cosmetics

MDPI

Review

Cosmetic Considerations of Semaglutide

Alaina Baggett [1,*], Carissa Saadi [1], Robert Saadi [1,2] and Vijay Patel [3]

[1] College of Medicine, University of Arkansas for Medical Sciences, Little Rock, AR 72205, USA
[2] Department of Otolaryngology, University of Arkansas for Medical Sciences, Little Rock, AR 72205, USA
[3] Department of Otolaryngology—Head & Neck Surgery, University of California San Diego, San Diego, CA 92093, USA
* Correspondence: adbaggett@uams.edu

Abstract

Semaglutide-induced facial changes, or "Ozempic face" popularized by media, have gained increasing recognition since the widespread and growing use of Ozempic (semaglutide) for weight loss. It refers to facial volume depletion and soft tissue laxity following rapid weight loss associated with this medication. Semaglutide use can also cause gastrointestinal side effects, volume loss, and decrease skin quality not only in the face but globally. As the use of Ozempic becomes increasingly popular, more patients are presenting to cosmetic clinics for these undesirable esthetic changes. While cosmetic changes following rapid weight loss is not new, such as those following bariatric interventions, the accessibility and ease of GLP-1, Glucose-like protein-1, makes this a growing concern among the community. It is important for clinicians to recognize these potential effects, counsel patients appropriately, and give options for treatment. This emerging esthetic concern highlights the need for further investigation into underlying causes, risk factors, and potential interventions.

Keywords: semaglutide; GLP-1 receptor agonist; Ozempic; wegovy; facial lipoatrophy; weight loss side effects; facial aging

Academic Editor: Enzo Berardesca

Received: 9 September 2025
Revised: 30 September 2025
Accepted: 30 September 2025
Published: 9 October 2025

Citation: Baggett, A.; Saadi, C.; Saadi, R.; Patel, V. Cosmetic Considerations of Semaglutide. *Cosmetics* **2025**, *12*, 221. https://doi.org/10.3390/cosmetics12050221

1. Introduction

GLP-1 receptor agonists are commonly used in the management of type 2 diabetes mellitus and have recently gained widespread popularity for their effectiveness in promoting weight loss. Obesity is a growing global concern and is associated with increased mortality due to its link with numerous chronic diseases [1]. Obesity has become a global epidemic in the past few decades but has been documented to exist for tens of thousands of years. However, these documented cases were the exception rather than commonplace like they are today [2]. Given the increased occurrence of obesity and its serious health implications, the availability of a medication that can promote rapid weight loss may appear to be a breakthrough. One medication which may be the answer to this is GLP-1 such as semaglutide. However, there is limited data on the long-term safety and effects of semaglutide. Given the serious health implications of obesity, the availability of a medication that can promote rapid weight loss may appear to be a breakthrough. However, there is limited data on the long-term safety and effects of Ozempic. Despite its intended use for diabetes and obesity, GLP-1 RAs are increasingly being used off-label by individuals who do not meet obesity criteria but wish to lose relatively small amounts of weight. While effective for weight reduction, GLP-1 use has led to undesirable esthetic side effects, such as facial volume loss and skin laxity—often termed "Ozempic face." We hope to explore

the pathophysiology, prevalence, and management of semaglutide-induced facial changes with this review.

2. Mechanism of GLP-1 Agonist

Glucagon is a hormone produced by the alpha cells of the pancreas and plays an important role in regulating glucose levels [3]. It is released from pancreatic alpha cells when blood glucose levels fall too low [3]. Glucagon then promotes glycogenolysis which releases glucose from glycogen reserves and increases blood glucose levels [3]. Apart from its role in blood glucose regulation, glucagon also has multiple metabolic effects. Glucagon acts on the brain to suppress appetite by signaling satiety, it increases metabolic rate, promotes lipolysis while inhibiting lipid synthesis, promotes hepatic glucose production, and activates autophagy to support cellular energy homeostasis [4–7]. Glucagon-like peptide (GLP-1) is an incretin hormone derived from the gastrointestinal system which mimics some of the same effects of glucagon [8]. However, unlike glucagon, GLP-1 promotes insulin secretion following ingestion of meals [9]. Synthetic GLP-1 peptides are used to lower blood glucose level by increasing insulin secretion and promoting glucose uptake into muscles [9]. These mechanisms make them an effective treatment in type 2 diabetes mellitus [10]. GLP-1 receptor agonists (GLP-1 RAs), such as semaglutide, function as GLP-1 analogs [11]. They decrease glucagon secretion, slow gastric emptying, and increase glucose-dependent insulin release [11]. These drugs are also known to reduce appetite and therefore food intake, leading to weight loss [8]. Additionally, glucagon-like peptide-1 receptor agonist may provide benefit to any organ system that expresses GLP-1 receptors which include the renal system, brain, gastrointestinal tract, and cardiovascular systems [9,10]. GLP-1 is produced by preproglucagon (PPG) neurons in the nucleus of the solitary tract of the brain. The GLP-1 receptors are found in the hypothalamus, amygdala, mesolimbic reward system, nucleus tractus olitarius (NTS), and area postrema (AP), which all play a role in the control of food intake and regulation [12,13]. It is shown to display a critical role with decreasing inflammation and neuroprotective factors [12]. Native GLP-1 has a very short half-life of 1–2 min which limits its therapeutic uses [11]. This short half-life is due to both inactivation by the enzyme dipeptideylpeptidas (DDP- and renal elimination [11]. In order to utilize these benefits for the treatment of diabetes and obesity, different GLP-1 derivatives with extended half-life have been created. These include liraglutide, exenatide, lixisenatide, albiglutide, and dulaglutide [14].

The current GLP-1 RAs that are FDA-approved include exenatide, lixisenatide, liraglutide, dulaglutide, semaglutide, and tirzepatide, all of which differ in terms of medication benefits and routes of administration [15]. Exenatide is a short-acting GLP-1 RA that is formulated to be administered subcutaneously twice a day. It is the first synthetic peptide in clinical use [13]. Lixisenatide, structurally similar to exenatide, is administered subcutaneously as well but is only once a day [13]. Exenatide was shown to reduce HbA1x by 0.78% while lixisenatide reduced HbA1c by 0.8–0.9% [13]. It is noted that use of exenatide twice daily and lixisenatide should be avoided in patients with decreased kidney function as they are mainly excreted by the kidneys [13]. Exenatide was later formulated into an extended release to be administered once weekly and was able to reduce HbA1c by 1.9% [13]. Other long-acting agents include liraglutide, liraglutide, dulaglutide, and semaglutide. Liraglutide is administered once daily by subcutaneous injection and is an analog of the native human GLP-1 and is resistant to dipeptidyl peptidase-4 (DDP-4) deactivation [13]. It was shown to reduce HbA1c by 1.12 [13]. Semaglutide is available in both oral and subcutaneous formulations [13]. Semaglutide is similar to liraglutide structurally but has some added resistance to DDP-4 degradation. Overall, semaglutide is better at lowering HbA1c than dulaglutide, liraglutide, and once weekly exenatide [13]. T he subcutaneous

formulation of semaglutide has shown to have the best reduction in HbA1c but the oral semaglutide has shown to provide similar glycemia control [13]. While the oral formulation may be preferred for some patients, the restrictions on medication timing and interactions with other medications limit its effectiveness [13].

