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Semaglutide and the Skin: An Overview of Current Evidence

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ABSTRACT

Background. Semaglutide is a GLP-1 receptor agonist with 94% sequence homology to human GLP-1. GLP-1 plays a role in the physiological regulation of appetite and energy intake.

Aim. This review summarizes current evidence on the effects of semaglutide on skin conditions and its cutaneous adverse reactions.

Materials and methods. A literature search was conducted in PubMed and Google Scholar databases using the keywords: "semaglutide", "skin", "glp-1 receptor agonists", "dermatology". Meta-analyses, systematic reviews, original studies, clinical trials, case series, and case reports in English were included without time restrictions. Eligible studies involved human subjects and provided data on semaglutide's effects on skin diseases and/or cutaneous side effects. Reference lists were also screened for additional relevant articles.

Results. Available literature indicates potential therapeutic benefits of semaglutide in several skin disorders, including hidradenitis suppurativa and psoriasis. However, reports of cutaneous adverse reactions and newly emerging skin conditions have also been documented.

Conclusions. Semaglutide may offer therapeutic value in dermatological conditions such as psoriasis and hidradenitis suppurativa, however cutaneous adverse reactions have also been reported. Further research is required to clarify its clinical utility and safety profile in dermatology.

Keywords: semaglutide, GLP-1 receptor agonists, skin

1. Introduction

Semaglutide, as a GLP-1 analogue, exhibits 94% sequence homology with the human GLP-1[1]. GLP-1 is a physiological modulator of appetite and caloric consumption, and its receptor is present in various brain areas associated with the regulation of appetite[1]. It binds with plasma albumin which is connected with decrease in renal clearance and protection from degradation. Moreover, semaglutide is stabilized against degradation by the DPP-4 enzyme[1]. Furthermore, it is involved in stimulating insulin secretion and decreasing glucagon secretion (both dependent from glucose levels). It also delays gastric emptying[1].

According to the FDA information for semaglutide, this GLP-1 receptor agonist is indicated in combination with diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus, to lower the risk of major adverse cardiovascular events in adults with type 2 diabetes mellitus and established cardiovascular disease, to lower the risk of sustained eGFR decline, end-stage kidney disease and cardiovascular death in adults with type 2 diabetes mellitus and chronic kidney disease[2], and alongside a reduced-calorie diet and enhanced physical activity: to lower the risk of major adverse cardiovascular (CV) events (CV death, non-fatal myocardial infarction, or non-fatal stroke) in adults with established CV disease and either obesity or overweight, to reduce excess body weight and maintain weight reduction long term in: adults and pediatric patients aged 12 years and older with obesity, adults with overweight who have at least one weight-related comorbidity[1].

Current literature provides evidence of semaglutide's therapeutic effects in various skin disorders. Conversely, cutaneous adverse reactions and the emergence of new skin conditions have also been reported. This literature review aims to summarize the available data on the impact of semaglutide on skin diseases, including semaglutide - associated cutaneous adverse events.

2. Research materials and methods.

This review was performed in Pubmed and Google Scholar electronic databases. Combinations of the following keywords were used for the search: "semaglutide", "skin", "glp-1 receptor agonists", "dermatology". Meta-analyses, systematic reviews, original studies, clinical trials, case series and case reports in English, with no time limits were included. The inclusion criteria were: 1) human subjects, 2) data on the effects of semaglutide treatment on skin conditions and/or cutaneous side effects. Additionally, the reference lists of the included studies were screened to identify further relevant articles.

3. Research results

A recent study on the effects of semaglutide on cellular regeneration in skin and retinal cells in vitro suggests that this GLP-1 receptor agonist exhibits potent antioxidant effects in human dermal fibroblasts and retinal endothelial cells through the reduction of oxidative stress, enhancement of cell viability, and reduction of apoptosis[3]. The authors concluded that by upregulating endogenous antioxidant genes and downregulating pro-inflammatory mediators, semaglutide further reinforces its protective effect against oxidative damage[3].

