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Review Article

Hidradenitis Suppurativa Subtypes: Exploring Proposed Phenotypes and Their Clinical Relevance

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Hidradenitis suppurativa (HS) is a multi-factorial disease with a wide range of presentations. Several classification systems have attempted to define unique phenotypes, aiming to guide clinical management and elucidate underlying mechanisms. However, overlapping features limit their practical utility. We reviewed existing literature and consolidated key features into four clinical domains: weight, smoking, atypical site involvement, and inflammatory load. By considering these factors, clinicians can adopt a more individualized, integrative approach to HS management. We recommend clinicians discuss weight management and smoking cessation with all HS patients. For pharmacological management, atypical site involvement, a moderate to severe assessment with HS-IGA (Hidradenitis Suppurativa Investigator Global Assessment), and a high HiSQoL (Hidradenitis Suppurativa Quality of Life) score may warrant early systemic therapy to control disease progression and reduce long-term complications.

1. INTRODUCTION

Hidradenitis suppurativa (HS) is a chronic inflammatory skin condition characterized by painful inflammatory nodules, abscesses, and tunnels.[1] The intertriginous areas, including the axillae, inguinal folds, inframammary areas, and buttocks, are the most commonly affected sites.[1] HS is diagnosed clinically, yet the average delay from symptom onset to diagnosis is approximately seven years, resulting in substantial disease burden and impaired quality of life.[2]

Prompt treatment of HS is essential due to its systemic inflammatory nature and associated comorbidities. The pathogenesis of HS involves complex immune dysregulation, with elevated levels of cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin(IL)-17, and IL-23.[3] Additionally, HS has been associated with comorbidities such as metabolic syndrome, dyslipidemia, adverse cardiovascular outcomes, polycystic ovarian syndrome, hyperthyroidism, inflammatory bowel disease, spondylarthritis, lymphoma, suicidality, sexual dysfunction, and significant psychosocial distress.[4] The physical manifestation of HS, including scarring, further contribute to long-term morbidity and highlight the importance of early intervention.[5]

In recent years, various classification systems have been proposed to subtype HS based on clinical presentation and disease characteristics.[6-12] However, the clinical relevance of these subtypes and their potential role in guiding management remain unclear. This review summarizes current research on HS subtypes and discusses practical considerations for integrating these classifications into clinical practice.

2. MATERIALS AND METHODS

We performed a literature review using PubMed and Google Scholar for studies published up to June 19th, 2025. Articles that proposed or described classification systems for HS subtypes, whether based on clinical observations, statistical analyses, or immunological data, were included. Search terms were “hidradenitis suppurativa,” “phenotypes,” “subtypes,” and “endotypes.” Only English-language articles were considered.

Included publications were assessed of bias using the Oxford Levels of Evidence scoring system.[13] A score of five represents a publication with robust methods whereas a lower score can suggest more bias.

3. RESULTS

Out of a total of 1079 screened articles, seven publications satisfied our inclusion criteria. Five studies had an Oxford Levels of Evidence score of 2 and 3, which consisted of prospective trials and cross-sectional studies.[6-8,10,12] A case series (n = 4) received a score of four and a publication based on clinical experience was allocated a score of five.[9, 11]

The complex pathophysiology of HS has prompted research exploring potential subtypes that may improve understanding of disease heterogeneity and inform future approaches to management (Table 1). These proposed classifications can be broadly grouped based on anatomical distribution, clinical patterns, and immunological or phenotypic clustering.

Table 1. Summary of published HS subtypes.