Tirzepatide is both a dual GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) receptor agonist (RA) [13]. Glucose-dependent insulinotropic polypeptide (GIP) is an incretin hormone like GLP that stimulates insulin secretion [16]. GIP has been shown in laboratory studies to preserve pancreatic beta cells through anti-apoptotic signaling and a growth factor for insulin-producing beta cells [17]. GIP has also been proposed to have a role in promoting fat accumulation but does not promote food intake [18,19]. Studies have shown that when used together, GLP-1 and GIP RA can demonstrate prominent weight lost and glycemic control [20]. Tirzepatide is a synthetic peptide analog of GIP but has activity at both GLP and GIP receptors, giving it better efficacy in glycemic control [13]. It is formulated to be administered once weekly as a subcutaneous injection and can reduce HbA1c by up to 2.07% when compared to placebo [13]. The dual receptor properties show greater glycemic control than the single receptor agonist available [13]. In a study regarding weight loss in patient, with obesity and diabetes, treatment with tirzepatide resulted in more weight reduction compared to semaglutide [21].

3. Trends in Usage

Obesity has become a global epidemic in recent years and is a major risk factor for numerous diseases. Its development is influenced by a combination of factors, including dietary habits, physical activity levels, socioeconomic status, and stress [22]. Overconsumption of high-processed foods puts patients at a higher risk of developing obesity, diabetes, and hypertension [23]. Given the well-established association between obesity and diabetes mellitus, medications that treat diabetes but also promote weight loss have a therapeutic advantage. GLP-,1 as discussed above, are used to treat type 2 diabetes and often have the beneficial, and desired, side effect of weight loss. Even in patients without diabetes, GLP-1s are able to be useful in weight loss and may help with current obesity pharmacology management. In a study of patients without diabetes, they found a greater than 10% weight loss in 69–69% of patients taking once weekly 2.4 mg of semaglutide subcutaneously for a 68-week trial [24]. GLP-1 receptor agonists have demonstrated efficacy in promoting significant weight loss in non-diabetic patients, with an average reduction of approximately 6 kg compared to placebo when taking once weekly 2.4 mg of semaglutide subcutaneously [25]. This offers a treatment option for obese patients who have not achieved significant weight loss with diet and exercise alone [26]. GLP-1s are now being requested by patients outside of diabetes management in order to achieve weight loss due to the significant reduction in mean body weight that can be achieved [27–29]. The use of GLP-1 has skyrocketed in recent years due to this [30]. A cohort study from the University of California Health Data Warehouse showed that Ozempic users increased from 569 in 2019 to 22,891 users in 2022 [31]. A study looking at prescribing rates in the UK showed an increase of GLP-1 RAs in 2022 and 2023 [32]. In another study with twenty-seven countries, most showed an increasing trend of semaglutide internet searches from January 2021 to August 2023 [30]. Canada and the United States showed the most sustained increase in interest during that time [30]. The searches containing semaglutide in this study had weight loss as a major theme, while diabetes was absent or weak [30]. With the rising popularity of semaglutide for weight loss, the likelihood of seeing rare side effects increases, which makes it essential to provide clinicians with adequate information for counseling patients.

4. Side Effects Associated with GLP-1 RA-Induced Weight Loss

4.1. Gastrointestinal

Gastrointestinal adverse events are common with use of GLP-1 RAs and most commonly include nausea, vomiting, and diarrhea [33]. Which are thought to be caused by delayed gastric emptying or inference of GLP-1 RA with receptors in the area postrema [34]. With the recent popularity of this drug for weight loss, there have been increasing studies on other potential side effects. These include the risk of developing gallbladder or biliary tract diseases such as cholelithiasis, cholecystitis, cholangitis, cholecystectomy, and gallbladder or bile duct obstruction [35]. Although obesity and type 2 diabetes are known risk factors for gallbladder and biliary tract disease, there is a lack of research investigating whether the risk of developing these conditions varies across different obesity profiles in patients using GLP-1 receptor agonists [35]. Randomized clinical trials that compared GLP-1 receptor agonists, such as liraglutide, to placebo for weight management found that there was a higher incidence of acute gallstone conditions such as cholelithiasis and cholecystitis in patients who were treated with liraglutide [35]. It is thought that this connection may be due to the delay in postprandial gallbladder filling caused by liraglutide [36]

4.2. Volume Loss

While gastrointestinal complications are associated with GLP-1 use, weight loss also associated with it can lead to concerns externally, particularly the unflattering cosmetic impact on facial appearance. In the body, GLP-1 RAs lower glucose and reduce total body adiposity from mimicking GLP-1, which allows for rapid weight loss [37]. In a 68-week study, participants using GLP-1 RA were seen to have a 14.9% loss in body weight compared to the placebo group that had a 2.4% loss in body weight [38]. This weight loss causes unintended consequences of subcutaneous fat loss, especially in the face, causing lax and droopy skin. In contrast to gradual weight loss, rapid fat reduction does not allow the skin enough time to retain elasticity and collagen. This process is thought to be multifactorial, involving not only the loss of dermal and subcutaneous adipose tissue but also alterations in adipose-derived stem cell proliferation, metabolic factors, and hormone secretion [39]. In a study by Nogueiras et al., it was suggested that when GLP-1 was infused into the central nervous system there was modulation of adipocyte metabolism which caused decreased fat storage [40]. It was also seen that the inhibition of adipocyte maturation caused a decrease in the activity of fibroblast in regenerating the dermis [41]. GLP-1 RAs also indirectly impact dermal estrogen by decreasing production through a decrease in adipose-derived stem cells [39,42]. In normal aging, decreased estrogen levels, seen in postmenopausal women, contribute significantly to skin aging [43]. This includes thinning of the skin, reduced collagen production, decreased elasticity, dryness, and the formation of wrinkles [43,44]. The critical role of estrogen in skin health is evidenced by studies demonstrating that estrogen replacement therapy in postmenopausal women can reverse many age-related changes, including increased skin thickness and elasticity, enhanced epidermal hydration, and a reduction in wrinkle formation [43,45–49]. This effect has been demonstrated in clinical trials showing that postmenopausal women undergoing hormone replacement therapy exhibited greater skin elasticity, increased water-holding capacity, and thicker skin compared to non-users [50,51].

4.3. Facial Aging

These effects are seen throughout the body; the most notable concern to patients is the appearance of accelerated facial aging, which may have an impact on their self-esteem

and quality of life. Patients may show a gaunt appearance, deepening wrinkles, and loss of facial volume in cheeks and lips, along with other facial structure changes that resemble premature aging [52,53]. Rapid fat volume loss in key facial areas associated with youthfulness contributes to the perception that patients who have undergone massive weight loss appear up to five years older than their age-matched peers [54]. While discontinuing GLP-1 use is an option for patients who worry about the long-term side effects on their skin, the weight gain after stopping does not redistribute to what it was before the medication, resulting in persistent facial aging [55].

4.4. Skin Quality

Beyond fat redistribution, which plays a central role in the development of "Ozempic face," patients also experience a measurable decline in skin quality, further compounding the esthetic impact. In a study by Sami et al., there were skin biopsies taken from three different groups of patients: patients who underwent cosmetic contouring procedures but no history of massive weight loss, patients who were morbidly obese and underwent bariatric surgery, and lastly, patients who had massive weight loss and were undergoing cosmetic contouring procedures after stable weight loss [56]. The study found that collagen was significantly thinner in the groups that had massive weight loss in both the papillary and reticular dermis with significantly less density and damage to the elastic fiber network in the reticular dermis [56]. This study illustrates the underlying cellular changes contributing to the decline in skin quality, which are consistent with the visible physical changes observed in the face. Another study by Rocha et al. compared the collagen and elastic fibers in the abdominal epigastric skin of patients who had undergone massive weight loss following bariatric surgery with those of patients with morbid obesity, aiming to determine whether the weight loss itself contributed to the clinically perceived deterioration in skin quality [57]. In this study there was a reduction of thick collagen fibers, increased thin collagen fibers, and increased elastic fiber density found in the massive weight loss group [57]. There were also alterations in the structure of the dermis and collagenous remodeling, with the reduction of thick, structured, and organized fibers with thin, loose, and misaligned fibers [57]. In an additional study, there was shown to be a decrease in elastic fiber content of the dermis of the abdominal region of patients who underwent surgical massive weight loss compared to those who had non-surgical massive weight loss [58]. Patients using GLP-1 also are at an increased risk of losing nutrients that play an essential role in wound healing [59]. The reduction in essential fatty acids due to decreased nutrient intake may compromise the integrity of the skin barrier, leading to a dull and dry complexion [52,60].