3.1 Hidradenitis Suppurativa

Hidradenitis suppurativa (HS) is a chronic, inflammatory, recurrent, debilitating disease of the skin[4,5]. It typically appears after puberty, characterized by painful, deep-seated, inflamed lesions in areas containing apocrine glands, most frequently affecting the axillae, inguinal, and anogenital regions[4,5]. Krajewski et al. suggested that the anti-inflammatory properties and effects on body weight reduction of GLP-1 receptor agonists make them a potential early intervention in HS, addressing central factors to the disease's pathogenesis: metabolic dysfunction and inflammation[6].

Current literature provides data on the effects of semaglutide in patients with HS.

A cross-sectional survey study of 22 participants who were prescribed semaglutide (40.9%), tirzepatide (36.4%), dulaglutide (18.2%), or liraglutide (4.5%) for weight loss or diabetes (not HS), focused on the severity of HS and quality of life based on the patient-reported outcomes. The average duration of the treatment was 17 months (range: 2-108). Changes in HS - specific health were not associated with the duration of treatment. Weight loss was reported in 77.3% of patients. 68.2% of the patients noted improved HS - specific health, 31.8% reported no change. Patients observed a reduction in flares (61.9%), new lesions (66.7%), pain (52.4%), drainage (61.9%), itch (47.6%), and odor (42.9%). 59.1% indicated that HS had less effect on their everyday activities. In general, 59.1% of participants stated that they would recommend GLP-1 receptor agonists to other patients with HS. These findings suggest that GLP-1 receptor agonists may serve as a valuable adjunctive therapy for HS, especially given the high prevalence of obesity and diabetes in this patient population[7].

A retrospective cohort study of semaglutide use for decreasing HS resource utilization analyzed a change in the usage of biologics (infliximab, adalimumab, secukinumab), antibiotics (doxycycline, minocycline, azithromycin, erythromycin, clindamycin, clarithromycin, metronidazole, rifampin), steroids (hydrocortisone, prednisone, betamethasone, budesonide, methylprednisolone, dexamethasone, triamcinolone), and the number of emergency department visits in patients with HS receiving semaglutide. On the basis of the observed decrease in risk ratios (RR), patients receiving semaglutide demonstrated reduced use of antibiotics (RR: 0.758, CI: 0.732 - 0.785), steroids (RR: 0.839, CI: 0.811 - 0.868), and visits to the emergency department (RR: 0.715, CI: 0.681 - 0.751). There was no observed difference in biologic usage (RR: 0.983, CI: 0.862 - 1.122). The authors suggested that biologics are typically used as a long-term therapy, while other interventions are often applied as temporary measures. Additionally, the observed reduction in healthcare resource utilization may reflect decreased treatment needs and could be associated with improvements in HS severity[8].

In a retrospective study, Posada et al. reported an improvement in HS in 27 out of 45 HS patients treated with semaglutide. The initial semaglutide dose was 0.52 ± 0.47 mg/week and was increased to 1.11 ± 0.82 mg/week at 6 months and 1.36 ± 0.86 mg/week at 12 months. The authors reported that higher doses of semaglutide were associated with improvement at 3, 6, and 12 months[9].

A systematic review by Krajewski et al. explored the potential role of GLP-1 receptor agonists, including semaglutide, in the management of HS. The authors highlighted GLP-1 receptor agonists as a potential therapeutic strategy for HS, especially when used early, prior to initiating biologic treatment. The authors suggested that their combined anti-inflammatory and weight-reducing effects may simultaneously address metabolic disturbances and systemic inflammation, which are central to HS pathophysiology, potentially postponing or decreasing reliance on biologics[6]. In combination therapy, GLP-1 receptor agonists could complement biologics by modulating metabolic and systemic inflammatory pathways, while biologics specifically inhibit cytokines such as TNF-α and IL-17[6]. The authors suggested that this approach may be particularly beneficial for patients with severe, treatment-resistant HS or relevant metabolic comorbidities, although additional research is necessary to confirm its safety and effectiveness[6].

