Quantitative Sensory Testing Offers a Novel Approach to Determining Pain Mechanisms in Hidradenitis Suppurativa

Patrick J. Speck, BS, Ali Alsouhibani, PhD, Danielle E. Mustin, MEng, Helen Li, MS, Jason Barron, MPH, Emily F. Cole, MD MPH, Daniel E. Harper, PhD, Lauren A.V. Orenstein, MD MSc

**Background:** Pain is rated by patients with hidradenitis suppurativa (HS) as the most important symptom, but little is known about the mechanisms driving HS pain. Quantitative Sensory Testing (QST) is a standardized suite of sensory examinations that could provide novel insights into the mechanisms of HS pain.

**Objective:** Characterize pain profiles in HS using QST.

**Methods:** 10 HS patients underwent QST of the dorsal hand (control) and inflamed HS lesion (test) sites per German Research Network on Neuropathic Pain protocol. Results were compared to age and sex-matched reference values for the hand and trunk. Patients also completed clinical skin exam and patient reported outcome surveys including itch, pain, quality of life, anxiety, and depression (Table 1).

**Results:** QST data are presented in Figure 1. Inflamed HS lesions demonstrated substantial desensitization to innocuous thermal stimuli (cold and warm detection thresholds, thermal sensory limen). Conversely, inflamed HS lesions demonstrated hypersensitivity to noxious mechanical stimuli (pain pressure threshold). Central sensitization markers (control wind-up ratio and noxious mechanical hypersensitivity) were elevated but inconclusive.

**Discussion:** QST is a novel yet feasible method for objectively measuring sensation and pain in HS. This study demonstrated patterns of thermal desensitization with increased mechanical sensitivity in inflamed HS lesions.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, yrs – median (IQR)</strong></td>
<td>37.5 (33.5-47)</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td>Female – 8 (80%)</td>
</tr>
<tr>
<td></td>
<td>Male – 2 (20%)</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td>White – 5 (50%)</td>
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<tr>
<td></td>
<td>Black – 4 (40%)</td>
</tr>
<tr>
<td></td>
<td>Asian – 1 (10%)</td>
</tr>
<tr>
<td><strong>BMI – median (IQR)</strong></td>
<td>35.6 (30.0-45.5)</td>
</tr>
<tr>
<td><strong>Hurley Stage</strong></td>
<td>I – 1 (10%)</td>
</tr>
<tr>
<td></td>
<td>II – 6 (60%)</td>
</tr>
<tr>
<td></td>
<td>III – 3 (30%)</td>
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<tr>
<td><strong>IHS4 – median (IQR)</strong></td>
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</tr>
<tr>
<td></td>
<td>Mild – 1 (10%)</td>
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<td></td>
<td>Moderate – 4 (40%)</td>
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<tr>
<td></td>
<td>Severe – 5 (50%)</td>
</tr>
<tr>
<td><strong>Itchy Quant – median (IQR)</strong></td>
<td>2.5 (0-3)</td>
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<tr>
<td><strong>Brief Pain Inventory – median</strong></td>
<td>24hr pain max – 2 (1-7)</td>
</tr>
<tr>
<td>(IQR)</td>
<td>Pain on average – 4 (1-6)</td>
</tr>
<tr>
<td></td>
<td>Pain interference – 0.5 (0-0.7)</td>
</tr>
<tr>
<td><strong>McGill Pain Questionnaire</strong></td>
<td># Descriptors selected – 7 (4-13.5)</td>
</tr>
<tr>
<td>(n=7)</td>
<td>Tender – 7 (100%)</td>
</tr>
<tr>
<td></td>
<td>Throbbing – 6 (86%)</td>
</tr>
<tr>
<td></td>
<td>Pain to light touch – 5 (71%)</td>
</tr>
<tr>
<td><strong>PainDETECT</strong></td>
<td>Unlikely neuropathy – 4 (40%)</td>
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<tr>
<td></td>
<td>Unclear – 2 (20%)</td>
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<tr>
<td></td>
<td>Likely neuropathy – 4 (40%)</td>
</tr>
<tr>
<td><strong>HiSQoL – median (IQR)</strong></td>
<td>Total – 10 (8.5-22)</td>
</tr>
<tr>
<td>(n=7)</td>
<td>Symptoms – 6 (3-8.5)</td>
</tr>
<tr>
<td></td>
<td>Psychosocial – 2 (1-7)</td>
</tr>
<tr>
<td></td>
<td>Activities – 4 (2.5-9.5)</td>
</tr>
<tr>
<td><strong>Skindex-16 – median (IQR)</strong></td>
<td>Symptoms – 46 (22-75)</td>
</tr>
<tr>
<td></td>
<td>Emotional – 51 (44-76)</td>
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<tr>
<td></td>
<td>Functional – 28 (8-72)</td>
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<tr>
<td><strong>GAD-7</strong></td>
<td>No anxiety – 5 (50%)</td>
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<td></td>
<td>Mild – 1 (10%)</td>
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<tr>
<td></td>
<td>Moderate – 3 (30%)</td>
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<tr>
<td></td>
<td>Severe – 1 (10%)</td>
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<tr>
<td><strong>PHQ-9</strong></td>
<td>No depression – 4 (40%)</td>
</tr>
<tr>
<td></td>
<td>Mild – 5 (50%)</td>
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<tr>
<td></td>
<td>Moderate – 0</td>
</tr>
<tr>
<td></td>
<td>Moderate-severe – 1 (10%)</td>
</tr>
<tr>
<td></td>
<td>Severe – 0</td>
</tr>
</tbody>
</table>
Fig. 1 Median (IQR) subject z-scores of control and inflamed test sites for each QST domain with shading of normal ranges. CDT- Cool Detection Threshold, WDT- Warm Detection Threshold, TSL- Thermal Sensory Limen, CPT- Cold Pain Threshold, HPT- Hot Pain Threshold, MDT- Mechanical Detection, MPT- Mechanical Pain Threshold, PPT- Pressure Pain Threshold, WUR- Wind-Up Ratio
Quantitative sensory testing in Hidradenitis suppurativa could provide insight into pain mechanisms


Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease that is characterized by recurrent nodules, abscesses, and scarring tunnels. Most people suffering from HS report moderate or worse pain, contributing to reduced quality of life.

The aim of our study was to determine the primary mechanism underlying the pain in individuals with HS using quantitative sensory testing (QST).

QST was performed in individuals with HS using the standardized protocol by the German Research Network on Neuropathic pain. The skin locations tested by QST included: (1) the dorsal hand (control site), (2) an actively inflamed HS lesion, and, if present, (3) a non-inflamed HS lesion. The tests included were cold, warm, and mechanical detection thresholds (CDT, WDT and MDT); cold, heat, and mechanical pain thresholds (CPT, HPT and MPT); thermal sensory limen (TSL); paradoxical heat sensations (PHS); pressure pain thresholds (PPTs); dynamic mechanical allodynia (DMA); mechanical pain sensitivity (MPS); and windup ratio (WUR).

Seven individuals with HS (age=39.7±12.5 years; 5 women) diagnosed by a dermatologist participated in the study. A total of 10 skin locations were tested by QST – 5 draining tunnels (inflamed), 2 scarred nodules (non-inflamed), 1 inflammatory nodule, 1 non-inflamed tunnel, and 1 perilesional site(s). The frequency of participants who had abnormal QST values (defined as a value greater than 95% confidence interval of the reference data) at the control site are as follows: Hyperalgesia (gain of function): WUR = 2/7 (29%), MPS = 4/7 (57%), DMA = 3/7 (43%), PPTs = 3/7 (43%); Hypoesthesia (loss of function): WDT = 1/7 (14%), CDT = 1/7 (14%). All other measures (i.e., MPT, TSL, CPT, HPT, PHS) were within normal range. One participant (14%) reported paradoxical sensations at an inflamed location (draining tunnel). Also, a high frequency of abnormalities in the thermal sensory limen (TSL), indicative of small fiber loss, were found: 5/6 (83%) at the inflamed location and 3/4 (75%) at the non-inflamed location.

