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Original Research Article

A retrospective study of GLP-1 receptor agonists uses in the management of hidradenitis suppurativa

Fatima Khan[1]*, Nneka Ede[2], Emily Clark[3], Venessa Pena-Robichaux[3]

[1]Dell Medical School, Austin, Texas
[2]Department of Internal Medicine, Dell Medical School, Austin, Texas
[3]Department of Internal Medicine, Division of Dermatology, Dell Medical School, Austin, Texas

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***Correspondence:**
Dr. Fatima Khan,
E-mail: fatima.sufia12@gmail.com

ABSTRACT

Background: Hidradenitis suppurativa (HS) is a chronic inflammatory disorder frequently associated with metabolic comorbidities. Given the anti-inflammatory and metabolic effects of GLP-receptor agonists (GLP-1Ras), these agents have been proposed as a potential adjunctive therapy for HS.
Methods: A retrospective chart review identified 151 HS patients prescribed a GLP-1RA between January 2020 and January 2025. After excluding individuals younger than 18 years and those initiating biologics, long-term immunomodulators, or anti-hormonal therapies after GLP-1RA start, 22 patients met inclusion criteria. Clinical improvement was assessed qualitatively through provider documentation of symptom or disease-status improvement.
Results: Among 22 patients, 63.6% demonstrated clinical improvement following GLP-1RA therapy. The cohort included 10 Black, 6 Hispanic, 5 White, and 1 Asian patient. Semaglutide was the most common agent (n=14), followed by tirzepatide (n=7), and exenatide (n=1). Race was significantly associated with improvement (p= 0.025). 92% of non-Black patients improved compared with 30% of Black patients (p= 0.011). This disparity persisted after adjusting for HS severity, treatment duration, comorbidities, insurance type, and income. No significant associations were observed for gender, age, ethnicity, comorbidities, or insurance type.
Conclusions: GLP-1RAs may offer symptomatic benefit for HS, with nearly two-thirds of patients showing improvement. However, marked racial differences in response highlight the need for equitable HS research and investigation into biological, structural, and socioeconomic contributors to treatment variability. Larger prospective studies are warranted to clarify therapeutic efficacy and drivers of demographic disparities.

Keywords: Hidradenitis suppurativa, GLP-1RA, Glucagon-like peptide receptor agonist, Metabolic disorders

INTRODUCTION

Hidradenitis suppurativa (HS) is a chronic, relapsing inflammatory skin condition that is characterized by painful, recurrent nodules, abscesses, tunnels, and scarring in intertriginous areas. Beyond its cutaneous manifestations, HS in increasingly recognized as a systemic condition associated with a broad range of metabolic and inflammatory comorbidities, including obesity, dyslipidemia, insulin resistance, and type 2 diabetes. These metabolic abnormalities not only contribute to disease severity but also reflect the complex interplay between cutaneous inflammation, systemic immune activation, and metabolic dysfunction.

HS imposes a significant burden on patients, often leading to significant physical pain, impaired mobility, and psychological morbidity, including depression and reduced quality of life.

Given the strong metabolic and inflammatory mechanisms of HS, emerging therapies target both pathways simultaneously. GLP-1 receptor agonists (GLP-1RAs), such as liraglutide and semaglutide, are synthetic peptides widely used for the treatment of type 2 diabetes and obesity. These agents exert pleiotropic effects that extend beyond glycemic control, including improving in insulin sensitivity, reductions in body weight, and modulation of systemic inflammation. Emerging evidence suggests that GLP-1RAs may attenuate key inflammatory pathways implicated in HS pathogenesis, such TNF-a, IL-17, and NF-kB signaling, while also reducing mechanical stress and friction in intertriginous areas through weight loss. Together, these metabolic and immunologic effects provide a compelling rationale for exploring GLP-1RAs as a potential adjunctive therapy for HS.[1-4]

Presently, most existing studies do not account for confounding from concurrent HS therapies, making it difficult to isolate the contributions of GLP-1RA therapy. This retrospective study evaluated the clinical outcomes of patients with HS who were treated with GLP-1RAs at a single academic center, applying strict inclusion criteria and excluding patients initiating new systemic HS therapies.

METHODS

A retrospective cohort study was conducted at Ascension/Dell Seton Medical Center Dermatology clinic to identify all patients with a documented diagnosis of HS who were prescribed a GLP-1RA between 01 January 2020 and 01 January 2025 after the University of Texas at Austin Institutional Review Board exemption determination. An initial cohort of 151 patients was identified through electronic health record query using ICD-10 codes for HS and pharmacologic records for GLP-1RA prescriptions. Extracted variables included demographic characteristics (age, sex, race, ethnicity), socioeconomic indicators (insurance type, estimated household income based on ZIP-code-linked census data), and clinical comorbidities relevant to HS and metabolic disease. HS severity was assessed using Hurley stage and/or HS-physician global assessment (HS-PGA) score prior to GLP-1RA initiation.