Lyons et al. reported the use of semaglutide and its impact on disease control and quality of life in 30 patients with obesity and HS. It was reported that the use of semaglutide, even at low doses (mean 0.8 mg per week), as an addition to conventional HS therapy resulted in enhanced quality of life and fewer disease flares. Furthermore, semaglutide exerted beneficial effects on systemic inflammation, weight reduction, and metabolic markers, including HbA1c[10].

A 2025 real-world analysis evaluating semaglutide's effect on major adverse cardiovascular events (MACE) in patients with HS found that the risk of acute myocardial infarction was 1.2% (HS + GLP) vs 1.6% (HS only) (risk difference (RD) -0.4% [95% CI: - 0.7 to - 0.1], z = - 2.60, p = 0.009, risk ratio (RR) 0.76 [0.62 - 0.94], odds ratio (OR) 0.76 [0.61 - 0.93]). Stroke incidence was 0.8% vs 1.2%, respectively (RD - 0.4% [- 0.6 to - 0.1], z = - 2.96, p = 0.003, RR 0.69 [0.54 - 0.88], OR 0.69 [0.54 - 0.88])[11]. The composite MACE analysis revealed an event rate of 1.7% in semaglutide users compared with 2.4% (RD - 0.6% [- 1.0 to 0.3], p <0.001, RR 0.73 [0.61 - 0.87], OR 0.72 [0.61 - 0.86]). Kaplan-Meier analysis showed higher event-free survival with semaglutide: at the last follow-up, AMI-free survival 84.7% vs 89.0% (log-rank p <0.001, [HR: 0.68] [95% CI: 0.55 - 0.84]), stroke-free survival 96.3% vs 69.3% (p <0.001, HR: 0.63 [95% CI: 0.49 - 0.80]), composite-MACE-free survival 81.9% vs 65.1% (p <0.001, HR: 0.66 [95% CI: 0.55 - 0.78]). The authors suggested that semaglutide may be associated with a significant reduction in MACE[11].

3.2 Psoriasis

Psoriasis is a chronic, immune-mediated disease presenting with cutaneous and systemic manifestations[12,13]. It is characterized by an intricate genetic architecture, and its worldwide prevalence is estimated at 2 - 3%[12,13]. Psoriasis has been connected with multiple comorbidities including: psoriatic arthritis, autoimmune disease, cardiovascular disease, obesity, metabolic syndrome, chronic obstructive pulmonary disease, sleep apnoea, liver disease, psychiatric illness, and addictive behaviour: smoking and alcohol abuse[12].

Several studies have described the impact of semaglutide on psoriasis. A randomized clinical trial evaluated its effects on psoriatic lesions in obese patients with type 2 diabetes mellitus. In the semaglutide arm, the median PASI decreased from 21 (IQR 19.8) at baseline to 10 (IQR 6) after 12 weeks ($p = 0.002$). In the control group, PASI decreased from 20.6 (IQR 8.9) to 15.9 (IQR 8.7; $p = 0.03$). Quality of life scores also improved with semaglutide. Median DLQI decreased from 14 (IQR 5) to 4 (IQR 4; $p = 0.002$) after 12 weeks, whereas the control group showed a reduction from 10.1 (IQR 4.3) to 8.1 (IQR 4.8); $p = 0.007$. A significant improvement in clinical outcomes was reported in patients receiving semaglutide, while the control group showed no meaningful change in psoriasis severity between baseline and week 12. At week 12, 46% of treated patients reached PASI 90 and 8% reached PASI 100 response in the semaglutide group, compared with only 7% reaching PASI 90 and none reaching PASI 100 in controls. Analysis of DLQI categories likewise confirmed a significant improvement in quality of life with semaglutide, an effect not reported in the control group[14].