Preliminary results suggest that peripherally, at the affected skin, a neuropathic component could play a major role in the perception of pain. However, the pain experienced by a subset of individuals may involve more than the pathology at the skin lesion and could be characterized as nociplastic. Other aspects such as descending inhibitory pathways should be investigated in future studies.
**Title:** Sociodemographic features associated with care by a Dermatologist for Hidradenitis Suppurativa

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**Background:** Care by a dermatologist is associated with treatment initiation, treatment escalation, and prescription of biologics to manage Hidradenitis Suppurativa (HS).\(^1,2\) Understanding whether access to care by a dermatologist differs among sociodemographic groups of HS patients may help identify disparate outcomes.

**Objectives:** Determine how care by a dermatologist is associated with race, sex, and insurance type among a cohort of HS patients.

**Methods:** Participants were identified from two healthcare systems in metropolitan Atlanta; one privately and one publicly owned. Patients seen \(\geq2\) times between 9/10/2014 and 10/7/2020 within the same healthcare system were included. Participants with dermatology as the first visit were excluded. Differences between healthcare systems were determined through \(\chi^2\) tests. Odds of receiving care by a dermatologist based on race, sex, and insurance type were calculated through mixed logistic regression models adjusted for fixed effects of race, sex, insurance type, age, and random effect of healthcare system.

**Results:** The study included 3309 participants: 2556 (77.2%) African American, 2430 (73.4%) female, and 1348 (40.7%) privately insured. 854 (25.8%) received care by a dermatologist. Public system participants were more likely to be African American (odds ratio, 95% CI; 4.44, 3.6-5.5) and receive care by a dermatologist (1.91, 1.6-2.2) and less likely to have private insurance (Cramer’s \(V = 0.66, p <0.0001\)). African Americans were more likely to receive care by a dermatologist compared to all other races (adjusted odds ratio, 95% CI; 1.91, 1.6-2.2) and less likely to have private insurance (Cramer’s \(V = 0.66, p <0.0001\)). African Americans were more likely to receive care by a dermatologist compared to all other races (adjusted odds ratio, 95% CI; 1.27, 1.0-1.6) and those privately insured were more likely to receive care by dermatologist than those publicly insured (1.35, 1.1-1.7).

**Discussion:** Private insurance was most strongly associated with receipt of care by a dermatologist. Contrary to initial hypotheses, African Americans and public health system participants were more likely to receive care by a dermatologist. Interventions targeting low socioeconomic status individuals with HS may improve overall access to care by dermatologists.

**References**


Background: Hidradenitis suppurativa (HS) is a debilitating chronic inflammatory condition that disproportionately affects African Americans and young females.\textsuperscript{1-3} Investigation of sociodemographic disparities in HS disease severity may improve surveillance and treatment among groups with high disease burden.

Objective: Characterize severity of HS using clinician documented Hurley Stage among a cohort of patients with HS.

Methods: Participants were recruited from two hospital systems in the metropolitan Atlanta area; one privately and one publicly owned system. Participants with ≥2 visits at the same healthcare institution and at least one dermatologist visit with a documented Hurley Stage between 9/10/2015 and 10/7/2019 were included. Differences in outcomes and covariates between healthcare systems were assessed using Chi-squared tests. Odds of having moderate to severe HS (Hurley stage 2 or 3) by race and sex were calculated through logistic regression models adjusted for the fixed effects of race, sex, age insurance status, and the random effect of healthcare system.

Results: The study included 449 participants (226 private, 223 public): 351 (78.2%) were African American, 327 (72.8%) were female, 227 (53.0%) had private insurance, and 328 (73.1%) were classified as moderate to severe HS. Participants in the private healthcare system were less likely to be African American and more likely to carry private insurance (p<0.001). Proportion of participants with moderate to severe HS were not different between healthcare systems. Men were more likely than females to have moderate to severe HS (adjusted odds ratio, 95% CI; 2.1, 1.2-3.6) than women but there were no associations between HS severity and race.

Discussion: Males may present to dermatologists later and with more severe disease. Further research may elucidate mechanisms for disparities in HS severity by sociodemographic characteristics, and public health interventions may enable earlier identification and treatment among populations with high disease burden.