Detailed treatment characteristics were collected, including GLP-1RA agent, dose, duration of therapy, and concurrent HS-directed treatment. To minimize confounding from escalation of HS-specific therapy, patients were excluded if they initiated a new biologic agent, long-term immunomodulator, or anti-hormonal therapy after starting a GLP-1RA. Additional exclusion criteria included age younger than 18 years and use of GLP-1RA for fewer than 60 consecutive days. After applying these criteria, 22 patients remained in the final cohort.

Clinical improvement was evaluated qualitatively through narrative review of treating providers' documentation. Improvement was defined as explicit provider-reported reduction in HS symptoms (e.g. pain, drainage, flare frequency), improvement in physical examination findings, or overall assessment of improvement disease status as determined by HS-PGA score. Statistical analyses were conducted in Python (v3.11.5). Descriptive statistics were used to summarize patient demographics, treatment characteristics, and clinical improvement. To assess the association between patient characteristics and improvement status, Chi-square, T-test, Mann-Whitney, and Fisher's exact tests were used as appropriate.

RESULTS

The study cohort included 22 patients with a mean age of 39.6 years. Among patients, 81.8% (n=18) were female, and 21.8% (n=4) were male. Racial distribution included 45.50% (n=10) Black patients, 27.30% (n=6) Hispanic patients, 22.70% (n=5) White patients, and 4.50% (=1) Asian patients. Disease severity by Hurley stage was as follows: 36.36% (n=8) of patients with Hurley stage 1; 31.81% (n=7) of patients were Hurley stage II; 27.27% (n=6) patients were Hurley stage III. Overall, 63.60% (n=14) patients demonstrated clinical improvement. Regarding treatment, 63.63% (n=14) of patients were receiving semaglutide, 31.82% (n=7) of patients using tirzepatide, and 4.55% (n=1) of patients was using exenatide. Complete patient demographics can be seen in Table 1.

Inferential statistics assessing the association between race and improvement demonstrated race was significantly associated with improvement (χ^2=9.324, p=0.025), with higher rates among White, Asian, and Hispanic patients compared to Black patients. No significant associations were observed for gender, age, ethnicity, comorbidities, or insurance type (Table 1).

Further analysis of race demonstrated no significant differences in HS severity by race. Mean Hurley stage was 1.80 among Black patients and 2.00 among non-Black patients (t=-0.541, p=0.595). Similarly, mean HS-PGA scores did not differ significantly between Black (1.75) and non-Black (2.14) participants (t=-0.990, p=0.348). Income was lower among Black patients (mean=$86,252) compared to non-Black patients (mean=$105,273), but this difference did not reach statistical significance (t-test p=0.093; Mann-Whitney p=0.156). When restricted to Black vs. White participants, the difference became significant (Mann-Whitney p=0.049). Treatment length did not significantly differ by race (Black mean=246.0 days; non-Black mean=183.17 days; p=0.554).

However, improvement rates differed significantly by race. Only 30% of Black patients showed improvement compared to 92% of non-Black patients (χ^2=6.497, p=0.011). When restricted to Black versus White participants, this relationship remained significant (Fisher's exact p=0.026).

Table 1: Summary of cohort characteristics, GLP-1 use analysis, and racial sub-analysis.

Variables		N	%
(A) Descriptive statistics of final cohort			
Age in years	39.63 (11.24) (mean (SD))		
Sex	Female	18	81.80
	Male	4	18.20
Ethnicity	Not Hispanic or Latino	12	54.50
	Hispanic or Latino	6	27.30
	Patient Declined	4	18.20
Race	Black or African American	10	45.50
	Hispanic or Latino	6	27.30
	White	5	22.70
	Asian	1	4.50
Language	English	20	90.90
	Spanish	1	4.50
	NaN	1	4.50
Insurance	Private	15	68.20
	Medicaid/self-pay	7	31.80
Biologics/anti-hormonal	Yes	12	54.50
	No	10	45.50
GLP-1	Semaglutide	14	63.63
	Tirzepatide	7	31.82
	Exenatide	1	4.55
Hurley stage	1	8	36.36
	2	7	31.81
	3	6	27.27
	Missing data	1	4.55
Clinical improvement	Yes	14	63.60
	No	8	36.40
Variable versus improvement		**P value**	
(B) Statistical analysis of patients with hidradenitis suppurativa and GLP-1 use			
Gender		1.000	
Age		0.118	
Race		0.025	
Ethnicity		0.163	
Insurance		1.000	
Income		0.151	
Comorbidities		0.903	
Diabetes mellitus		1.000	
(C) Subanalysis of Black patients versus non-Black patients			
Hurley stage		0.595	
HS-PGA score		0.348	
Income		0.093	
Treatment length		0.554	
Clinical improvement (versus White patients)		0.011 (0.026)	

Among Black patients only, improvement was not significantly associated with insurance type (Fisher's exact OR=1.5, p=1.000), use of biologics/antihormonals (OR=inf, p=0.200), income (t=-0.915, p=0.387), HS severity as measured by Hurley stage (t=0.503, p=0.629), or treatment length (t=-0.034, p=0.974).