Author	Study Design	Oxford Levels of Evidence	Subtypes	Features
Martorell et al. (2019)[6]	Prospective Study (n = 197)	2	Follicular	Early onset (17.6 vs 26.7 years); more nodules; multiple comedomes (5.7 vs 0.9)
			Inflammatory	More abscesses and fistulas (4 and 3 vs 0.6 and 0.3); higher IgA (497.7 vs 262.3); higher IHS4 (21.0 vs 7.5); more aggressive disease
Canoui-Poitrine et al. (2013)[7]	Cross-sectional Study (n = 618)	2	Axillary-mammary (48%)	Breast and armpit lesions (0.74); hypertrophic scars (0.41)
			Follicular	Breast and armpit lesions (0.96) ears, chest, back, or legs (0.55); follicular lesions: pilonidal sinus (0.48), comedomes (0.74), severe acne (0.47); family history of HS (0.44); more often male (OR = 4.6, p < 0.001); smokers (OR = 2.2, p = 0.005), and more severe disease (OR = 1.6, p < 0.001)
			Gluteal	Gluteal lesions (0.54); papules and folliculitis (0.71); less obesity (OR = 0.6, p – 0.03); less severe (OR = 0.9, p < 0.001)
Riera-Marti et al.(2024)[8]	Cross-sectional Study (n = 83)	3	Gluteal: latent cluster 1	Elderly females; later diagnosis; more sinus tracts
			Gluteal: latent cluster 2	Males; early onset; more nodules and folliculitis
van der Zee and Jemec (2015)[9]	Clinical Experience	5	Regular	Lacks distinguishing features of other subtypes
			Frictional furuncle	Overweight; deep nodules and abscesses in friction areas (abdomen, thighs, buttocks); few tunnels or fistulas
			Scarring folliculitis	Pustules, cysts, superficial nodules, cribriform scarring, double-ended comedomes; buttocks, inguinal, and public regions; small Hurley stage 1lesions prone to scarring; overweight and smokers
			Conglobata	Cyst; acne conglobate on face/back; family history; moderate-severe (Hurley II-III); usually non-overweight men
			Syndromic	Associated with yoderma gangrenosum; arthritis; acne
			Ectopic	Primarily facial lesions
Cazzaniga et al. (2021)[10]	Cross-sectional Study (n = 965)	2	Axillary-mammary	Most severe; obese females; axillary-groin (0.85) and mammary lesions (0.59)
			Axillary-gluteal	Non-obese males; moderate severity; gluteal (0.50) and genital (0.17) lesions; acne and pilonidal cysts
			Axillary-groin	Non-obese females; milder disease; axillary (0.52) and groin (0.66)
Naasan and Affleck(2015)[11]	Case Series (n = 4)	4	Typical	Axillae, groin, buttocks, and inframammaryregions
			Atypical	Face, neck, distal limbs, retroauricular sites
Gonzalez-Manso et al.(2021)[12]	Cross-sectional Study (n = 103)	2	Cluster 1 (64.9%)	Non-obese males; posterior nodules; early onset; higher IL-10; gamma-secretase mutations; pilonidal sinus history
			Cluster 2 (35.1%)	Obese males/females; anterior sites; more sinus tracts; later-onset; higher IL-1, CRP, IL-17; IL-6

SUBTYPES BASED ON ANATOMICAL DISTRIBUTION

HS subtypes have been categorized based on anatomical distribution, with one study distinguishing typical versus atypical site involvement. It proposed a simple anatomical classification consisting of a typical subgroup, with lesions confined to intertriginous areas such as the axillae, groin, buttocks, and inframammary regions, and an atypical subgroup involving nonflexural areas such as the face, neck, distal limbs, and retroauricular regions.[10] Atypical site involvement may represent a distinct clinical phenotype with potential implications for disease severity and manage-

ment.[10] As an example, facial HS can be more visible and can impact quality of life to a greater extent. However, treating this can be more difficult with the skin sensitivity limiting potential topical treatments and the complex anatomy preventing the usage of surgical techniques. Similarly, a cross-sectional study of 618 patients identified three anatomical subtypes: axillary-mammary, follicular, and gluteal.[7] The axillary-mammary group predominantly exhibited lesions in the armpit and breast with hypertrophic scarring. This group was also more likely to consist of men, non-smokers, and individuals without a family history of HS. The follicular subtype, in addition to armpit and breast involvement, frequently affected the ears and chest and was associated with comedones, epidermal cysts, papules, folliculitis, pilonidal sinuses, severe acne, and a history of smoking. The gluteal subtype involved papules and folliculitis of the buttocks, a milder disease course, and a higher prevalence of smoking.[7]

Latent class analysis of 83 patients confirmed that the gluteal subtype was more frequently observed among non-obese males, smokers, individuals with arthropathy, and those with a history of pilonidal sinuses.[8] Two distinct clusters were identified within this group. Cluster one predominantly included female patients with later disease onset and more tunnels, while cluster two consisted mainly of males with earlier onset, nodular lesions, and folliculitis. These findings suggest heterogeneity even within anatomically defined subtypes.[8]