Together, the loss of facial fat, hormonal disruptions, structural skin degradation, and nutritional deficiencies highlight the significant esthetic drawbacks that may outweigh the benefits for individuals using GLP-1 agonists primarily for non-medical, cosmetic weight loss (Figure 1). This off-label use of GLP-1 is steadily increasing, as shown by data from Denmark, where a proportion of semaglutide uses for patients with type 2 diabetes (T2D) declined from 99% in 2018 to 67% in 2023 [61]. This correlates with the increasing popularity of GLP-1 being used among those without diabetes, despite not being approved for weight loss in non-diabetic patients [39]. With this increasing patient population, there is an increased demand for cosmetic treatments to restore facial appearance.

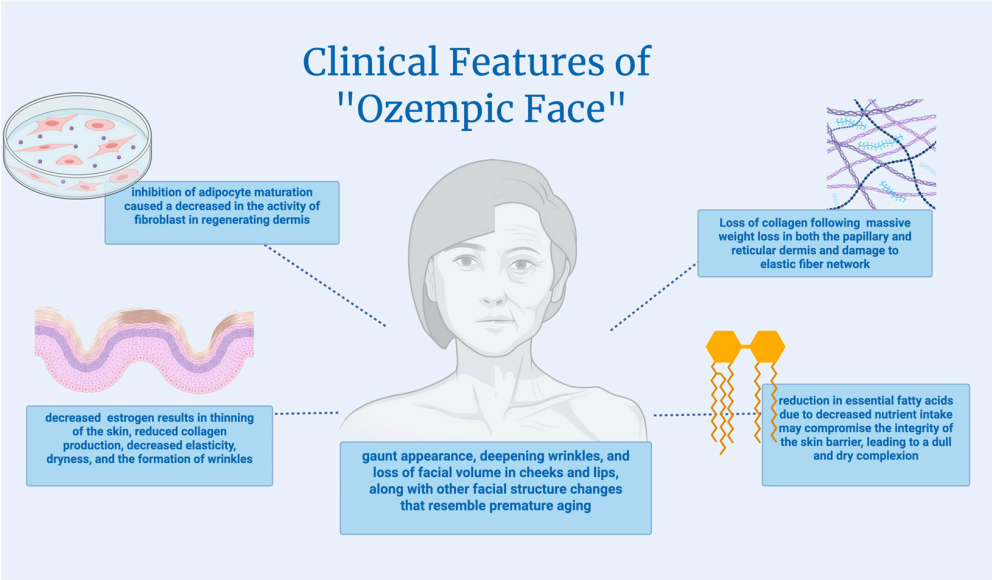

Figure 1. Clinical Features of "Ozempic Face" [62].

5. Management

Although GLP-1s have gained popularity, the esthetic consequences of rapid weight loss with them have not been extensively studied and the esthetic management has not been thoroughly optimized. The first signs of "Ozempic face" may be worsening of existing wrinkles followed by pronounced changes in proportions to the cheeks, lips, and overall facial symmetry [39]. These facial changes may be further exacerbated by the loss of elastin, collagen, fatty acids, and vitamins, accelerating the signs of aging [39]. Current management of these side effects include skin tightening, fat transfer, filler, body contouring, and surgical interventions (Figure 2). Most patients will see the best benefits from a combination of many treatments.

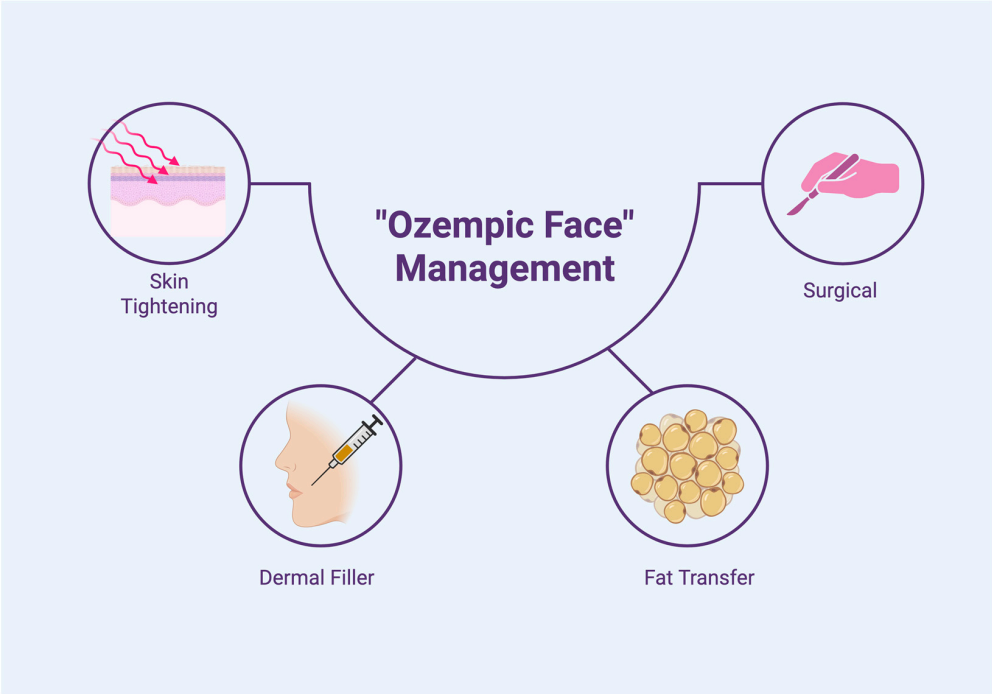

Figure 2. "Ozempic Face" management [63].

5.1. Skin Tightening

Non-invasive procedures for skin tightening include radiofrequency (RF) therapy, ultrasound-based technology, and laser therapy. All report high patient satisfaction but have different indications for use based on severity of skin laxity and where on the body. RF technology creates thermal energy to target dermal layers, inducing tissue injury that triggers a repair response, leading to collagen creation, tissue contraction, and overall skin remodeling [64–69]. Monopolar capacitively couple radiofrequency (MRF) in particular has shown an Investigator Global Aesthetic Improvement Score (IGAIS) of 73%, with subject satisfaction questionnaires supporting these results [65]. Microfocused ultrasounds are also increasing in popularity due to their low recovery time and minimal risk. This ultrasound technology works by stimulating elastin remodeling and collagen creation with the focal heating of the dermis [70]. When comparing the effectiveness of the microfocused ultrasound with reduction in skin laxity, tightening of cheek tissue, and improvement in jawline definition, two-thirds of patients saw an improvement at 90 days [70]. A multimodal device combining radiofrequency (RF), ultrasound, and 808 nm laser energy has shown promise in treating skin laxity across various regions, including the face, neck, arms, and lower limbs [71]. Patients report high satisfaction rates with this combination device, offering an effective and non-invasive solution for improving skin laxity [71].

5.2. Filler

Dermal filler is becoming increasingly popular for the replacement of volume depletion in facial aging. There are several categories of filler such as bovine collagen and hyaluronans. The goal of these procedures is to provide long-lasting, natural-looking, predictable, patient-customized, and biocompatible results, though each treatment type differs in how well it achieves these characteristics [72,73]. When first introduced, hyaluronic acid filler showed a superior result when used on nasolabial folds compared to bovine collagen and started its popularity on the market [74,75]. Hyaluronic acid fillers naturally have a short lifespan in the body; however, when cross-linked with divinyl sulfone, the longevity increases, and studies have shown they elicit minimal immunological reaction [76]. In one study assessing both patient and Investigator Global Aesthetic Improvement Scale (IGAIS) scores, 95% of subjects demonstrated improved full-face IGAIS ratings, and 100% of participants reported satisfaction with their esthetic outcome [77]. Current treatments of facial aging with HA filler have evolved to treat multiple facial regions simultaneously as opposed to isolated areas, aiming to achieve a more harmonious and natural overall esthetic outcome [78–82].