References
Background: Pain is rated by patients with hidradenitis suppurativa (HS) as their most important symptom. Yet, little is known about the direct impact of pain on the overall HS experience or pain management strategies commonly employed by HS patients.

Objective: This qualitative study sought to characterize pain experiences and personal pain management strategies in patients with HS.

Methods: Semi-structured interviews were conducted among English-speaking patients ≥18 years of age with a confirmed HS diagnosis and an average Numeric Rating Scale pain score of ≥1 over the preceding week. Interviews were audio-recorded, transcribed verbatim, and data were analyzed using thematic analysis. Interviews continued until thematic saturation was reached, reaching 21 total participants.

Results: Most participants self-managed their pain with minimal input from medical providers. Factors contributing to self-management of pain included ineffective patient-provider communication, poor access to specialty care during disease flares, and patients’ beliefs that there were no safe, effective prescription treatment options. Participants frequently developed a pain management routine with therapeutic escalation determined by pain severity and accessible medications. Several participants who lacked access to effective pain therapies used over the counter medications at dangerously high doses or took analgesics that were not prescribed to them. Additionally, study participants discussed non-pharmacological coping strategies as essential to self-management, such as physical positioning of their body, distraction, or mental willpower.

Discussion: Understanding the pain experiences of patients with HS may elucidate current gaps in patient care, strengthen patient-provider communication, and inform future strategies for improving pain control in this population. Importantly, it is critical that providers recognize and address unsafe pain management practices among HS patients.
Background: Pain is a leading cause of impaired quality of life (QoL) among individuals with hidradenitis suppurativa (HS). The pain experience in HS is not well characterized, and little is known about the ways in which HS pain reduces QoL.

Objective: To characterize pain experiences among individuals with HS.

Method: 21 HS patients > age 18 who had average pain severity of ≥ 1 on the Numeric Rating Scale during the past week were recruited. Semi-structured interviews were conducted, and patient-reported surveys including DLQI, Itchy Quant, Pain Quality Assessment Scale-Revised, GAD-7, and PHQ-9 were administered. Recruitment continued until thematic saturation was reached, and interviews were transcribed and analyzed using thematic analysis.

Results: Participants were predominantly female (16/21), African American (15/21), and had moderate-to-severe HS. (Table 1) Most described pain character as complex, using terms corresponding with both nociceptive and neuropathic pain types. Pruritis was also common. The physical impacts of HS pain included decreased mobility, poor sleep quality, difficulty concentrating, and clothing restrictions. Psychosocial impacts included depression, feelings of a loss of control, social isolation, impaired intimate relationships, and decreased ability to fulfill social responsibilities. Although chronic pain contributed to reduced quality of life, acute pain associated with HS flares appeared to result in the most psychological distress and impact on activities.

Discussion: HS pain has complex symptom characteristics and results in physical and psychosocial impact. Establishing effective therapies for symptomatic management of HS pain, particularly pain associated with HS flares, has the potential to greatly improve patients’ QoL.
<table>
<thead>
<tr>
<th>Table 1: Participant characteristics and patient reported outcomes</th>
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<tbody>
<tr>
<td><strong>Age, median (Q1-Q3) years</strong></td>
</tr>
<tr>
<td><strong>Sex, female, n (%)</strong></td>
</tr>
<tr>
<td><strong>Ethnicity, non-Hispanic/Latino, n (%)</strong></td>
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<tr>
<td><strong>Race, n (%)</strong></td>
</tr>
<tr>
<td>Black/African American</td>
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<tr>
<td>White/Caucasian</td>
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<tr>
<td>Native Hawaiian or Pacific Islander</td>
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<tr>
<td>Asian</td>
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<tr>
<td>American Indian or Alaskan Native</td>
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<tr>
<td><strong>Hurley Stage</strong></td>
</tr>
<tr>
<td>Stage I, n (%)</td>
</tr>
<tr>
<td>Stage II, n (%)</td>
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<tr>
<td>Stage III, n (%)</td>
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<tr>
<td><strong>DLQI, median (Q1-Q3)</strong></td>
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<tr>
<td>Small effect, n (%)</td>
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<tr>
<td>Moderate effect, n (%)</td>
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<tr>
<td>Very large effect, n (%)</td>
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<tr>
<td>Extremely large effect, n (%)</td>
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<tr>
<td><strong>PHQ-9, median (Q1-Q3)</strong></td>
</tr>
<tr>
<td>No depression, n (%)</td>
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<tr>
<td>Mild depression, n (%)</td>
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<tr>
<td>Moderate depression, n (%)</td>
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<tr>
<td>Severe depression, n (%)</td>
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<tr>
<td><strong>GAD-7, median (Q1-Q3)</strong></td>
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<tr>
<td>No anxiety, n (%)</td>
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<tr>
<td>Mild anxiety, n (%)</td>
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<tr>
<td>Moderate anxiety, n (%)</td>
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<tr>
<td>Severe anxiety, n (%)</td>
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Characterization of B cell Dynamics in Hidradenitis Suppurativa: Evidence for antigen-driven pathophysiology