A cross-sectional study of 965 patients from the Italian National HS Registry further supported anatomical subtyping.[14] The axillary-mammary subtype was identified as the most severe and more prevalent among obese female patients with axillary, groin, and mammary involvement. The axillary-gluteal subtype affected non-obese males with moderate disease and involvement of gluteal and genital areas, often accompanied by acne and pilonidal cysts. The axillary-groin subtype, consisting of non-obese females with milder disease, was primarily characterized by axillary and groin lesions.[14]

SUBTYPES BASED ON CLINICAL PATTERNS AND DISEASE SEVERITY

Other proposed classifications focus on clinical characteristics beyond anatomical location. A prospective study of 197 patients identified two subtypes based on disease presentation.[6] The follicular subtype was associated with an earlier age of onset and a higher prevalence of nodules with comedones. The inflammatory subtype exhibited more abscesses, tunnels, elevated IgA levels, and a more aggressive disease course.

Another study proposed six subtypes based on clinical experience, including a regular subtype, considered the most common; a frictional furuncle type seen in overweight patients with deep nodules and abscesses in areas of friction; a scarring folliculitis type characterized by pustules, cysts, superficial nodules, cribriform scarring, and double-ended comedones, often seen in overweight smokers; a conglobata type with cyst formation and acne conglobata lesions of the face and back, primarily affecting men; a syndromic type associated with pyoderma gangrenosum, arthritis, and acne; and an ectopic type with lesions predominantly affecting the face.[9]

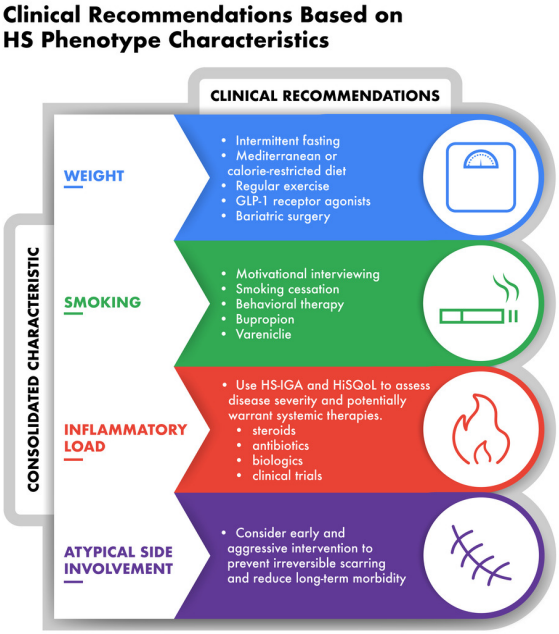

Figure 1. Clinical recommendations based on HS phenotype characteristics

PHENOTYPIC AND IMMUNOLOGICAL CLUSTERING OF HS

A cross-sectional study of 103 patients applied clustering analysis incorporating clinical and immunological parameters to explore potential HS subtypes and identified two clusters.[11] Cluster one, comprising non-obese males with posterior lesions, earlier disease onset, and a history of pilonidal sinus, demonstrated elevated IL-10 levels. Cluster two involved obese males and females with anterior site involvement, later onset, more tunnels, and higher concentrations of IL-1, CRP, IL-17, and IL-6. These findings suggest a potential link between inflammatory profiles, disease distribution, and clinical phenotype.[11]

4. DISCUSSION

Although several classification systems have been proposed, there is limited clinical relevance due to the complex clustering and failure to correlate the subtypes with treatment outcomes. To simplify phenotyping and guide pharmacological and integrative management, we consolidated recurring characteristics from the literature into four domains: weight, smoking, anatomical site involvement, and inflammatory load (Figure 1).

4.1. WEIGHT

Obesity has emerged as a relevant factor in certain HS phenotypes. Overall, individuals with HS demonstrate a higher prevalence of obesity compared to the general population,

and weight reduction has been associated with decreased disease severity and symptom burden.[15] Some proposed subtypes (axillary-mammary, frictional furuncle, scarring folliculitis, and cluster 2) appear more strongly linked to obesity, suggesting a potential role in disease pathogenesis for specific patient groups.[9,11,14] Consequently, addressing obesity is recommended as part of integrative HS management.