5.3. Fat Transfer

While GLP-1 receptor agonists effectively reduce dermal and subcutaneous adipose tissue, this effect is non-selective and impacts fat stores throughout the body, including the face. Autologous fat transfer can be used to correct the unwanted fat loss in the face. Autologous fat transfer offers a biocompatible alternative to synthetic facial fillers by replenishing lost volume with the patient's own tissue [83]. Because fat is biocompatible, readily accessible, relatively permanent, and able to integrate into surrounding tissues, it serves as an ideal filler [84–86]. Adipose tissue is harvested and prepared with different cannula sizes and filter-cartridge techniques which then divide them based on parcel size and type such as marcofat, microfat, and nanofat [87]. Macrofat and microfat are used to provide volume restoration to areas of facial deflation, and nanofat is used to improve skin texture and pigment [87]. Autologous fat transfer shows high patient satisfaction rates with low complication rates when compared to fillers [88]. In a study that compiled volume retention rate from autologous fat grafting, it showed an average retention rate of 46% with the range being 26 to 83% [89]. This varied response to retention has patients

hesitant about the long-term solution. One study investigated the clinical use of milled electrospun poly(lactic-co-glycolic acid) (PLGA) fibers co-injected with adipose tissue to improve engraftment outcomes. The findings demonstrated enhanced graft volume retention, increased vascularity, and improved tissue reperfusion compared to adipose tissue injections alone [90]. Even though current AFT treatments have various fat retention rates, there is still a high patient satisfaction rate of 91.1% a little over a year following the procedure [91].

GLP-1 RAs also indirectly accelerate facial aging by inhibiting adipose-derived stem cell proliferation, which plays a key role in skin regeneration and repair [39]. By stimulating adipose-derived stem cell proliferation, you may be able to attenuate these side effects to improve facial alterations with GLP-1 RA therapy and improve skin quality [39]. Intradermal injections of purified autologous processed lipoaspirate cells, which contain approximately 30% adipose-derived stem cells (ADSCs), have been shown to enhance skin quality and restore volume by promoting collagen production and releasing growth factors that counteract signs of skin aging [92]. These cells are also reported to be able to control and manage the damage of neighboring cells, helping restore surrounding skin [93].

5.4. Surgical Interventions

Patients also have the option for surgical intervention to correct skin laxity following weight loss. The most common procedures underwent are liposuction, abdominoplasty, body lift, brachioplasty, and facelift [94,95]. With these options available, each patient can undergo a combination of each and often require staging of their surgeries for optimal outcomes [96].

Liposuction is commonly used in adjunction to body-contouring procedures. It may be performed preoperatively to refine regional contour and is associated with improved overall patient satisfaction [97].

Abdominoplasty is the surgical removal of excess skin off the adnominal wall [98]. It also comes with its own set of complications such as wound infection, seroma, hematoma, deep vein thrombosis, and pulmonary embolism [98].

A total body lift surgery encompasses three portions: an upper body lift (UBL), low body lift (LBL), and buttock augmentation [99]. The UBL leaves a transverse scar across the bra line and allows for breast reshaping [99]. The LBL has a circumferential hip transverse incision that allows for a reduction in loose skin with the addition of buttock augmentation if necessary [99].

Brachioplasty is a procedure that improves arm deformity that can be caused by major weight loss, such as excess skin in a canopy-like shape, draping from the axilla to the elbow [100]. This appearance is distressing for patients and often requires correction. A brachioplasty removes the excess tissue and can leave the patient with several complications including nerve damage, lymphatic issues, and an unappealing scar [100].

Major weight loss often results in increased skin laxity and excess skin on the face. A facelift offers patients the opportunity to rejuvenate their facial appearance and can also include neck lifting [95]. This procedure removes excess skin and addresses facial and neck deflation to better define the facial contours [95]. Surgeons may also combine the facelift with fat grafting to restore volume to the face. However, like any surgery, it carries risks such as seromas, hematomas, and soft tissue deflation [95].

Patients undergoing these procedures following major weight loss experience significant improvements in mental health, which positively impact multiple aspects of their lives [101]. These benefits include increased physical activity levels, improved social relationships, and increased work performance. These improvements highlight the psychosocial improvements in quality of life that body contouring surgery provides beyond

esthetic outcomes [102]. Although many patients would benefit from body contouring surgery, there are many factors in play that restrict access to these surgeries. Many patients are not able to afford these surgeries; some do not have insurance or may have only partial insurance coverage [103].

5.5. Current Management

Current measures available include a combination of dermal fillers, autologous fat grafting, facelift procedures, and potential nutrition optimization to limit effects [39]. Dermal fillers are a popular option due to their minimally invasive procedures and immediate results. However, their long-term interaction with GLP-1 therapy and durability of results has not been extensively reviewed. Hyaluronic acid filler requires maintenance every 6–18 months, which is not possible for all patients. For those who want longer lasting results, autologous fat grafting may be an option. Autologous fat grafting refers to harvesting fat from the same person who will receive it, offering a natural and more permanent solution [43]. Given it is derived from the patient, there is minimal risk of allergic reaction, and it produces a more natural and permanent result compared to dermal filler. While both filler and autologous fat grafting effectively restore facial volume, a facelift, or rhytidectomy with or without platysmaplasty, procedure may be appropriate in more severe cases of skin laxity.

These common side effects raise an important clinical and ethical question of whether rapid weight loss is worth the facial consequences, especially for patients utilizing the medication for cosmetic weight. To answer this question, we need a deeper understanding of the long-term esthetic impact of GLP-1 therapy and what therapies work for the best outcomes. Additionally, there is a growing need for appropriate patient counseling on these side effects as well as standardization of cosmetic correction.

6. Conclusions

While morphologic changes happen diffusely across the body with semaglutide use, "Ozempic face" is the most distressing to patients and has become a real and increasingly observed side effect of this popular weight loss medication. For clinicians to properly prescribe GLP-1 for weight loss, they need to counsel the patient on the potential facial esthetic repercussions and be aware of available strategies to improve the appearance of the face after treatment. Ultimately, there is a need for more studies on long-term esthetic outcomes and optimal strategies for prevention or correction.

Author Contributions: A.B.: Conceptualization, methodology, formal analysis, resources, data curation, writing—original draft preparation, writing—review and editing, visualization. C.S.: Conceptualization, methodology, formal analysis, resources, data curation, writing—original draft preparation, writing—review and editing, visualization. R.S.: Conceptualization, methodology, formal analysis, resources, data curation, writing—review and editing, supervision, project administration. V.P.: Conceptualization, methodology, formal analysis, resources, data curation, writing—review and editing, supervision, project administration. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new data was created during this study.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

GLP-1	Glucose-like protein-1
GLP-1 RA	Glucose-like protein-1 receptor agonist
PPG	Preproglucagon (PPG)
DDP-4	Dipeptidyl peptidase-4
GIP	Glucose- dependent insulinotropic polypeptide
T2D	Type 2 diabetes
MRF	Monopolar capacitively couple radiofrequency
IGAIS	Investigator Global Aesthetic Improvement Scale
PLGA	Poly(lactic-co-glycolic acid
ADSC	Adipose-derived stem cell
UPL	Upper body lift
LBL	Lower body lift