Gordon A. Dale, Jason Barron, Patrick Speck, Joshy Jacob & Lauren A. V. Orenstein

Hidradenitis suppurativa (HS) is a high-morbidity, chronic inflammatory skin disorder that affects an estimated 1% of adults in developed countries and is disproportionately prevalent among African Americans and women. Lesions in HS primarily affect intertriginous sites and present as painful inflammatory nodules, subcutaneous abscesses, and scarring subcutaneous tunnels. Histologically, these lesions are rich in inflammatory infiltrate including T cells, neutrophils, macrophages, dendritic cells, B cells and plasma cells. While the role of B cells and their late differentiation partner, plasma cells, are unclear in HS, emerging evidence has suggested a link between these cells, disease progression, and treatment response. Here, we further characterize B cells in HS using single cell RNA-seq (scRNA-seq) and bacteriophage immunoprecipitation sequencing (PhIP-seq) to identify local and global alterations to B cell and plasma cell populations in HS. Using published bulk RNA-seq data from HS lesions and control skin, we extracted CDR3 sequences using MiXCR software. Diversity analysis demonstrated an increase in clonotypes in HS skin compared to control skin (p<0.05). Further analysis of clonal populations in HS skin using D50 diversity index revealed selective clonal expansions (p<0.01). To understand whether B cell axis alterations in HS were limited to the lesion, we assayed serum from 10 HS patients and 14 controls using PhIP-seq. HS patients possessed serum autoreactivity to a set of four autoantigens compared to controls, including type I collagen (p<0.01 for each). Together, these studies provide evidence for local and global alteration to B cell populations in an antigen-specific manner in HS.

References:

Hidradenitis suppurativa diagnosis and management: A Guide for the Internist

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Lauren.Orenstein@emory.edu
October 15, 2021
Capital Health Internal Medicine Grand Rounds
Disclosures

• ChemoCentryx- Investigator and Consultant
• Novartis- Consultant
• Pfizer- Grant recipient

This presentation will discuss off label uses of medications for HS.
Learning objectives

1. Discuss how HS disrupts patients’ Quality of Life
2. Recognize the clinical features of hidradenitis suppurativa, even early in the disease course
3. Screen for the medical comorbidities associated with HS
4. Understand the role of medical and surgical therapies in HS
5. Initiate first line medical therapies for HS patients presenting in the outpatient and inpatient settings
What is hidradenitis suppurativa (“HS”)

- HS a **chronic and recurrent** inflammatory skin disease
- Locations: Axillae, groin, buttocks, and chest
- *Not* an infection
- *Not* hygiene problem
- *Not* patient’s fault
Epidemiology of HS

- Prevalence ~0.4%\(^1\)
- Average time to diagnosis ~ 10 years\(^2\)
- Disproportionately African Americans and women\(^3\)
  - African American > caucasians; 3.1:1
  - F > M; 2.4:1

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Quality of Life in HS comparable to stroke and end-stage heart failure

• Pain, itch, odor
• Physical limitations
• Wound care
• Depression, anxiety, substance use and 2.4x ↑ suicide risk
• Employment challenges
• Impaired sexual health and relationships

What is the role of internal medicine?

