

“Ozempic Face” and the Dental Professional: Managing GLP-1 Receptor Agonist–Associated Facial Volume Loss Through Biostimulators and Minimally Invasive Orofacial Harmonization — A Narrative Review

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Abstract

Background: Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) such as semaglutide and the dual agonist tirzepatide produce mean weight reductions of 15% to 21% in pivotal trials and have become one of the fastest-growing drug classes in the United States. Rapid fat loss is accompanied by facial deflation, laxity and an aged appearance popularly called “Ozempic face”. Because the lower third of the face, the lips and the masticatory system are the daily territory of dentistry, dental professionals are often the first clinicians to observe these changes, yet the dental literature has barely addressed them.

Materials and Methods: A narrative review was conducted in PubMed/MEDLINE (January 2014 to October 2026) using combinations of the terms semaglutide, tirzepatide, GLP-1, facial volume, aging, filler, biostimulator, poly-L-lactic acid, calcium hydroxylapatite, botulinum toxin, dentistry and oral health. Pivotal trials, imaging and survey studies, systematic and narrative reviews, consensus statements and state regulatory sources on dental scope of practice were synthesized qualitatively.

Results: Imaging data indicate a median midfacial volume loss of about 9%, roughly 7% per 10 kg lost, predominantly in superficial fat compartments; patient-reported facial volume loss rises from 13% with less than 10% weight loss to 50% with more than 20%. Current evidence attributes the phenotype to the speed and magnitude of weight loss superimposed on aging, rather than to a drug-specific toxicity. Contributing mechanisms include fat-compartment deflation, lean-mass loss, nutritional deficiency and skeletal aging. Oral consequences (xerostomia, dysgeusia, reflux-related erosion risk) and aspiration risk during sedation are also relevant to dental care. Treatment evidence is preliminary: a 2026 systematic review found only four uncontrolled studies with 98 participants. Biostimulators (poly-L-lactic acid and calcium hydroxylapatite) appear well suited to a tissue-rebuilding rather than an “inflating” strategy, with hyaluronic acid used selectively for structural support.

Conclusion: Dentists trained in facial anatomy can contribute to the recognition, staged planning and conservative management of GLP-1–associated facial change, provided they work within state scope-of-practice rules, coordinate with prescribers, time interventions to weight stability and maintain complication protocols. A five-step dental clinical pathway and a research agenda are proposed.

Key Word: GLP-1 receptor agonists; semaglutide; tirzepatide; facial volume loss; biostimulators; orofacial harmonization; dentistry.

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I. Introduction

Few drug classes have changed clinical medicine as rapidly as the incretin-based therapies. In the STEP 1 trial, once-weekly semaglutide 2.4 mg produced a mean body-weight reduction of 14.9% at 68 weeks, compared with 2.4% with placebo [1]. In SURMOUNT-1, the dual GIP/GLP-1 receptor agonist tirzepatide reduced body weight by 15.0%, 19.5% and 20.9% at the 5, 10 and 15 mg doses over 72 weeks [2]. A 2026 critical appraisal in the *Journal of Dental Research* reported that prescriptions of GLP-1 receptor agonists for obesity treatment grew by 587% between 2019 and 2024 [3], and these agents are now among the most widely prescribed medications in the United States [4].

The cardiometabolic benefits of this weight loss are well established. A less welcome consequence, however, has entered both the clinic and popular vocabulary: “Ozempic face”, a cluster of facial hollowing, skin laxity, deepened folds and an apparently accelerated aging [5,6]. The term is misleading, because similar changes follow any substantial and rapid weight loss, including bariatric surgery [5,6]. It is nonetheless clinically useful, because it captures a new and very large population of patients who seek aesthetic restoration after medically indicated weight loss.

The dental professional occupies a privileged position in relation to this phenomenon. The perioral region, the lips, the support provided by the dentition and alveolar bone, the masticatory muscles and the lower facial contour are evaluated at every routine dental visit. Dentists also see patients more regularly than most aesthetic physicians, frequently over many years. The aim of this narrative review is therefore threefold: to synthesize current evidence on the phenotype and mechanisms of GLP-1 receptor agonist–associated facial change; to appraise the minimally invasive options for its management, with emphasis on biostimulators; and to propose a dental clinical pathway framed by the regulatory, safety and ethical realities of practice in the United States.

