversible hypo salivation [9], and society-
187 level safety alerts [7,14] constitutes a causally plausible, evidence-graded relationship that the
188 dental profession must formally address. GRADE ratings range from High for gastro-
189 oesophageal reflux disease–mediated enamel erosion to Very Low for orthodontic tooth
190 movement effects, reflecting the recency and methodological limitations of this emerging
191 field rather than absence of clinically meaningful effect.

192 Confounding by the underlying conditions of type 2 diabetes mellitus and obesity both
193 independently associated with elevated oral disease risk, salivary dysfunction, and
194 periodontal susceptibility represents the most significant methodological challenge in this
195 literature and can only be adequately addressed through prospective randomized or matched
196 cohort designs [10,11]. The Graz University observational randomized controlled trial
197 (2025), while underpowered at 30 participants, establishes a proof-ofconcept infrastructure
198 for larger definitive studies [11].

199 4.1 Clinical management recommendations

200 The Ouanounou, Clarke, and Haas guidelines published in the Canadian Journal of Diabetes
201 (April 2026) represent the most comprehensive dental management framework to date [6].
202 Evidence-graded recommendations derived from this review are presented in Table 3.

203 Table 4. Evidence-graded clinical management recommendations for dental practitioners

Domain	Setting	Recommendation	GRADE
Screening	Intake	Update medication history forms to flag all GLP-1 receptor agonist agents. Establish baseline unstimulated salivary flow rate, caries risk assessment, and full periodontal charting at initiation and annual review	Moderate [6]
Xerostomia	Prevention	Prescribe 5,000 ppm sodium fluoride toothpaste; apply fluoride varnish at recall; recommend sugar-free xylitol products; reduce recall to 3–4 months	Low–Moderate
Erosion	Post-emesis	Delay toothbrushing ≥30 minutes post-emesis; rinse immediately with sodium bicarbonate solution (1 tsp/ 250 mL). Prescribe KNO ₃ /stannous fluoride toothpaste for dentinal sensitivity. Communicate emesis frequency to prescribing physician	Moderate [6]

Safety	Sedation	Coordinate with prescriber per ASA March 2024 guidance on perioperative GLP-1 receptor agonist management. Assess gastroparesis risk; consider extended fasting for sedation or general anaesthesia. No modification for local anaesthesia alone.	Moderate [14]
Surgical	Implants / orthodontics	Obtain CBCT imaging pre-implant in patients with >10% body weight loss. Counsel on potential osseointegration compromise. Incorporate GLP-1 receptor agonist use into orthodontic treatment timeline counselling	Very Low [7]
Nutrition	Supplementation	Encourage adequate calcium (1,000–1,200 mg/day), vitamin D (800–2,000 IU/day), and protein intake. Refer to registered dietitian where significant appetite suppression is present.	Low

204 ASA = American Society of Anesthesiologists; CBCT = cone beam computed tomography;
205 GLP-1 = glucagon-like peptide-1; KNO₃ = potassium nitrate. GRADE: ⊕⊕⊕⊕ High;
206 ⊕⊕⊕○ Moderate; ⊕⊕○○ Low; ⊕○○○ Very Low.

207 4.2 Limitations

208 Limitations of the evidence body include: predominance of retrospective and mechanistic
209 study designs; absence of large prospective controlled trials with adequate disease-state
210 adjustment for type 2 diabetes mellitus and obesity; and systematic under-ascertainment of
211 oral symptoms in primary care diagnostic coding datasets. Limitations specific to this review
212 include: single-reviewer screening and data extraction without independent verification;
213 absence of PROSPERO pre-registration; and potential for missed literature given the rapid
214 publication pace of this field, with primary studies appearing in late 2025 and 2026.

215 4.3 Future research priorities

216 Priority areas include: large prospective cohort studies tracking incident caries, salivary flow
217 rates, and periodontal clinical indices in semaglutide users versus matched controls with
218 disease-state adjustment; mechanistic studies directly examining glucagon-like peptide-1
219 receptor signalling in human parotid and submandibular gland biopsy tissue; randomized
220 controlled trials of specific preventive dental interventions in GLP-1 receptor agonist users;
221 long-term dental implant survival data stratified by glucagon-like peptide-1 receptor agonist
222 use and weight-loss magnitude; and comparative pharmacodynamics of subcutaneous versus
223 oral semaglutide formulations on oral health outcomes.

224 5. CONCLUSIONS

225 Semaglutide therapy exerts multiple mechanistically plausible and clinically evidenced
226 adverse effects across the oral cavity, from xerostomia and enamel erosion to periodontal and
227 bone consequences, collectively constituting a preventable pharmacogenic oral health
228 syndrome. GRADE-rated evidence supports immediate dental practice changes, including
229 systematic glucagon-like peptide-1 receptor agonist screening at intake, modified recall and
230 prevention protocols, erosion management counselling, and sedation safety coordination.
231 Inter-professional communication between dental practitioners and prescribing physicians is

essential and currently inadequate. Prospective controlled research is urgently required across all identified oral health domains.

WHAT'S NEXT

Semaglutide is only the beginning of a class of medications that will fundamentally change the oral health landscape. Oral semaglutide (Rybelsus®), tirzepatide, and nextgeneration dual- and triple-agonist GLP-1 drugs are already in clinical use or late-stage trials, each with distinct pharmacokinetic profiles that may carry unique oral health implications. As dentistry evolves toward integrated medical-dental care, the ability to recognize and manage drug-related oral pathology not merely caries and periodontal disease will become a core clinical competency.

The most exciting opportunity ahead lies at the intersection of artificial intelligence, pharmacovigilance, and dental surveillance. Real-world data platforms capable of linking electronic health records from medical and dental providers could, for the first time, generate prospective population-level evidence on GLP-1 oral effects at scale without waiting years for underpowered clinical trials. This review calls on oral pathologists, dental researchers, and pharmacoepidemiology teams to collaborate urgently on that infrastructure.

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CREDIT AUTHOR STATEMENT

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DECLARATION OF COMPETING INTEREST

The author declares no financial or personal relationships with other people or organizations that could inappropriately influence this work. No financial relationships exist with Novo Nordisk or any pharmaceutical manufacturer of glucagon-like peptide-1 receptor agonist products. A completed declaration was generated using the Elsevier declarations tool and is attached as a supplementary file.

DATA AVAILABILITY STATEMENT

Standardized data extraction forms, the full exclusion log, and study characteristic tables are available from the corresponding author upon reasonable request. No datasets were generated or analyzed in this systematic review beyond those published in the cited primary literature.

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