

GLP-1 Receptor Agonists in Overweight and Obese Patients With Hidradenitis Suppurativa

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INTRODUCTION

Hidradenitis suppurativa (HS) is a chronic, multifactorial inflammatory disease that is influenced by obesity and metabolic dysfunction. Overweight and obese individuals may experience more severe HS due to increased skin folds and body surface friction.¹ Furthermore, metabolic dysfunction, which drives pro-inflammatory states, has also been implicated in HS pathogenesis.¹ While weight reduction and improvements in metabolic state have been associated with disease amelioration, current evidence regarding the specific benefits of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in HS is limited to a few studies.¹⁻⁴

This IRB-exempt retrospective study analyzed electronic medical records of adult patients seen at the University of Pennsylvania between 01/01/2019–12/31/2023 with a diagnosis of HS (ICD-10 L73.2) and concomitant body mass index (BMI) $\geq 27\text{kg/m}^2$ with at least one weight-related comorbidity (diabetes, hypertension, and/or hyperlipidemia) or BMI $\geq 30\text{kg/m}^2$.⁵ All patients with ≥ 2 HS dermatology appointments and GLP-1RA use during the study period were included. HS disease activity was defined by treating clinicians' documentation and is reported as improvement, no change, or worsening disease. The chi-square tests evaluated categorical variables. Wilcoxon rank sum test assessed associations between clinical parameters (weight change, age at HS diagnosis, GLP-1RA duration, BMI, and hemoglobin A1c [HbA1c]) and HS disease activity.

Of 425 HS patients identified, 142 (33.4%) met the inclusion criteria. Demographics and HS clinical characteristics are outlined in Tables 1 and 2. 44.4% (n=63) of patients had a documented Hurley stage at the initial visit, of which 57.1% (n=36) had stage II-III disease. Most patients (83.8%, n=119) were not receiving GLP-1RA therapy prior to their initial HS appointment. The average duration of GLP-1RA therapy from its initiation to the most recent HS visit was 564.2 days (range: 14–2905).

Improvement of HS was experienced by 59.9% (n=85) of patients, while 30.3% (n=43) had no change in disease activity, and 9.9% (n=14) experienced worsening. Baseline BMI was $41.1\text{kg} \pm 8.8$, and HbA1c was 6.7 ± 2.2 . Patients who improved vs those with no change or worsening disease were more likely to experience a higher degree of BMI reduction (BMI change: $-6.2\text{kg} \pm 13.5$ vs $-0.4\text{kg} \pm 13.8$, $P=0.01$). No association was found between age of HS diagnosis ($P=0.68$), GLP-1RA duration ($P=0.98$), baseline BMI ($P=0.46$), or baseline HbA1c ($P=0.89$) with improvement of HS. Reduction in HbA1c was not associated with HS improvement ($P=0.23$). There was no difference in improvement rates between never smokers and former or current smokers ($P=0.40$).

Our findings suggest that GLP-1RA use may be associated with improved HS outcomes in overweight and obese patients, potentially through mechanisms related to weight reduction. Furthermore, improving overall metabolic state likely reduces baseline inflammation and increases the likelihood of response to standard HS treatments. While GLP-1RAs may be promising as an adjunct therapy for these HS patients, further investigation is needed to elucidate the degree to which weight reduction and metabolic/inflammatory modulation contribute to their therapeutic potential before their routine use can be recommended. Notably, the retrospective nature of our study and the lack of a comparator group limit our ability to determine causality. Furthermore, given that HS patients often receive multimodal treatment, future prospective studies should control for concomitant therapies to better isolate the effects of GLP-1RAs. Additional studies should also assess whether insurance status or healthcare utilization patterns, potential confounders of our study, contribute to differences in HS outcomes. Despite these limitations, our findings highlight a potential association between GLP-1RA use and HS improvement, warranting further investigation in randomized controlled trials.

TABLE 1.

Baseline Demographic and Clinical Characteristics	
Clinical Parameter	Results n (%)
Sex	
Male	12 (8.5%)
Female	130 (91.5%)
Race/Ethnicity	
Black	82 (57.7%)
White	46 (32.4%)
Asian	6 (4.2%)
Hispanic/Latino	5 (3.5%)
American Indian/Alaskan Native/Native Hawaiian	3 (2.1%)
Smoking status	
Current	21 (14.8%)
Former	28 (19.7%)
Never	93 (65.5%)
Metabolic parameters	
Initial body mass index (kg/m ² , mean ± SD)	41.1 ± 8.8
Initial hemoglobin A1C (mean ± SD)	6.7 ± 2.2
GLP1-RA received	
Semaglutide	125 (88.0%)
Liraglutide	46 (32.4%)
Tirzepatide	44 (31.0%)
Dulaglutide	9 (6.3%)
Exenatide	2 (1.4%)
Switched to a different GLP-1RA	59 (41.5%)
Comorbidities	
Obesity	119 (83.8%)
Pre-diabetes mellitus	23 (16.2%)
Diabetes mellitus	65 (45.8%)
Hypertension	70 (49.3%)
Hyperlipidemia	55 (38.7%)
Polycystic ovarian syndrome	23 (16.2%)
Hyperthyroidism or hypothyroidism	18 (12.7%)
Coronary artery disease	2 (1.4%)

GLP1-RA, glucagon-like peptide-1 receptor agonist

TABLE 2.

Hidradenitis Suppurativa Clinical Activity and Treatments Received	
Clinical Parameter	Results n (%)
Age at HS diagnosis (years, mean ± SD)	38.6 ± 12.0
Duration of HS symptoms prior to initial visit (years, mean ± SD)	11.4 ± 9.1
Hurley stage at initial visit	
I	27 (19.0%)
II	22 (15.5%)
III	14 (9.9%)
Not documented	79 (55.6%)
Hurley stage at most recent visit	
I	26 (18.3%)
II	30 (21.1%)
III	24 (16.9%)
Not documented	62 (43.7%)
HS treatments received at any point in HS course	
Topical antibiotics	124 (87.3%)
Oral antibiotics	93 (65.5%)
Antiseptic washes	94 (66.2%)
Biologics	33 (23.2%)
Spirololactone	51 (35.9%)
Metformin	4 (2.8%)
HS course post GLP1-RA initiation	
Improved	85 (59.9%)
No change	43 (30.3%)
Worsening	14 (9.9%)
Duration of HS follow-up (days)	
Mean	1804
Median	1219
Range	41-5825

HS, hidradenitis suppurativa
GLP1-RA, glucagon-like peptide-1 receptor agonist

DISCLOSURES

Dr Taylor has served as a consultant, advisory board member, and/or speaker for AbbVie, Arcutis, Armis Scientific, Avita, Beiersdorf, Biorez, Bristol-Myers Squibb, Cara Therapeutics, Dior, Eli Lilly, EPI Health, Evolus, Galderma, GloGetter, Hugel America, Incyte, Johnson & Johnson, L'Oreal USA, Medscape, MJH LifeSciences, Pfizer, Piction Health, Sanofi, Scientis US, UCB and Vichy Laboratories. She has received royalties from McGraw-Hill. She has served as an investigator for Allergan, Concert Pharmaceuticals/Sun Pharma, Croma-Pharma GmbH, Eli Lilly, and Pfizer. Dr. Ogunleye has served as a consultant, advisory board member, and/or speaker for Beiersdorf, MJH LifeSciences, Dermatology Times, Veradermics, and Health Central. Author Encarnacion, Author Desir, Author Anusionwu, Author Hislop, and Author Feldman have no conflicts of interest to declare.

Ethical Approval: Reviewed and exempted by the University of Pennsylvania; IRB # 855643.

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