II. Materials and Methods

This is a narrative review. Searches were run in PubMed/MEDLINE from January 2014 to October 2026, combining the terms semaglutide, tirzepatide, GLP-1, incretin, facial volume, facial aging, Ozempic face, filler, biostimulator, poly-L-lactic acid, calcium hydroxylapatite, botulinum toxin, lip, perioral, masseter, dentist, dentistry and oral health. Pivotal randomized trials of the drugs, imaging and survey studies quantifying facial

change, systematic, scoping and narrative reviews, expert consensus documents on injectable complications, and studies on oral and perioperative implications were prioritized. Classic anatomical references on facial fat compartments and skeletal aging were added for context. Because scope of practice in the United States is defined state by state, publicly available regulatory and professional sources were consulted for that section. Articles were selected for relevance and recency, and findings were synthesized qualitatively; no meta-analysis was attempted, given the heterogeneity and the predominance of uncontrolled designs.

III. The Clinical Phenotype of GLP-1–Associated Facial Change

A. Quantifying facial deflation

The first objective, imaging-based quantification came from a retrospective cohort of 20 patients with computed tomography or magnetic resonance images obtained before and after GLP-1 receptor agonist prescription. After an average weight loss of 11.0 kg, the median decrease in total midfacial volume was 9.0%; superficial volume decreased by 11.0% and deep volume by 7.0%, and linear regression indicated a loss of about 7% of midfacial volume for every 10 kg of weight lost [7]. Superficial volume loss correlated with weight loss, whereas deep volume loss did not [7], which suggests that the superficial fat compartments are the first to deflate.

Patient-reported data are consistent with these findings. In an anonymous survey of 1,226 patients in a virtual weight-management program, decreased facial volume was reported by 13% of respondents who lost less than 10% of their body weight, 37% of those who lost 10% to 20%, and 50% of those who lost more than 20%; skin sagging rose from 15% to 44% and 72% across the same categories [8]. Overall, more than 60% of participants reported concerns such as skin laxity, facial volume loss, hair shedding or reduced muscle strength [9]. The authors note that the sample was largely composed of White, female telemedicine patients, which limits generalization [9].

Practitioners report the same trend from the provider side. In a survey of 406 aesthetic health care professionals, respondents described a mean increase of 137% in the number of patients receiving GLP-1 agonists between 2023 and 2024, and identified midface volume loss and face and neck laxity as the changes most affected [10]. That survey was conducted with industry participation, and its treatment preferences should be read with that in mind.

B. A weight-loss phenotype, not a drug toxicity

A 2026 PRISMA-guided review of 46 reports concluded that the available evidence favors weight-loss–associated deflation superimposed on baseline anatomy and aging, rather than a drug-specific adverse effect, while acknowledging that the independent effect of the rate of weight loss remains uncertain [5]. A contemporary surgical review reached the same conclusion and added that GLP-1 use should be considered neither an absolute advantage nor an automatic contraindication to aesthetic procedures [6]. For the clinician, this distinction matters: the relevant variables are the magnitude and speed of weight loss, age, baseline facial volume and skin quality, not the brand of the medication.

IV. Pathophysiology: Compartments, Lean Mass, Nutrition and Bone

The face is not a single fat depot. Its subcutaneous fat is partitioned into discrete compartments separated by septa, and these compartments age and deflate semi-independently [11]. This anatomy explains why rapid weight loss does not shrink the face uniformly but produces localized hollowing, step-offs and shadows, particularly in the midface and periorbital region. Beneath the soft tissues, the facial skeleton itself remodels with age, with resorption at the orbital rim, maxilla and mandible reducing the scaffold on which soft tissues drape [12]. In older patients who lose weight quickly, soft-tissue deflation is therefore superimposed on a shrinking skeletal frame.

Recent reviews have extended this model. Dermal white adipose tissue, skeletal remodeling and sarcopenia have been proposed as contributors to the GLP-1 phenotype [13]. Body-composition data from incretin trials indicate that lean soft tissue accounts for roughly 25% to 30% of total weight lost, a change that is pharmacologically distinct from age-related sarcopenia but relevant to tissue quality [14]. Nutrition is a further, often overlooked, factor: in patients receiving these drugs, vitamin D deficiency reaches 13.6% at 12 months and about 22.4% have at least one documented deficiency within the first year, which may compromise collagen synthesis and dermal repair [15]. These observations support an integrated approach in which aesthetic restoration is coordinated with nutritional optimization and the prescribing physician [13,15].