A prospective cohort study of 43 patients with psoriasis and obesity found that six months of semaglutide treatment led to significant reductions in PASI (- 48%), BMI, and both preperitoneal and superficial fat, together with improvements in DLQI, BDI, and metabolic parameters. Baseline disease severity, depressive symptoms, insulin resistance, and preperitoneal fat showed a negative association with PASI improvement, and these relationships persisted after adjustment. The greatest correlations with ΔPASI were observed for reductions in superficial fat, DLQI, and BDI. After adjustment, changes in BMI and glycemic markers were no longer significantly associated. Overall, the findings indicate that semaglutide benefits both cutaneous disease activity and broader systemic health in patients with psoriasis and obesity[15].

A 2021 report described a case of a 73-year-old male patient with T2DM, class III obesity, and plaque psoriasis, treated with topical therapy and adalimumab, but without therapeutic success. At baseline, his PASI was 33.2, and the DLQI was 26.0. Semaglutide was added to ongoing metformin therapy (initiated at 0.25 mg/week for 4 weeks, then 0.50 mg/week for 12 weeks, and subsequently 1 mg/week). After 4

months, glycemic parameters showed improvement, the patient experienced weight loss, and an improvement in his psoriatic plaques was reported: PASI decreased to 8.0 (- 76%) and DLQI to 3. After 10 months, there was continued improvement in glycemic control, weight-related parameters, and psoriasis severity with reductions of 32% in HbA1c, 16.3% in BMI, and 92% in PASI compared with baseline. DLQI declined to 0[16]. Another case reported in 2023 describes a 50-year-old woman with psoriasis, T2D, and obesity who had not responded to guselkumab. At baseline, her PASI was 12.0 and her DLQI was 20. Semaglutide was initiated (0.25 mg/week for 4 weeks, then 0.50 mg/week for 16 weeks, followed by 1 mg/week). After 4 months, PASI had decreased to 4.0 and DLQI to 5. After 10 months, PASI and DLQI had declined by 98.3% and 95%, respectively, demonstrating marked improvement in psoriatic lesions and a substantial enhancement in quality of life[17].

3.3 Hair Loss

In an analysis of the FDA Adverse Event Reporting System from 2022 to 2023, Godfrey et al. reported increased reporting odds of alopecia for semaglutide (ROR: 2.46, 95% CI: 2.14-2.83) and tirzepatide, whereas no increase was observed for liraglutide, dulaglutide, exenatide, or lixisenatide[18].

A retrospective cohort study of 283 patients analyzed the use of GLP-1 receptor agonists and hair loss. 35 out of 283 patients experienced hair loss. The analysis found no statistically significant association between the use of specific GLP-1 receptor agonist and androgenetic alopecia in either chi-squared tests or logistic regression models. Nonetheless, the odds ratio observed for semaglutide (OR = 6.97) indicated a potential trend toward increased hair loss. For telogen effluvium, most results were nonsignificant, although tirzepatide demonstrated a borderline p value (.0537)[19].

A pharmacovigilance study evaluated hair loss-related adverse events associated with semaglutide based on the FAERS database (Q4/2003 - Q3/2023) using disproportionality analysis. A disproportionality analysis was performed for GLP-1 receptor agonists, SGLT2 inhibitors, dipeptidyl peptidase-4 inhibitors, sulfonylureas, metformin, and insulin. The relative reporting ratio, proportional reporting ratio, and reporting odds ratio for semaglutide were each 1.24 (95% CI, 1.08 - 1.42), with a chi-squared value of 9.45. The authors reported that no positive signal linking semaglutide to hair loss was identified[20].