References

1. Lin, X.; Li, H. Obesity: Epidemiology, Pathophysiology, and Therapeutics. *Front. Endocrinol.* **2021**, *12*, 706978. [CrossRef]
2. Haslam, D. Weight management in obesity—Past and present. *Int. J. Clin. Pract.* **2016**, *70*, 206–217. [CrossRef]
3. Mariam, Z.; Niazi, S.K. Glucagon-like peptide agonists: A prospective review. *Endocrinol. Diabetes Metab.* **2023**, *7*, e462. [CrossRef]
4. Hayashi, Y. Glucagon regulates lipolysis and fatty acid oxidation through inositol triphosphate receptor 1 in the liver. *J. Diabetes Investig.* **2020**, *12*, 32–34. [CrossRef] [PubMed]
5. Sadeghi, A.; Niknam, M.; Momeni-Moghaddam, M.A.; Shabani, M.; Aria, H.; Bastin, A.; Teimouri, M.; Meshkani, R.; Akbari, H. Crosstalk between autophagy and insulin resistance: Evidence from different tissues. *Eur. J. Med. Res.* **2023**, *28*, 456. [CrossRef] [PubMed]
6. Conceição-Furber, E.; Coskun, T.; Sloop, K.W.; Samms, R.J. Is Glucagon Receptor Activation the Thermogenic Solution for Treating Obesity? *Front. Endocrinol.* **2022**, *13*, 868037. [CrossRef]
7. Zeigerer, A.; Sekar, R.; Kleinert, M.; Nason, S.; Habegger, K.M.; Müller, T.D. Glucagon's Metabolic Action in Health and Disease. *Compr. Physiol.* **2021**, *11*, 1759. [CrossRef] [PubMed]
8. Drucker, D.J.; Nauck, M.A. The incretin system: Glucagon-like peptide-1 receptor agonists and dipeptidyl peptidase-4 inhibitors in type 2 diabetes. *Lancet* **2006**, *368*, 1696–1705. [CrossRef]
9. Liu, Q.K. Mechanisms of action and therapeutic applications of GLP-1 and dual GIP/GLP-1 receptor agonists. *Front. Endocrinol.* **2024**, *15*, 1431292. [CrossRef]
10. Cornell, S. A review of GLP-1 receptor agonists in type 2 diabetes: A focus on the mechanism of action of once-weekly agents. *J. Clin. Pharm. Ther.* **2020**, *45* (Suppl. S1), 17–27. [CrossRef]
11. Müller, T.D.; Finan, B.; Bloom, S.; D'ALessio, D.; Drucker, D.; Flatt, P.; Fritsche, A.; Gribble, F.; Grill, H.; Habener, J.; et al. Glucagon-like peptide 1 (GLP-1). *Mol. Metab.* **2019**, *30*, 72–130. [CrossRef]
12. Diz-Chaves, Y.; Mastoor, Z.; Spuch, C.; González-Matías, L.C.; Mallo, F. Anti-Inflammatory Effects of GLP-1 Receptor Activation in the Brain in Neurodegenerative Diseases. *Int. J. Mol. Sci.* **2022**, *23*, 9583. [CrossRef]
13. Alfaris, N.; Waldrop, S.; Johnson, V.; Boaventura, B.; Kendrick, K.; Stanford, F.C. GLP-1 single, dual, and triple receptor agonists for treating type 2 diabetes and obesity: A narrative review. *eClinicalMedicine* **2024**, *75*, 102782. [CrossRef]
14. Müller, T.D.; Clemmensen, C.; Finan, B.; Dimarchi, R.D.; Tschöp, M.H. Anti-obesity therapy: From rain-bow pills to polyagonists. *Pharmacol. Rev.* **2018**, *70*, 712–746. [CrossRef]
15. Turkistani, Y. Glucagon-like peptide-1 receptor agonists: A review from a cardiovascular perspective. *Front. Cardiovasc. Med.* **2025**, *12*, 1535134. [CrossRef]
16. Bull, S.T.; Nuffer, W.; Trujillo, J.M. Tirzepatide: A novel, first-in-class, dual GIP/GLP-1 receptor agonist. *J. Diabetes Its Complicat.* **2022**, *36*, 108332. [CrossRef]
17. Trumper, A.; Trumper, K.; Horsch, D. Mechanisms of mitogenic and anti-apoptotic signaling by glucose-dependent insulinotropic polypeptide in beta(INS-1)-cells. *J. Endocrinol.* **2002**, *174*, 233–246. [CrossRef] [PubMed]
18. Seino, Y.; Fukushima, M.; Yabe, D. GIP and GLP-1, the two incretin hormones: Similarities and differences. *J. Diabetes Investig.* **2010**, *1*, 8–23. [CrossRef] [PubMed]
19. Fukuda, M. The Role of GIP Receptor in the CNS for the Pathogenesis of Obesity. *Diabetes* **2021**, *70*, 1929–1937. [CrossRef]
20. Holst, J.J.; Rosenkilde, M.M. GIP as a Therapeutic Target in Diabetes and Obesity: Insight from Incretin Co-agonists. *J. Clin. Endocrinol. Metab.* **2020**, *105*, e2710–e2716. [CrossRef] [PubMed]