V. The Lower Face and the Oral Cavity: Why the Dentist Sees It First

A. Perioral and lower-third changes

The lower face, including the lips, chin and jawline, shows an aging pattern that combines skeletal remodeling, soft-tissue descent and muscle hyperactivity, and this pattern differs among ethnic groups: perioral atrophy and rhytids predominate in patients of European descent, whereas chin retrusion and jawline heaviness

are more common in East and South Asian patients [16]. Weight loss accelerates these changes by removing the fat that supports the lips and the labiomental and mandibular contours. For the dentist, the clinical consequences are concrete: reduced lip support can alter incisal display, accentuate perioral rhytids around a recently completed restoration, and change the perceived outcome of prosthodontic or orthodontic treatment. A structured lip assessment that considers volume, shape and symmetry, perioral aging, tissue quality and previous treatments, such as the recently proposed Lip Complexity Classification, can help stratify these patients, although that framework still requires validation [17].

B. Oral manifestations of incretin therapy

The same patients may present with oral complaints. Gastrointestinal adverse effects, reduced fluid intake and possible salivary changes underlie a cluster of complaints described colloquially as “Ozempic mouth”, “Ozempic teeth” and “Ozempic breath” [18]. Pharmacovigilance analyses have reported xerostomia, dysgeusia and anosmia, particularly with semaglutide, while nausea (15% to 50%), vomiting (5% to 24%) and gastroesophageal reflux pose a plausible but unquantified risk of erosion and secondary caries [3]. Pharmacovigilance data also suggest that semaglutide may impair salivary gland responsiveness, a concern that becomes more relevant as tirzepatide is used for obstructive sleep apnea [19]. At the same time, preclinical data point to anti-inflammatory and osteoprotective effects on periodontal tissues, and no prospective study has yet used a dental outcome as an endpoint in patients receiving these drugs [3].

C. Sedation and aspiration risk

Delayed gastric emptying has prompted concern about pulmonary aspiration during anesthesia. Anesthesia guidance has moved from blanket preoperative withholding toward individualized risk stratification based on symptoms and dose-escalation phase [20], and the dental anesthesia literature has identified GLP-1 use as a potential blind spot in the preoperative assessment for dental sedation [21]. Any dental practice offering aesthetic or surgical procedures under sedation should therefore record incretin use, dose and recent escalation in the medical history.

VI. Minimally Invasive Management Options

A 2026 systematic review registered in PROSPERO identified only four uncontrolled observational studies, with 98 treated participants, evaluating interventions for facial changes associated with GLP-1–related weight loss; all reported favorable within-group results, but the absence of comparators precluded conclusions on effectiveness or superiority [22]. The options summarized in Table 1 must therefore be read as reasoned extrapolations from the broader aesthetic literature, not as validated GLP-1–specific protocols.

Table 1. Minimally invasive options for GLP-1–associated facial change and their relevance to dental practice

Modality	Mechanism / role	Evidence and cautions	Relevance to the dental setting
Hyaluronic acid (HA) fillers	Immediate volumetric and structural support	Considered first-line by many providers [10]; should restore support rather than “inflate” the face [4]	Lip support, labiomental fold, chin and jawline in the lower third
Poly-L-lactic acid (PLLA)	Gradual polymer hydrolysis, fibroblast stimulation and neocollagenesis [23]	Staged sessions, delayed onset; effects reported for 2 years or longer in industry-authored review [24]; nodule risk [25]	Perioral and midface deflation, jawline definition
Calcium hydroxylapatite (CaHA), incl. hyperdilute	Immediate scaffold plus neocollagenesis and elastogenesis [23,26]	Hyperdilution favors skin quality over volume [26]; pilot histology shows distinct profile from PDLLA [27]	Laxity of lower face and submandibular region
Botulinum toxin type A	Neuromodulation of hyperactive or hypertrophic muscles	Reduces bigonial width with repeat treatment and no permanent adverse events reported [28]	Masseteric hypertrophy, bruxism-related contour, gummy smile, perioral lines
Autologous fat grafting	Volume and regenerative cell transfer	GLP-1 biology may antagonize graft survival; no direct clinical data [29]	Usually outside dental scope; referral
Energy-based devices	Dermal remodeling and tightening	Combined in regenerative protocols [30]; GLP-1–specific evidence lacking [4]	Referral or interdisciplinary care

Sources as cited. Evidence in GLP-1 users is largely uncontrolled; options reflect extrapolation from general aesthetic practice.

A. Hyaluronic acid: support, not inflation

Hyaluronic acid fillers remain the most direct way to restore lost volume, and in the provider survey they were judged the best treatment for facial concerns in about half of GLP-1 patients [10]. In this population, however, the goal is to restore structural support at strategic points (for example, the lateral midface, chin and prejowl area) rather than to replace every milliliter of lost fat. Over-filling a deflated face whose weight may still be changing risks a heavy, unnatural result and is at odds with the current shift toward natural-looking outcomes [4,22].