Patients with excess weight should receive counseling on sustainable dietary changes and physical activity. Low-friction forms of exercise, such as walking and swimming, are generally preferred. Dietary modifications may target a 500-750 Kcal/day caloric deficit, tailored to the individual's lifestyle and preferences.[16] In cases of severe obesity, pharmacological options such as a GLP-1 receptor agonist and bariatric procedures may be considered, particularly when intertriginous areas are affected.[17]

Intermittent fasting, typically involving 16 hours of fasting followed by an eight-hour eating window, improves insulin sensitivity, which may contribute to the pathogenesis of HS through androgen receptor modulation and insulin-like growth factor-1 (IGF-1) pathways.[18] Given the association between insulin resistance and HS, we recommend assessing insulin sensitivity using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) index, even in patients with normal body mass index (BMI).[19]

The gut microbiome also plays a role in metabolic regulation, primarily through short-chain fatty acids (SCFAs) production.[20] Although the microbiome remains a complex and evolving field with limited direct clinical application, several microbial markers have been identified that may assist in managing metabolic status. For example, supplementation with *Akkermansia muciniphila,* a probiotic associated with improved insulin sensitivity and reduced body weight, has shown promise, likely mediated by P9 protein, which exerts GLP-1-like effects.[21,22] Furthermore, the *Prevotella/ Bacteroides* (P/B) ratio may provide insights into dietary responsiveness. Individuals with a high P/B ratio (> 0.01) appear to benefit more from fiber-rich diets, such as New Nordic Diet (whole grain, fruits, and vegetables), whereas those with a low P/B ratio may respond better to higher protein and animal fat intake typical of a Western diet.[23]

4.2. SMOKING

Smoking is another factor in HS subtyping, with certain phenotypes (gluteal, scarring folliculitis, and follicular) showing a higher prevalence of tobacco use.[7,9] Smoking cessation is an essential component of HS management, and all patients with a history of smoking should be counseled on its potential impact on disease severity. Clinicians should offer resources for behavioral counseling, and consider pharmacological interventions such as nicotine replacement therapy, varenicline, or bupropion, based on individual risk factors and preferences.[24]

4.3. TYPICAL VS ATYPICAL SITES

Anatomical distribution is central to several proposed HS classifications,[7,8,14] with a distinction made between typical and atypical sites.[10] Typical HS lesions most frequently involve intertriginous areas, such as the groin, buttocks, and axillae.[25] For these patients, strategies to minimize friction, including loose-fitting clothing or specialized garments such as HidraWear are recommended.[26] Similarly, low-friction exercise like walking and swimming may help reduce symptom exacerbation.

Involvement of atypical sites, such as the face, neck, or distal extremities, can warrant earlier, more aggressive treatment to prevent irreversible scarring. Scarring can result in permanent disfigurement, and frequently necessitates surgical intervention.

4.4. INFLAMMATORY LOAD

The extent of inflammatory load is a key factor in determining the appropriate treatment intensity. To objectively quantify disease severity, we recommend the use of Hidradenitis Suppurativa Investigator Global Assessment (HS-IGA).[14] This system utilizes a maximum of the lesion count from the two regions: upper and lower body. This lesion count is then grouped into a six point scoring system. We suggest that a HS-IGA of 2 is a good time to start discussing and considering systemic therapies.

The usage of self-reported outcomes is also helpful to assess quality of life impact. The Hidradenitis Suppurativa Quality of Life (HiSQoL) is a scale which assesses associated symptoms, psychosocial implications, and adaptations of activity.[27] A higher score suggests a greater impact on quality of life. An international group of HS experts determined that both the HS-IGA and HiSQoL are two of the most useful assessments to implement in clinical practice.[28]

Early introduction of biologics is recommended to mitigate long-term complications, including scarring and development of comorbidities. Currently approved biologics for HS include adalimumab, secukinumab, and bimekizumab.[29] In cases where disease remains refractory to these options, enrollment in clinical trials may be appropriate, particularly as novel biologics and Janus kinase (JAK) inhibitors are under investigation.