21. Aronne, L.J.; Horn, D.B.; le Roux, C.W.; Ho, W.; Falcon, B.L.; Valderas, E.G.; Das, S.; Lee, C.J.; Glass, L.C.; Senyucel, C.; et al. Tirzepatide as Compared with Semaglutide for the Treatment of Obesity. *N. Engl. J. Med.* **2025**, *393*, 26–36. [CrossRef]
22. Ueda Yamaguchi, N.; de Almeida, L.; Carvalho Gomes Corrêa, R.; Grossi Milani, R.; Ueda Yamaguchi, M. Global Perspectives on Obesity and Being Overweight: A Bibliometric Analysis in Relation to Sustainable Development Goals. *Int. J. Environ. Res. Public Health* **2025**, *22*, 146. [CrossRef]
23. Nardocci, M.; Polsky, J.Y.; Moubarac, J.-C. Consumption of ultra-processed foods is associated with obesity, diabetes and hypertension in Canadian adults. *Can. J. Public Health* **2020**, *112*, 421–429. [CrossRef]
24. Bergmann, N.C.; Davies, M.J.; Lingvay, I.; Knop, F.K. Semaglutide for the treatment of overweight and obesity: A review. *Diabetes Obes. Metab.* **2022**, *25*, 18–35. [CrossRef]
25. Vahora, I.; Moparthi, K.P.; Al Rushaidi, M.T.; Muddam, M.R.; Obajeun, O.A.; Abaza, A.; Jaramillo, A.P.; Idris, F.S.; Shaikh, H.A.; Mohammed, L. Efficacy of Glucagon-Like Peptide 1 (GLP-1) Receptor Agonists for Weight Loss Management in Non-Diabetic Patients. *Cureus* **2024**, *16*, e65050. [CrossRef] [PubMed]
26. Moiz, A.; Filion, K.B.; Tsoukas, M.A.; Yu, O.H.; Peters, T.M.; Eisenberg, M.J. Mechanisms of GLP-1 Receptor Agonist-Induced Weight Loss: A Review of Central and Peripheral Pathways in Appetite and Energy Regulation. *Am. J. Med.* **2025**, *138*, 934–940. [CrossRef] [PubMed]
27. Arastu, N.; Cummins, O.; Uribe, W.; Nemec, E.C. Efficacy of subcutaneous semaglutide compared to placebo for weight loss in obese, non-diabetic adults: A systematic review & meta-analysis. *Int. J. Clin. Pharm.* **2022**, *44*, 852–859. [CrossRef]
28. Wang, J.-Y.; Wang, Q.-W.; Yang, X.-Y.; Yang, W.; Li, D.-R.; Jin, J.-Y.; Zhang, H.-C.; Zhang, X.-F. GLP−1 receptor agonists for the treatment of obesity: Role as a promising approach. *Front. Endocrinol.* **2023**, *14*, 1085799. [CrossRef] [PubMed]
29. Ansari, H.U.H.; Qazi, S.U.; Sajid, F.; Altaf, Z.; Ghazanfar, S.; Naveed, N.; Ashfaq, A.S.; Siddiqui, A.H.; Iqbal, H.; Qazi, S. Efficacy and Safety of Glucagon-Like Peptide-1 Receptor Agonists on Body Weight and Cardiometabolic Parameters in Individuals With Obesity and Without Diabetes: A Systematic Review and Meta-Analysis. *Endocr. Pract.* **2024**, *30*, 160–171. [CrossRef]
30. Mahase, E. GLP-1 agonists: US sees 700% increase over four years in number of patients without diabetes starting treatment. *BMJ* **2024**, *386*, q1645. [CrossRef]
31. Watanabe, J.H.; Kwon, J.; Nan, B.; Reikes, A. Trends in glucagon-like peptide 1 receptor agonist use, 2014 to 2022. *J. Am. Pharm. Assoc.* **2023**, *64*, 133–138. [CrossRef]
32. Ibrahim, A.R.N.; Orayj, K.M. Impact of ADA Guidelines and Medication Shortage on GLP-1 Receptor Agonists Prescribing Trends in the UK: A Time-Series Analysis with Country-Specific Insights. *J. Clin. Med.* **2024**, *13*, 6256. [CrossRef] [PubMed]
33. Wharton, S.; Calanna, S.; Davies, M.; Dicker, D.; Goldman, B.; Lingvay, I.; Mosenzon, O.; Rubino, D.M.; Thomsen, M.; Wadden, T.A.; et al. Gastrointestinal tolerability of once-weekly semaglutide 2.4 mg in adults with overweight or obesity, and the relationship between gastrointestinal adverse events and weight loss. *Diabetes Obes. Metab.* **2021**, *24*, 94–105. [CrossRef]
34. Holst, J.J.; Andersen, D.B.; Grunddal, K.V. Actions of glucagon-like peptide-1 receptor ligands in the gut. *Br. J. Pharmacol.* **2022**, *179*, 727–742. [CrossRef] [PubMed]
35. Ko, H.Y.; Bea, S.; Yoon, D.; Hong, B.; Bae, J.H.; Cho, Y.M.; Shin, J.-Y. Incretin-based drugs and the risk of gallbladder or biliary tract diseases among patients with type 2 diabetes across categories of body mass index: A nationwide cohort study. *Lancet Reg. Health West. Pac.* **2025**, *56*, 101242. [CrossRef] [PubMed]
36. Nerild, H.H.; Brønden, A.; Gether, I.M.; Hellmann, P.H.; Baekdal, M.; Gillum, M.P.; Svenningsen, J.S.; Hartmann, B.; Rathor, N.; Angelene, H.; et al. Liraglutide changes postprandial responses of gut hormones involved in the regulation of gallbladder motility. *Diabetes Obes. Metab.* **2023**, *25*, 1632–1637. [CrossRef]
37. Chung, L.T.K.; Hosaka, T.; Yoshida, M.; Harada, N.; Sakaue, H.; Sakai, T.; Nakaya, Y. Exendin-4, a GLP-1 receptor agonist, directly induces adiponectin expression through protein kinase A pathway and prevents inflammatory adipokine expression. *Biochem. Biophys. Res. Commun.* **2009**, *390*, 613–618. [CrossRef]
38. Wilding, J.P.H.; Batterham, R.L.; Calanna, S.; Davies, M.; Van Gaal, L.F.; Lingvay, I.; McGowan, B.M.; Rosenstock, J.; Tran, M.T.; Wadden, T.A.; et al. Once-Weekly Semaglutide in Adults with Overweight or Obesity. *N. Engl. J. Med.* **2021**, *384*, 989–1002. [CrossRef]
39. Ridha, Z.; Fabi, S.G.; Zubair, R.; Dayan, S.H. Decoding the Implications of Glucagon-like Peptide-1 Receptor Agonists on Accelerated Facial and Skin Aging. *Aesthetic Surg. J.* **2024**, *44*, NP809–NP818. [CrossRef]
40. Nogueiras, R.; Pérez-Tilve, D.; Veyrat-Durebex, C.; Morgan, D.A.; Varela, L.; Haynes, W.G.; Patterson, J.T.; Disse, E.; Pfluger, P.T.; López, M.; et al. Direct Control of Peripheral Lipid Deposition by CNS GLP-1 Receptor Signaling Is Mediated by the Sympathetic Nervous System and Blunted in Diet-Induced Obesity. *J. Neurosci.* **2009**, *29*, 5916–5925. [CrossRef]
41. Schmidt, B.A.; Horsley, V. Intradermal adipocytes mediate fibroblast recruitment during skin wound healing. *Development* **2013**, *140*, 1517–1527. [CrossRef]
42. Steiner, B.M.; Berry, D.C. The Regulation of Adipose Tissue Health by Estrogens. *Front. Endocrinol.* **2022**, *13*, 889923. [CrossRef]
43. Thornton, M.J. Estrogens and aging skin. *Derm.-Endocrinol.* **2013**, *5*, 264–270. [CrossRef]
44. Bolognia, J.; Braverman, I.; Rousseau, M.; Sarrel, P. Skin changes in menopause. *Maturitas* **1989**, *11*, 295–304. [CrossRef] [PubMed]