B. Biostimulators: rebuilding the tissue

Biostimulatory injectables shift the therapeutic focus from immediate volumization to biologically driven regeneration. Calcium hydroxylapatite microspheres provide immediate support while acting as a scaffold for new collagen, whereas poly-L-lactic acid microparticles stimulate fibroblasts gradually and produce cumulative improvement over staged sessions [23]. Hyperdilute calcium hydroxylapatite emphasizes neocollagenesis, neoelastogenesis and matrix remodeling over large fields [26], and a small split-face histological study suggested complementary profiles: poly-D,L-lactic acid favored angiogenesis and collagen remodeling, while calcium hydroxylapatite increased elastin density by 13% [27]. Because the GLP-1 phenotype involves both lost volume and weakened dermal support, biostimulators are conceptually attractive, and a review focused on this population reported clinically meaningful corrections of midface and perioral volume loss and jawline contour [24]. That review was authored largely by manufacturer employees, and independent controlled data are still needed. Regenerative protocols that combine multilayer fat grafting, biostimulatory scaffolds, hybrid HA complexes and energy-based devices in a staged plan have also been proposed [30].

C. Neuromodulators and the masticatory system

Botulinum toxin type A has a long history in dentistry for therapeutic indications such as oromandibular dystonia and sialorrhea, and dentists starting with neurotoxin are advised to begin with these well-documented indications, with precise targeting and interdisciplinary diagnosis [31]. In the lower face, masseter injection reduces bigonial width, with treatment repeated at regular intervals to maintain results [28]. After weight loss, a hypertrophic masseter that was previously concealed by facial fat may become prominent, and the interplay between bruxism, temporomandibular disorders and facial contour falls naturally within the expertise of dentists trained in orofacial pain.

D. Fat grafting and energy-based devices

Autologous fat grafting is the classical restorative option after massive weight loss, but a 2026 scoping review identified several mechanisms through which GLP-1 receptor agonism, including adipocyte browning, enhanced lipolysis and altered differentiation of adipose-derived stem cells, could compromise graft survival; no clinical study has directly tested this question [29]. Energy-based devices are increasingly used in this population, although GLP-1–specific evidence is lacking [4]. Both modalities generally lie outside the dental scope of practice and are better handled through referral.

VII. Timing, Safety and Complication Preparedness

Timing is probably the most important decision. Proposed frameworks anchor elective aesthetic procedures to weight stability and symptom burden, noting that weight regain after drug discontinuation is frequent and threatens the durability of results [20]. A health-first approach that prioritizes tissue preservation during active weight loss and sequences corrective treatment afterwards has also been recommended [13]. In practice, conservative biostimulatory or structural treatment can be considered during the plateau or maintenance phase, with larger volumetric corrections deferred until the weight trajectory is stable.

Complication preparedness is non-negotiable. Expert consensus classifies filler complications by time of onset and gives particular attention to vascular compromise and retinal artery occlusion, recommending emergency protocols with the necessary agents readily available in the office [32]. For poly-L-lactic acid, international consensus now distinguishes noninflammatory from inflammatory nodules and provides a management algorithm [25]. Reports of filler nodules attributed to tirzepatide have been questioned for insufficiently excluding infectious and biofilm-related causes [33], a reminder that sepsis and careful differential diagnosis remain essential. Patient-reported outcome measurement should accompany clinical photography: a 15-item FACE-Q short form validated specifically in GLP-1 users, with minimally important differences of 6 to 10 points, is now available [34].

VIII. A Proposed Dental Clinical Pathway

Integrating the evidence above, a five-step pathway is proposed for dental practices (Table 2). It is intended as a pragmatic framework to be tested, not as a validated protocol.

Table 2. Proposed five-step dental pathway for patients with GLP-1–associated facial change

Step	Action	Supporting rationale
1. Screen	Record GLP-1/GIP agonist use, dose, escalation phase, percentage and speed of weight loss; screen for xerostomia, erosion and reflux	Facial and oral effects scale with weight loss [3,8]; sedation risk [21]
2. Document	Standardized photographs or 3D facial scan, lip and lower-face assessment, FACE-Q baseline	Objective and patient-reported baselines [17,34]; digital facial workflows [35]
3. Coordinate	Communicate with the prescriber; address nutrition and protein intake; confirm weight trajectory	Nutritional deficiencies and lean-mass loss [14,15]
4. Stage treatment	Oral health first; then structural support and biostimulation within scope; defer large volumization until weight is stable	Timing frameworks [13,20]; biostimulators [23]
5. Safeguard and refer	Emergency protocol for vascular events; nodule algorithm; refer upper face, fat grafting and devices	Consensus documents [25,32]

The first two steps are ones that almost every dental practice can implement immediately, regardless of whether it offers injectables. The digital workflow already used in dentistry, including cone-beam computed tomography, virtual planning and three-dimensional records, as described for the surgical management of facial fractures [35], offers a natural infrastructure for documenting facial change over time.