3.4 Other

A recent study by Levy et al. analyzed a large cohort of patients (after propensity score matching, both the treated and control groups comprised 206,844 patients each) to examine the effects of GLP-1 receptor agonists treatment on cancer risk in individuals with obesity. In propensity-matched cohorts,

the incidence of skin cancer within 5 years was 457 (22.1) in the treated group versus 560 (27.1) in controls (p = 0.001). For melanoma, it was 92 (4.4) versus 112 (5.4), respectively (p = 0.16). The risk of melanoma over follow-up was: HR 0.972 (95% CI 0.621 - 1.521) at 1 year, HR 0.837 (95% CI 0.609 -1.152) at 3 years, and HR 0.631 (95% CI 0.479 - 0.832) at 5 years. For skin cancer, the HRs were 0.631 (95% CI 0.507 - 0.785) at 1 year, 0.677 (95% CI 0.587 - 0.782) at 3 years, and 0.622 (95% CI 0.55 - 0.704) at 5 years. In a subgroup analysis of cancer risk by individual drugs, semaglutide (100,494 matched pairs) showed risk reductions in skin cancers (HR = 0.476, 95% CI = 0.393 - 0.576), melanoma (HR = 0.548, 95% CI = 0.367 - 0.818)[21].

Castellanos et al. described a 76-year-old female patient who developed a three-week petechial rash in her lower extremities and reported recent semaglutide use. Laboratory tests showed elevated transaminases, alkaline phosphatase, total bilirubin, and inflammatory markers. Imaging revealed hepatosplenomegaly. Autoimmune testing showed: positive ANA direct, elevated dsDNA (123 IU/ml), positive anti-histone antibody, low C4, and normal C3 levels. Anti-mitochondrial M2 and anti-smooth antibodies were detected. Anti-SSA, Anti-SSB, P-ANCA, and C-ANCA were negative. The patient met ACR/EULAR criteria for SLE, likely drug-induced. Following administration of high doses of steroids, an improvement in liver function tests was observed[22].

Another case report described a 14-year-old female patient with congenital linear scleroderma since the age of six after various therapies including solumedrol pulse dosing, MTX, low-dose oral steroids (for over a year), and MMF. Tocilizumab 162 mg every other week in combination with MMF 1000 mg BID and MTX 20 mg weekly was initiated as the patient experienced worsening erythema, atrophy of the left shoulder and scapular region, and progressive thinning of the subcutaneous fatty tissue, confirmed on MRI. After four months of this therapy progressive skin hardening and limited mobility of her left arm was observed. Semaglutide therapy was initiated due to weight gain (presumably related to prolonged steroid use). In the following period, mobility of the left arm improved, skin hardness decreased. After 7 months of semaglutide treatment, no worsening or flares of scleroderma were reported, and the patient noted improved arm mobility. On examination, mild atrophy of the left arm with a faint purple-to-pink discoloration of the upper arm and left posterior shoulder was observed. The skin was supple on palpation, no pronounced erythema was observed[23].

3.5 Adverse reactions of semaglutide therapy - cutaneous manifestations

The available literature reports cutaneous adverse reactions associated with semaglutide treatment. In a scoping review, Tran et al. summarized dermatologic adverse events reported in patients treated with

semaglutide[24]. Injection site reactions occurred in 3.5% (151/4308) of semaglutide users (plus one case report) versus 6.7% (201/3010) in the placebo/comparator group ($p < .001$)[24]. Among altered skin sensation events reported in 42/334 patients in the oral semaglutide (50 mg weekly) group, and 4/333 in the placebo group: dysaesthesia was reported in 1.8% versus 0% ($p = .014$), hyperesthesia in 1.2% versus 0% ($p = .046$), neuralgia in 0.9% versus 0.3% ($p = .317$), pain of skin in 2.4% versus 0% ($p = .005$), paresthesia in 2.7% versus 0.3%, sensitive skin in 2.7% versus 0% ($p = .003$), skin discomfort in 0.3% vs 0% ($p = .317$), skin burning sensation in 1.8% versus 0.6% ($p = .156$), respectively[24,25]. Alopecia occurred in 6.9% of semaglutide users versus 2.7% in the placebo group[25]. A different study reported a burning sensation in 0.2% vs 0% ($p = .478$) patients, and alopecia in 0.2% vs 0.5% ($p = .617$), respectively[24,26].