• Early detection and diagnosis
• Trust in health system
• Identification and management of comorbidities
• Inpatient management
• Initial outpatient management
• Recognizing when to refer to specialist
Making the Diagnosis

1) Is there more than one inflamed lesion?
2) Is the course chronic and recurrent?
3) Are lesions bilateral?
4) Are lesion locations classic for HS (axillae, groin, buttocks)?

If “yes” to all 4, then diagnose HS; if “no,” think of alternative diagnoses

Lesion morphologies in HS

Figure 2. Schematic Representation of the Hidradenitis Suppurativa Lesion Types Described in This Study

A Papule
B Pustule
C Nodule
D Plaque
E Ulcer
F Abscess
G Comedo
H Tunnel

Differential Diagnosis

• Bacterial infection
• HSV
• Cutaneous Crohn’s Disease
<table>
<thead>
<tr>
<th>Comorbidity</th>
<th>Evidence Level</th>
<th>Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco smoking</td>
<td>II</td>
<td>B</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
<td>II</td>
<td>B</td>
</tr>
<tr>
<td>Spondyloarthritis</td>
<td>II</td>
<td>B</td>
</tr>
<tr>
<td>Sexual dysfunction</td>
<td>II</td>
<td>B</td>
</tr>
<tr>
<td>Obesity</td>
<td>II</td>
<td>B</td>
</tr>
<tr>
<td>Depression</td>
<td>II</td>
<td>B</td>
</tr>
<tr>
<td>Generalized anxiety disorder</td>
<td>II</td>
<td>B</td>
</tr>
<tr>
<td>Suicidality</td>
<td>II</td>
<td>B</td>
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<tr>
<td>Substance use disorder</td>
<td>II</td>
<td>B</td>
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<tr>
<td>Polycystic ovarian syndrome</td>
<td>II</td>
<td>B</td>
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<td>Dyslipidemia</td>
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<td>Diabetes mellitus</td>
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<td>B</td>
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<tr>
<td>Metabolic syndrome</td>
<td>II</td>
<td>B</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>II</td>
<td>B</td>
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</table>
1. Avoid blaming or shaming the patient.
2. HS medications work best at preventing flares.
3. Tailor initial treatment to impact that disease has on QoL and patient preferences. Ladder therapy can waste precious time!
4. Adequate therapeutic trial requires 3-4 months of consistent use.
5. Surgery may debulk inflammatory disease but should not be used as monotherapy.
Primary care toolkit for HS Management

- **Mild, widespread**
  - Doxycycline 100 mg BID
    - Daily topical antiseptic
  - Spironolactone 75-100 mg daily
    - Daily topical antiseptic
    - Topical clindamycin 1% BID prn

- **Mild, localized**
  - Topical clindamycin 1% BID
    - Daily topical antiseptic: benzoyl peroxide, chlorhexidine or zinc pyrithione

- **Spironolactone 75-100 mg daily**
  - Doxycycline 100 mg BID
    - Daily topical antiseptic
    - Topical clindamycin 1% BID prn
Topical clindamycin 1%

*For papules and pustules, apply twice daily as needed. For prevention, apply once daily to HS affected skin.*

- 12 week placebo-controlled RCT\(^1\) showed:
  - ↓ pustule count
  - Improved patient self-assessment
  - No change in nodule/abscess count

- Increases rates of *S. aureus* resistance in individuals with HS\(^2\) – combine with antimicrobial.

Antimicrobial washes

Lather onto HS affected skin, allow soap to sit on skin for 5 minutes, then rinse.

• Primary for antimicrobial stewardship

Oral doxycycline

Take 100 mg po BID with food and a full glass of water. Do not lie flat until at least 1 hour after taking medication.