IX. Scope of Practice in the United States

Dental scope of practice for botulinum toxin and dermal fillers is defined state by state, which produces a heterogeneous regulatory landscape [36]. In New Jersey, dentists may use FDA-approved injectables after completing a course approved by the state Board of Dentistry; in Pennsylvania, current policy permits both therapeutic and cosmetic use within the scope of training; in New York, stand-alone cosmetic procedures not tied to dental treatment are not clearly within scope [37]. By contrast, the North Carolina Board of Dental Examiners has stated that purely aesthetic facial enhancement is not part of dental practice, whereas South Carolina issued a statement in 2017 permitting cosmetic neuromodulator injection by dentists [36]. Dentists must therefore verify the rules of their own state, the required training and the anatomical boundaries before offering any of the options discussed here. The most defensible position, both legally and clinically, is to link treatment to the perioral and lower-face region and to a documented dental or functional rationale.

From a practice-management perspective, these services also raise questions of investment, training and patient flow that are part of the broader strategic and economic evolution of dental practice in the United States [38]. Expansion should follow competence and regulation, not the other way around.

X. Ethical and Psychosocial Considerations

Two ethical issues deserve emphasis. First, the demand for aesthetic correction should never become an argument against a medically indicated therapy; the cardiometabolic benefits of weight loss outweigh its facial consequences [1,2]. Conversely, cosmetic prescribing of these drugs raises concerns about nonmaleficence, scope of practice and prescribing cascades that have been discussed in the dermatology literature [39], and dentists should not be drawn into that role. Second, patients who lose weight with GLP-1 receptor agonists may hold distinct expectations: in a prospective comparison with post-bariatric patients, GLP-1 users had higher preoperative satisfaction scores but greater emotional reactivity to residual irregularities, and lower baseline psychosocial scores predicted postoperative dissatisfaction [40]. Careful counseling, realistic simulation and documentation of informed consent are therefore essential.

XI. National Interest and Significance

The scale of incretin use in the United States [3,4] means that GLP-1–associated facial change is no longer a niche aesthetic concern but a population-level phenomenon intersecting with obesity care, oral health and patient adherence. Unwanted aesthetic and muscle effects have been described as a potential threat to treatment adherence [9], which links facial care, indirectly, to the success of a public-health strategy against obesity. Dentists, who see patients regularly and examine the lower face and oral cavity at each visit, are well placed to detect oral adverse effects, flag sedation risk, document facial change objectively and refer appropriately. Building this competence into continuing education, aligning it with state regulation, and generating dental-specific outcome data would strengthen the contribution of dentistry to integrated, interdisciplinary care.

XII. Limitations and Research Agenda

This review has the limitations inherent to a narrative design: study selection was not systematic, and the treatment literature in GLP-1 users consists almost entirely of uncontrolled, small and, in part, industry-sponsored studies [10,22,24]. Survey populations were largely White and female [9], limiting generalization to the diverse U.S. population. Priority research questions include: prospective, controlled comparisons of HA, poly-L-lactic acid and calcium hydroxylapatite in GLP-1 users; the effect of treatment timing relative to weight stability and drug discontinuation; lower-face and perioral volumetric changes measured by three-dimensional scanning; the prevalence of erosion, xerostomia and caries in incretin users [3]; and the impact of nutritional optimization on aesthetic outcomes [15].

XIII. Conclusion

"Ozempic face" is best understood as the facial expression of rapid, substantial weight loss superimposed on aging, not as a direct drug toxicity. Its main features are superficial fat-compartment deflation, laxity and lower-face changes that are especially visible in the perioral region. The dental professional is well positioned to recognize these changes, to identify the associated oral and sedation risks, and, where state law and training permit, to contribute to conservative, staged management in which biostimulators rebuild tissue and hyaluronic acid restores support without over-filling. The proposed five-step pathway (screen, document, coordinate, stage and safeguard) offers a practical starting point, but its value, like that of every treatment discussed here, still needs to be confirmed by controlled clinical research.

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