Malignant neoplasms: squamous cell carcinoma of the skin was reported in 0.8% (1/128) semaglutide patients versus 0% (0/129) in the placebo group ($p = .313$), basal cell carcinoma in 0.8% (1/130) vs 0% (0/129) ($p = .317$)[24,27], and unspecified malignant neoplasm in 0.4% (2/494) vs 0.3% (1/360), $p = .757$[24], respectively.

Tran et.al summarized several isolated reactions which were reported only as single case reports, including angioedema[28], bullous pemphigoid[29], dermal hypersensitivity reaction (2 cases)[30], eosinophilic fasciitis[31], and leukocytoclastic vasculitis[24,32]. In the review by Tran et. al dermatologic adverse events not otherwise specified occurred in 4.1% (22/533) of semaglutide users versus 1.5% (4/274) in the placebo/comparator group ($p = .042$)[24]. The authors underlined that differences in dosage and routes of administration may affect the type and severity of cutaneous manifestations, highlighting the need for further research[24]. In 2024 Pinheiro et. al reported a case of skin-limited leukocytoclastic vasculitis induced by once-weekly subcutaneous semaglutide[33]. In 2025, Stark et al. reported four cases of allodynia in patients receiving semaglutide therapy, potentially related to dose escalation[34].

4. Discussion

In this review, we summarized the available evidence on the effects of semaglutide therapy in skin diseases including psoriasis and hidradenitis suppurativa, as well as the range of cutaneous adverse reactions reported during treatment.

A 2024 review suggested that the anti-inflammatory actions of GLP-1 receptor agonists extend beyond their established effects on weight loss and glycemic control[35]. The author concluded that GLP-1 receptor agonists improve disease outcomes and decrease systemic inflammation primarily through modulation of immune-cell signaling, decreasing NF-κB pathway activation and reducing pro-inflammatory cytokine levels[35].

Mintoff et al. reported that individuals with HS tend to exhibit a worse metabolic profile, characterized by increased central adiposity measures, higher systolic blood pressure, greater insulin resistance

markers, and a higher prevalence of metabolic syndrome[36]. The authors highlighted that even when categorized by body composition phenotypes, HS patients - particularly those with normal weight (classified as metabolically healthy) - still display adverse metabolic characteristics compared with controls. Metabolically unhealthy status combined with overweight or obesity further amplifies HS risk[36].

Zouboulis et al. suggested that obesity is a key predisposing factor for HS, likely driven in part by endocrine mechanisms that sustain a chronic pro-inflammatory state[37]. The authors noted that obesity influences disease prognosis, while weight reduction may have a beneficial impact on the prevalence and severity of HS, and in some cases may even lead to spontaneous remission[37,38].

A systematic review and meta-analysis from 2012 found that individuals with psoriasis have over 50% higher odds of obesity compared with those without the disease. The pooled OR for obesity was greater in patients with moderate to severe psoriasis than in those with mild forms. Moreover, patients with existing psoriasis were more likely to develop new-onset obesity than non-psoriatic individuals[39]. The authors concluded that weight-reduction strategies for obese patients with psoriasis may help improve obesity-related comorbidities and potentially reduce psoriasis severity[39].

5. Conclusions

Current literature describes the effects of semaglutide therapy on skin diseases, including psoriasis and hidradenitis suppurativa. Nonetheless, cutaneous adverse reactions associated with this GLP-1 receptor agonist have also been reported and warrant careful consideration. Further research is needed to clarify the drug's potential therapeutic role in dermatological conditions.

Disclosure

Author's Contribution

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