5. CONCLUSION

Multiple classification systems have been proposed to characterize HS subtypes, drawing on clinical experience, anatomical distribution, and statistical clustering. However, significant overlap between phenotypes limits their clinical applicability. By synthesizing findings across the literature, we identified recurring domains, obesity, smoking history, atypical site involvement, and inflammatory load, that may prove actionable for individualized management (Table 2). We recommend clinicians incorporate these domains into an integrative care approach. Addressing obesity and smoking should be prioritized for all applicable patients. The presence of atypical site involvement may

Table 2. Clinical Recommendations Based on HS Phenotype Characteristics

Consolidated Characteristic	Clinical Recommendations
Weight	Intermittent fasting, Mediterranean or calorie-restricted diet, regular exercise, GLP-1 receptor agonists, bariatric surgery
Smoking	Motivational interviewing, smoking cessation, behavioral therapy, bupropion, varenicline
Inflammatory Load	Use IHS4 to assess disease severity; a score> 4 may warrant systemic therapies including steroids, antibiotics, or biologics
Atypical Side Involvement	Consider early and aggressive intervention to prevent irreversible scarring and reduce long-term morbidity

warrant earlier, more aggressive treatment. Finally, disease severity should be assessed using validated outcome measure such as the HS-IGA to guide systemic therapy decisions.

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CONFLICTS OF INTEREST

RKS serves as a scientific advisor for LearnHealth, Codex Labs, and Arbonne and as a consultant to Burt's Bees, Novozymes, Nutrafol, Novartis, Bristol Myers Squibb, Abbvie, Leo, Almirall, Galderma, UCB, Incyte, Pfizer, Sanofi, Sun, and Regeneron Pharmaceuticals.

DATA AVAILABILITY

The data underlying this article will be shared on reasonable request to the corresponding author.

ETHICS

No human or animal subjects were used in this review.

PATIENT CONSENT

Not Applicable

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REFERENCES

1. Sabat R, Alavi A, Wolk K, et al. Hidradenitis suppurativa. *Lancet*. 2025;405(10476):420-438. doi:10.1016/S0140-6736(24)02475-9

2. Saunte DM, Boer J, Stratigos A, et al. Diagnostic delay in hidradenitis suppurativa is a global problem. *Br J Dermatol*. 2015;173(6):1546-1549. doi:10.1111/bjd.14038

3. Vossen A, van der Zee HH, Prens EP. Hidradenitis Suppurativa: A Systematic Review Integrating Inflammatory Pathways Into a Cohesive Pathogenic Model. *Front Immunol*. 2018;9:2965. doi:10.3389/fimmu.2018.02965

4. Cartron A, Driscoll MS. Comorbidities of hidradenitis suppurativa: A review of the literature. *Int J Womens Dermatol*. 2019;5(5):330-334. doi:10.1016/j.ijwd.2019.06.026

5. Krakowski AC, Admani S, Uebelhoer NS, Eichenfield LF, Shumaker PR. Residual scarring from hidradenitis suppurativa: fractionated CO2 laser as a novel and noninvasive approach. *Pediatrics*. 2014;133(1):e248-51. doi:10.1542/peds.2012-3356

6. Martorell A, Jfri A, Koster SBL, et al. Defining hidradenitis suppurativa phenotypes based on the elementary lesion pattern: results of a prospective study. *J Eur Acad Dermatol Venereol*. 2020;34(6):1309-1318. doi:10.1111/jdv.16183

7. Canoui-Poitrine F, Le Thuaut A, Revuz JE, et al. Identification of three hidradenitis suppurativa phenotypes: latent class analysis of a cross-sectional study. *J Invest Dermatol*. 2013;133(6):1506-1511. doi:10.1038/jid.2012.472

8. Riera-Marti N, Vilarrasa E, Lopez-Llunell C, Gamissans M, Sin M, Romani J. Gluteal Hidradenitis Suppurativa: Analysis of 83 Patients. *Actas Dermosifiliogr*. 2024;115(2):137-142. doi:10.1016/j.ad.2023.09.021

9. van der Zee HH, Jemec GB. New insights into the diagnosis of hidradenitis suppurativa: Clinical presentations and phenotypes. *J Am Acad Dermatol*. 2015;73(5 Suppl 1):S23-6. doi:10.1016/j.jaad.2015.07.047

10. Cazzaniga S, Pezzolo E, Bettoli V, et al. Characterization of Hidradenitis Suppurativa Phenotypes: A Multidimensional Latent Class Analysis of the National Italian Registry IRHIS. *J Invest Dermatol*. 2021;141(5):1236-1242e1. doi:10.1016/j.jid.2020.08.032

11. Naasan H, Affleck A. Atypical hidradenitis suppurativa. *Clin Exp Dermatol*. 2015;40(8):891-893. doi:10.1111/ced.12655