- Adverse reactions
  - GI upset
  - Sun sensitivity
  - Teratogenic
  - ↓ effectiveness of OCPs

Spironolactone

*Take 100 mg po daily with food.*

- Two case series, n=66 \(^1,2\)
- Dose: 50-200 mg daily
  - In case series no difference between doses <75 mg vs >100 mg\(^1\)
  - Avg dose 112 mg/day\(^1\)
- Routine potassium monitoring *not* needed for young, health women\(^3\)
- Counseling:
  - Common side effects: breast tenderness, menstrual irregularities, dizziness
  - Not safe in pregnancy

3. JAMA Dermatol. 2015;151(9):941-44. PMID: 25796182
### Primary care toolkit for HS Management

<table>
<thead>
<tr>
<th>Mild, widespread</th>
<th>Mild, localized</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Doxycycline 100 mg BID AND • Daily topical antiseptic</td>
<td>• Spironolactone 75-100 mg daily AND • Daily topical antiseptic • Topical clindamycin 1% BID prn</td>
</tr>
<tr>
<td>• Spironolactone 75-100 mg daily AND • Daily topical antiseptic • Topical clindamycin 1% BID prn</td>
<td>• Topical clindamycin 1% BID AND • Daily topical antiseptic: benzoyl peroxide, chlorhexidine or zinc pyrithione</td>
</tr>
</tbody>
</table>
What does treatment success look like?

• Clinically effective
  • Fewer flares
  • Flares less often
  • Less pain, drainage, and odor
  • Improvement in anemia

• Prevent disease progression

• Ability to return to work/normal activities

• Medication tolerable and acceptable to patient
When to refer HS patients to dermatology

- Any signs of scars/tunnels (moderate or worse disease)
- Mild skin disease refractory to ≥3 months doxycycline or spironolactone
- Significant QoL impact
Clindamycin + Rifampin for HS

- **Case series:** \( n = 178 \)

- **Adverse reactions**
  - ~15\% of patients stop treatment early d/t adverse effects
  - Common AEs: Diarrhea, nausea, vaginitis
  - Serious AEs: Hepatitis, *C. difficile* associated diarrhea, interstitial nephritis, liver injury

- **Medicine-Derm collaboration**
  - Rifampin = CyP450 inducer \( \rightarrow \) Many drug interactions
    - ↓OCP effectiveness
    - Statins: pravastatin and rosuvastatin fewer interactions
  - TB screening in high risk patients
Adalimumab & Infliximab for HS

• **Adverse effects:**
  - **Serious infections** (~2/100 pt-years in patients with psoriasis and PsA)
  - **Lymphoma risk** is exceedingly rare
  - **Skin cancer** risk ↑2x

• **Contraindications:** Heart failure, recent malignancy, untreated latent TB, demyelinating disease, active untreated infection

• **Role of internist:**
  - Routine vaccines; no live vaccines
  - COVID-19 vaccination
  - Pneumonia vaccination: PCV13(Prevnar) on initiation, PPSV23(Pneumovax) 1 year later, booster PPSV23 5 years later, PPSV23 at age 65
  - Age appropriate cancer screening

1. NEJM. 2016;375(5):422-34. PMID: 27518661
Promising HS therapies in the pipeline

• Phase 3 Trials
  • **IL-17 monoclonal antibodies:** Bimekizumab, Secukinumab

• Phase 2 Trials
  • **C5a:** Avacopan, IFX-1
  • **CD40:** Iscalimab
  • **IL-1A:** Bermekimab
  • **IL-23:** Risankizumab-rzaa
  • **IL-36:** Imsidolimab
  • **JAK-1:** INCB054707
  • **Leukotriene A4 hydrolase:** LYS006

www.clinicaltrials.gov
Office-based procedures for HS

Deroofing:

Excision:
Local excision candidate

- 40 yo F with history of HS in bilateral axillae x 10 years
- Disease stable for past 5 years
- Failed: Doxycycline, Clindamycin/Rifampin, and adalimumab. Does not want to start infliximab.
Excision L axilla
Post-op week 6: Scar contraction and limited range of motion

Post-op month 4
Inpatient Management of HS

- Limited evidence
- Acute Medical Management
  - IV antibiotics – clindamycin, ertapenem\(^1,2\) others
  - Oral or IV steroids
- Pain management
- Surgical management: Incision and drainage
- Discharge with long-term management plan
  - ≥ 1 month of oral antibiotics + antimicrobial wash
    - Doxycycline 100 mg BID
    - Clindamycin 300 mg BID + Rifampin 600 mg daily
  - Dermatologist/PCP follow up

1. Int J Dermatol. 2018;57(9):1088-93. PMID:29774531