DISCUSSION

This retrospective study suggests that GLP-1 inhibitors may provide symptomatic benefit for HS. Approximately two-thirds of the cohort demonstrated clinical improvement following GLP-RA initiation, supporting the hypothesis that these agents may have a therapeutic role in HS management.[5] These findings are consistent with prior

retrospective studies that have shown that GLP-1RAs demonstrate quantitative improvement in HS-related scores over time.[6-8]

Notably, racial disparities in improvement were observed. While the majority of White, Asian, and Hispanic patients experienced clinical benefit, only 30% of Black patients reported improvement. Further analyses revealed that this disparity could not be fully explained by differences in HS Hurley stage, HS-PGA score, treatment duration, or other socioeconomic factors. Although income differences between racial groups approached significance, they did not entirely account for the lower response rate observed among Black patients.

The mechanism underlying this discrepancy remains unclear. Possible explanations include genetic variants which may contribute to inter-individual response differences, increased disease severity at baseline that is not fully captured by Hurley stage or HS-PGA scores, structural barriers to care, and disparities in access to dermatologic management. These findings underscore the need for equitable inclusion of diverse populations in HS clinical research and for further exploration of how socioeconomic and systemic factors may influence treatment outcomes.

Expanding on this, it is critical to recognize that GLP-1 receptor agonists may exert their therapeutic effects through multiple pathways beyond glycemic control and weight reduction. Emerging evidence suggests that GLP-1RAs have anti-inflammatory properties, modulating key immune pathways implicating in HS pathogenesis such as the NLRP3 inflammasome and cytokine networks involving IL-1b and TNF-a.[9,10] These immunomodulatory effects may contribute to the reduction in lesion formation and disease activity observed in some patients. Moreover, genetic polymorphisms affecting GLP-1 receptor expression or downstream signalling could underlie inter-individual variability in treatment response, particularly across different ancestral backgrounds.[11]

Psychosocial factors, including chronic stress and systemic inflammation, also play a significant role in HS severity and treatment outcomes. GLP-1RAs may indirectly improve HS by mitigating metabolic syndrome components that exacerbate systemic inflammation. However, social determinants such as healthcare access, provider bias, and socioeconomic status remain critical barriers that disproportionality affect Black patients, potentially limiting their benefit from these therapies.

These findings must be interpreted within the broader context of well-documented racial inequities in HS epidemiology, disease severity, and treatment access. Black patients experience more than double the incidence of HS compared to White individuals and have significantly higher odds of presenting with Hurley stage III disease, emergency department utilization, and hospitalization, even after adjusting for demographic and clinical factors.[12] Hispanic patients similarly present with increased severity and greater healthcare utilization.[13] Importantly, Black patients are less likely to receive advanced therapies such as biologics or small-molecule inhibitors and face markedly lower odds of GLP-1RA prescribing across multiple healthcare systems, despite bearing the highest disease burden.[14] These intersecting inequities raise concern that emerging HS treatments, including GLP-1RAs, may reproduce or widen existing gaps in care unless access barriers and structural determinants are directly addressed.

CONCLUSION

Overall, the observed improvement in many patients supports the potential utility of GLP-1RAs as an adjunctive therapy in HS, particularly in individuals with comorbid obesity, diabetes, or metabolic syndrome. Yet, the heterogeneity in treatment response, both within this cohort and across published studies, highlights the need to better define which biological, metabolic, and social determinants shape GLP-1RA effectiveness in HS. Future investigation should incorporate mechanistic endpoints to clarify the relative contributions of weight-dependent and weight-independent pathways. Equally important is the need for adequately powered studies that recruit diverse populations, given the substantial racial disparities in HS burden, disease severity, and access to advanced therapies. Prospective trials that integrate social determinants of health, neighborhood-level factors, and structural barriers to care will be essential to understanding whether access or differential biological response contributes more strongly to the racial gap observed in this cohort. Ultimately, inclusive research is necessary to determine the true therapeutic potential of GLP-1RAs in HS and to ensure that emerging treatments benefit patients equitably.

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