12. Gonzalez-Manso A, Agut-Busquet E, Romani J, et al. Hidradenitis Suppurativa: Proposal of Classification in Two Endotypes with Two-Step Cluster Analysis. *Dermatology*. 2021;237(3):365-371. doi:10.1159/000511045

13. Howick J, Glasziou P, Greenhalgh T, et al. *The Oxford 2011 Levels of Evidence*. ICJLL; 2011.

14. Garg A, Zema C, Kim K, et al. Development and initial validation of the HS-IGA: a novel hidradenitis suppurativa-specific investigator global assessment for use in interventional trials. *Br J Dermatol*. 2022;187(2):203-210. doi:10.1111/bjd.21236

15. Kromann CB, Ibler KS, Kristiansen VB, Jemec GB. The influence of body weight on the prevalence and severity of hidradenitis suppurativa. *Acta Derm Venereol*. 2014;94(5):553-557. doi:10.2340/00015555-1800

16. Kim JY. Optimal Diet Strategies for Weight Loss and Weight Loss Maintenance. *J Obes Metab Syndr*. 2021;30(1):20-31. doi:10.7570/jomes20065

17. Shah VN, Akturk HK, Kruger D, et al. Semaglutide in Adults with Type 1 Diabetes and Obesity. *NEJM Evid*. 2025;4(8):EVIDoa2500173. doi:10.1056/EVIDoa2500173

18. Yuan X, Wang J, Yang S, et al. Effect of Intermittent Fasting Diet on Glucose and Lipid Metabolism and Insulin Resistance in Patients with Impaired Glucose and Lipid Metabolism: A Systematic Review and Meta-Analysis. *Int J Endocrinol*. 2022;2022:6999907. doi:10.1155/2022/6999907

19. Madeira FB, Silva AA, Veloso HF, et al. Normal weight obesity is associated with metabolic syndrome and insulin resistance in young adults from a middle-income country. *PLoS One*. 2013;8(3):e60673. doi:10.1371/journal.pone.0060673

20. Mann ER, Lam YK, Uhlig HH. Short-chain fatty acids: linking diet, the microbiome and immunity. *Nat Rev Immunol*. 2024;24(8):577-595. doi:10.1038/s41577-024-01014-8

21. Depommier C, Everard A, Druart C, et al. Supplementation with Akkermansia muciniphila in overweight and obese human volunteers: a proof-of-concept exploratory study. *Nat Med*. 2019;25(7):1096-1103. doi:10.1038/s41591-019-0495-2

22. Yoon HS, Cho CH, Yun MS, et al. *Akkermansia muciniphila* secretes a glucagon-like peptide-1-inducing protein that improves glucose homeostasis and ameliorates metabolic disease in mice. *Nat Microbiol*. 2021;6(5):563-573. doi:10.1038/s41564-021-00880-5

23. Hjorth MF, Roager HM, Larsen TM, et al. Pre-treatment microbial Prevotella-to-Bacteroides ratio, determines body fat loss success during a 6-month randomized controlled diet intervention. *Int J Obes (Lond)*. 2018;42(3):580-583. doi:10.1038/ijo.2017.220

24. Corelli RL, Hudmon KS. Medications for smoking cessation. *West J Med*. 2002;176(2):131-135.

25. Ballard K, Shuman VL. Hidradenitis Suppurativa. In: *StatPearls*. ; 2025.

26. HidraWear. https://hidrawear.com/

27. Kirby JS, Thorlacius L, Villumsen B, et al. The Hidradenitis Suppurativa Quality of Life (HiSQOL) score: development and validation of a measure for clinical trials. *Br J Dermatol*. 2020;183(2):340-348. doi:10.1111/bjd.18692

28. Mastacouris N, Tannenbaum R, Strunk A, et al. Outcome Measures for the Evaluation of Treatment Response in Hidradenitis Suppurativa for Clinical Practice: A HiSTORIC Consensus Statement. *JAMA Dermatol*. 2023;159(11):1258-1266. doi:10.1001/jamadermatol.2023.3282

29. Charrow A, Santiago-Soltero K, Porter M. Biologics in hidradenitis suppurativa: Progress and new directions. *J Am Acad Dermatol*. 2024;91(6S):S27-S30. doi:10.1016/j.jaad.2024.09.027