45. Stevenson, S.; Thornton, J. Effect of estrogens on skin aging and the potential role of SERMs. *Clin. Interv. Aging* **2007**, *2*, 283–297. [CrossRef]
46. Thornton, M.J. Oestrogen functions in skin and skin appendages. *Expert Opin. Ther. Targets* **2005**, *9*, 617–629. [CrossRef] [PubMed]
47. Thornton, M.J. The biological actions of estrogens on skin. *Exp. Dermatol.* **2002**, *11*, 487–502. [CrossRef] [PubMed]
48. Piérard-Franchimont, C.; Cornil, F.; Dehavay, J.; Deleixhe-Mauhin, F.; Letot, B.; Piérard, G. Climacteric skin ageing of the face—A prospective longitudinal comparative trial on the effect of oral hormone replacement therapy. *Maturitas* **1999**, *32*, 87–93. [CrossRef]
49. Grosman, N.; Hvidberg, E.; Schou, J. The Effect of Oestrogenic Treatment on the Acid Mucopolysaccharide Pattern in Skin of Mice. *Acta Pharmacol. Toxicol.* **1972**, *30*, 458–464. [CrossRef]
50. Brincat, M.; Baron, Y.M.; Galea, R. Estrogens and the skin. *Climacteric* **2005**, *8*, 110–123. [CrossRef]
51. Pivazyan, L.; Avetisyan, J.; Loshkareva, M.; Abdurakhmanova, A. Skin Rejuvenation in Women using Menopausal Hormone Therapy: A Systematic Review and Meta-Analysis. *J. Menopausal Med.* **2023**, *29*, 97–111. [CrossRef]
52. O'Neill, E.S.; Wiegmann, A.L.; Parrella, N.; Pittman, T.; Hood, K.; Kurlander, D. Injectable Weight Loss Medications in Plastic Surgery: What We Know, Perioperative Considerations, and Recommendations for the Future. *Plast. Reconstr. Surg. Glob. Open* **2024**, *12*, e5516. [CrossRef]
53. Lambros, V. Val Facial Aging: A 54-Year, Three-Dimensional Population Study. *Plast. Reconstr. Surg.* **2020**, *145*, 921–928. [CrossRef]
54. Couto, R.A.; Waltzman, J.T.; Tadisina, K.K.; Rueda, S.; Richards, B.G.; Schleicher, W.F.; Marten, E.; Larson, J.D.; Rotemberg, S.C.; Zins, J.E. Objective Assessment of Facial Rejuvenation After Massive Weight Loss. *Aesthetic Plast. Surg.* **2015**, *39*, 847–855. [CrossRef]
55. Humphrey, C.D.; Lawrence, A.C. Implications of Ozempic and Other Semaglutide Medications for Facial Plastic Surgeons. *Facial Plast. Surg.* **2023**, *39*, 719–721. [CrossRef] [PubMed]
56. Sami, K.; Elshahat, A.; Moussa, M.; Abbas, A.; Mahmoud, A. Image analyzer study of the skin in patients with morbid obesity and massive weight loss. *Eplasty* **2015**, *15*, e4. [PubMed]
57. Rocha, R.I.; Junior, W.C.; Modolin, M.L.A.; Takahashi, G.G.; Caldini, E.T.E.G.; Gemperli, R. Skin Changes Due to Massive Weight Loss: Histological Changes and the Causes of the Limited Results of Contouring Surgeries. *Obes. Surg.* **2021**, *31*, 1505–1513. [CrossRef] [PubMed]
58. Hany, M.; Zidan, A.; Ghozlan, N.A.; Ghozlan, M.N.; Abouelnasr, A.A.; Sheta, E.; Hamed, Y.; Kholosy, H.; Soffar, M.; El Midany, W.M.; et al. Comparison of Histological Skin Changes After Massive Weight Loss in Post-bariatric and Non-bariatric Patients. *Obes. Surg.* **2024**, *34*, 855–865. [CrossRef]
59. Taraschi, F.; Salgarello, M. GLP-1 Agonists in Plastic Surgery: Impact on Aesthetic Outcomes—Two Case Reports. *Aesthetic Plast. Surg.* **2025**, *49*, 4527–4530. [CrossRef]
60. Draelos, Z.; Baalbaki, N.; Cook, S.; Raab, S.; Colón, G. The effect of a ceramide-containing product on stratum corneum lipid levels in dry legs. *J. Drugs Dermatol.* **2020**, *19*, 372–376. [CrossRef]
61. Mailhac, A.; Pedersen, L.; Pottegård, A.; Søndergaard, J.; Mogensen, T.; Sørensen, H.T.; Thomsen, R.W. Semaglutide (Ozempic®) Use in Denmark 2018 Through 2023—User Trends and off-Label Prescribing for Weight Loss. *Clin. Epidemiol.* **2024**, *16*, 307–318. [CrossRef]
62. Baggett, A. Clinical Features of Ozempic Face | BioRender [Internet]. 2025. Available online: https://app.biorender.com/citation/68a297146460b30eca97e2f9 (accessed on 28 September 2025).
63. Baggett, A. "Ozempic Face" Management | BioRender [Internet]. 2025. Available online: https://app.biorender.com/citation/68a2982d948430ca9ae6cf47 (accessed on 28 September 2025).
64. Narins, D.J.; Narins, R.S. Non-Surgical Radiofrequency Facelift. *J. Drugs Dermatol.* **2003**, *2*, 495–500.
65. Hantash, B.M.; Renton, B.; Berkowitz, R.L.; Stridde, B.C.; Newman, J. Pilot clinical study of a novel minimally invasive bipolar microneedle radiofrequency device. *Lasers Surg. Med.* **2009**, *41*, 87–95. [CrossRef]
66. Bassichis, B.A.; Dayan, S.; Thomas, J.R. Use of a nonablative radiofrequency device to rejuvenate the upper one-third of the face. *Otolaryngol. Head Neck Surg.* **2004**, *130*, 397–406. [CrossRef] [PubMed]
67. Abraham, M.; Chiang, S.; Keller, G.; Rawnsley, J.; Blackwell, K.; Elashoff, D. Clinical evaluation of non-ablative radiofrequency facial rejuvenation. *J. Cosmet. Laser Ther.* **2004**, *6*, 136–144. [CrossRef] [PubMed]
68. Austin, G.K.; Struble, S.L.; Quatela, V.C. Evaluating the effectiveness and safety of radiofrequency for face and neck rejuvenation: A systematic review. *Lasers Surg. Med.* **2022**, *54*, 27–45. [CrossRef]
69. Migliardi, R.; Tofani, F.; Donati, L. Non-Invasive Peri-Orbital Rejuvenation: Radiofrequency Dual Radiowave Energy Source (RF) and Light Emission Diode System (LED). *Orbit* **2009**, *28*, 214–218. [CrossRef] [PubMed]
70. Oni, G.; Hoxworth, R.; Teotia, S.; Brown, S.; Kenkel, J.M. Evaluation of a microfocused ultrasound system for improving skin laxity and tightening in the lower face. *Aesthetic Surg. J.* **2014**, *34*, 1099–1110. [CrossRef]
71. Scarcella, G.; Pennati, B.M. A temperature-controlled radio frequency combined with ultrasounds and laser for skin laxity and cellulitis. *SAGE Open Med. Case Rep.* **2024**, *12*, 2050313X241230444. [CrossRef]
72. Newman, J. Review of soft tissue augmentation in the face. *Clin. Cosmet. Investig. Dermatol. CCID* **2009**, *2*, 141–150. [CrossRef]

73. Siperstein, R.; Nestor, E.; Meran, S.; Grunebaum, L. Asplit-face, blind, randomized placebo-controlled clinical trial investigating the efficacy and safety of hyaluronic acid filler for the correction of atrophic facial scars. *J. Cosmet. Dermatol.* **2022**, *21*, 3768–3778. [CrossRef]
74. Narins, R.S.; Brandt, F.; Leyden, J.; Lorenc, Z.P.; Rubin, M.; Smith, S. A randomized, double-blind, multi-center comparison of the efficacy and tolerability of restylane versus zyplast for the correction of nasolabial folds. *Dermatol. Surg.* **2003**, *29*, 588–595. [CrossRef]
75. Lheritier, C.M.; Converset, S.; Rzany, B.-J.; Cartier, H.; Ascher, B. Efficacy of a New Hyaluronic Acid Dermal Filler on Nasolabial Folds Correction: A Prospective, Comparative, Double-Blinded Clinical Trial. *Dermatol. Surg.* **2024**, *50*, 746–751. [CrossRef]
76. Larsen, N.E.; Pollak, C.T.; Reiner, K.; Leshchiner, E.; Balazs, E.A. Hylan gel biomaterial: Dermal and immunologic compatibility. *J. Biomed. Mater. Res.* **1993**, *27*, 1129–1134. [CrossRef]
77. Talarico, S.; Meski, A.P.; Buratini, L.; Manela-Azulay, M.; Simpson, H.; Sidou, F.; Kerrouche, N. High patient satisfaction of a hyaluronic acid filler producing enduring full-facial volume restoration: An 18-month open multicenter study. *Dermatol. Surg.* **2015**, *41*, 1361–1369. [CrossRef] [PubMed]
78. Delorenzi, C.; Weinberg, M.; Solish, N.; Swift, A. The long-term efficacy and safety of a subcutaneously injected large-particle stabilized hyaluronic acid–based gel of nonanimal origin in esthetic facial contouring. *Dermatol. Surg.* **2009**, *35*, 313–321. [CrossRef] [PubMed]
79. Callan, P.; Halstead, M.B.; Rogers, J.D.; Goodman, G.J.; Liew, S.; Muzikants, P.; Scamp, T.; Carlisle, I. Efficacy and safety of a hyaluronic acid filler in subjects treated for correction of midface volume deficiency: A 24 month study. *Clin. Cosmet. Investig. Dermatol.* **2013**, *6*, 81–89. [CrossRef]
80. Rzany, B.; Cartier, H.; Kestemont, P.; Trevidic, P.; Sattler, G.; Kerrouche, N.; Dhuin, J.-C.; Ma, M.Y. Full-face rejuvenation using a range of hyaluronic acid fillers: Efficacy, safety, and patient satisfaction over 6 months. *Dermatol. Surg.* **2012**, *38*, 1153–1161. [CrossRef] [PubMed]
81. Brandt, F.S.; Cazzaniga, A. Hyaluronic acid gel fillers in the management of facial aging. *Clin. Interv. Aging* **2008**, *3*, 153–159. [CrossRef]
82. Farhi, D.; Trevidic, P.; Kestemont, P.; Boineau, D.; Cartier, H.; Bodokh, I.; Brun, P.; Ascher, A.; Savary, J.; for the Emervel French Survey Group. The Emervel French Survey: A Prospective Real-Practice Descriptive Study of 1,822 Patients Treated for Facial Rejuvenation with a New Hyaluronic Acid Filler. *J. Drugs Dermatol. JDD* **2013**, *12*, e88–e93.
83. Scarborough, D.A.; Schuen, W.; Bisaccia, E. Fat Transfer for Aging Skin: Technique for Rhytids. *Dermatol. Surg. Oncol.* **1990**, *16*, 651–655. [CrossRef]
84. Coleman, S.R. Structural Fat Grafts. *Clin. Plast. Surg.* **2001**, *28*, 111–119. [CrossRef]
85. Coleman, S.R. Long-Term survival of fat transplants: Controlled demonstrations. *Aesthetic Plast. Surg.* **1995**, *19*, 421–425. [CrossRef]
86. Gornitsky, J.; Viezel-Mathieu, A.; Alnaif, N.; Azzi, A.J.; Gilardino, M.S. A systematic review of the effectiveness and complications of fat grafting in the facial region. *JPRAS Open* **2019**, *19*, 87–97. [CrossRef]
87. Strong, A.L.; Rohrich, R.J.; Tonnard, P.L.; Vargo, J.D.; Cederna, P.S. Technical Precision with Autologous Fat Grafting for Facial Rejuvenation: A Review of the Evolving Science. *Plast. Reconstr. Surg.* **2024**, *153*, 360–377. [CrossRef]
88. Groen, J.-W.; Krastev, T.K.; Hommes, J.; Wilschut, J.A.; Ritt, M.J.P.F.; van der Hulst, R.R.J.W. Autologous Fat Transfer for Facial Rejuvenation: A Systematic Review on Technique, Efficacy, and Satisfaction. *Plast. Reconstr. Surg. Glob. Open* **2017**, *5*, e1606. [CrossRef]
89. Lv, Q.; Li, X.; Qi, Y.; Gu, Y.; Liu, Z.; Ma, G.-E. Volume Retention After Facial Fat Grafting and Relevant Factors: A Systematic Review and Meta-analysis. *Aesthetic Plast. Surg.* **2021**, *45*, 506–520. [CrossRef] [PubMed]
90. Das, D.; Lawrence, W.R.; Diaz-Starokozheva, L.; Salazar-Puerta, A.I.; Ott, N.; Goebel, E.R.; Damughatla, A.; Vidal, P.; Gallentine, S.; Moore, J.T.; et al. Injectable pulverized electrospun poly(lactic-co-glycolic acid) fibers improve human adipose tissue engraftment and volume retention. *J. Biomed. Mater. Res. Part A* **2023**, *111*, 1722–1733. [CrossRef] [PubMed]
91. Krastev, T.K.; Beugels, J.; Hommes, J.; Piatkowski, A.; Mathijssen, I.; Van Der Hulst, R. Efficacy and safety of autologous fat transfer in facial reconstructive surgery a systematic review and meta-analysis. *JAMA Facial Plast. Surg.* **2018**, *20*, 351–360. [CrossRef] [PubMed]
92. Park, B.S.; Jang, K.A.; Sung, J.H.; Park, J.S.; Kwon, Y.H.; Kim, K.J.; Kim, W.S. Adipose-derived stem cells and their secretory factors as a promising therapy for skin aging. *Dermatol. Surg.* **2008**, *34*, 1323–1326. [CrossRef]
93. Kim, W.-S.; Park, B.-S.; Sung, J.-H. Protective role of adipose-derived stem cells and their soluble factors in photoaging. *Arch. Dermatol. Res.* **2009**, *301*, 329–336. [CrossRef]
94. Tremp, M.; Delko, T.; Kraljević, M.; Zingg, U.; Rieger, U.M.; Haug, M.; Kalbermatten, D.F. Outcome in body-contouring surgery after massive weight loss: A prospective matched single-blind study. *J. Plast. Reconstr. Aesthetic Surg.* **2015**, *68*, 1410–1416. [CrossRef]

95. Handal, M.; Handal, J.; Finkelstein, P.; Kichler, K.; Nevill, T.J. Cosmetic Procedures After Massive Weight Loss Surgery: A Guide for Prospective Patients. *Cureus* **2024**, *16*, e72864. [CrossRef]
96. Giordano, S.; Salval, A.; di Summa, P.; Oranges, C.M. Evolving Body Contouring Strategies for Patients After Massive Weight Loss: Insights from Bariatric and Pharmacologic Interventions. *Surgeries* **2025**, *6*, 42. [CrossRef]
97. Gunnarsson, G.L.; Gudjonsdottir, L.R.B.; Koidil, A.; Haukeland, L.; Berg, E.; Thomsen, J.B. Efficacy of Liposuction of the Posterior Arm Fat Pad in Axillobrachioplasty after Massive Weigh Loss: A Pilot Study. *Plast. Reconstr. Surg. Glob. Open* **2024**, *12*, e6251. [CrossRef]
98. Cannistrà, C.; Lori, E.; Arapis, K.; Gallo, G.; Varanese, M.; Pironi, D.; De Luca, A.; Frusone, F.; Amabile, M.I.; Sorrenti, S.; et al. Abdominoplasty after massive weight loss. Safety preservation fascia technique and clinical outcomes in a large single series-comparative study. *Front. Surg.* **2024**, *11*, 1337948. [CrossRef] [PubMed]
99. Hurwitz, D.J.; Davila, A.A. Update on Oblique Flankplasty: Easily Executed, Long-Lasting, Integral Component of Total Body Lift Surgery. *Aesthetic Surg. J.* **2023**, *44*, NP77–NP86. [CrossRef] [PubMed]
100. Elsaka, D.M.M.; Nassar, A.T.; Abdel-Wahab, M.M.; Samy, M. Post Massive Weight Loss Brachioplasty Aesthetic Outcome and Complications. *Egypt. J. Hosp. Med.* **2023**, *92*, 2653. [CrossRef]
101. Cuomo, R.; Cuccaro, C.; Seth, I.; Rozen, W.M.; Vastarella, M.G.; Lombardo, G.A.G.; Ciancio, F.; Pagliara, D.; Pieretti, G.; Ciccarelli, F. Experience in Post-Bariatric Abdominoplasty for Patients with Significant Weight Loss: A Prospective Study. *J. Pers. Med.* **2024**, *14*, 681. [CrossRef]
102. Berkane, Y.M.; Saget, F.M.; Lupon, E.M.; Mocquard, C.M.; Pluvy, I.M.; Watier, E.; Lellouch, A.G.; Duisit, J.M.; Chaput, B.; Bertheuil, N. Abdominoplasty and Lower Body Lift Surgery Improves the Quality of Life after Massive Weight Loss: A Prospective Multicenter Study. *Plast. Reconstr. Surg.* **2024**, *153*, 1101E–1110E. [CrossRef]
103. Jiang, Z.; Zhang, G.; Huang, J.; Shen, C.; Cai, Z.; Yin, X.; Yin, Y.; Zhang, B. A systematic review of body contouring surgery in post-bariatric patients to determine its prevalence, effects on quality of life, desire, and barriers. *Obes. Rev.* **2021**, *22*, e13201. [CrossRef] [PubMed]