



# Hidradenitis Suppurativa Flare Management: a Review of Medical and Procedural Strategies

Chelsea Moon<sup>1</sup> · Raveena Ghanshani<sup>1</sup> · Ashley B. Crew<sup>2</sup> · Meagan Hughes<sup>2</sup> · Jennifer L. Hsiao<sup>2</sup> · Katrina H. Lee<sup>2</sup>

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## Abstract

**Purpose of Review** While flares are a well-recognized feature of hidradenitis suppurativa (HS), defining flares and selecting an appropriate management strategy can be challenging. This narrative review discusses approaches to HS flare identification and management, highlighting both preventive and therapeutic strategies.

**Recent Findings** For decades, topical and systemic antibiotics have been a mainstay of HS flare treatment due to their anti-inflammatory properties and quick onset. According to a 2024 survey of HS experts, trimethoprim-sulfamethoxazole and amoxicillin-clavulanate were the second and third most prescribed antibiotic monotherapy following tetracyclines, respectively. Preliminary evidence suggests that pairing amoxicillin-clavulanate with a prednisone taper may have potential for gaining rapid control of HS flares. Among topical treatments, clascoterone and ruxolitinib emerge as promising non-antimicrobial options for mild to moderate HS. Procedural strategies for flare management include intralesional steroids for acutely inflamed nodules or tunnels and punch incision and drainage for relief of painful abscesses. A survey study of 900 individuals with self-reported HS found that complementary and alternative medicine (CAM) interventions are perceived to be one of the most helpful tools for flare management.

**Summary** Flares have significant implications for the quality of life in patients with HS. In order to synergize anchor therapy of baseline disease with prompt control of flare activity, a multimodal approach of medical, procedural, and CAM interventions combined with trigger avoidance and patient education is critical.

**Keywords** Hidradenitis suppurativa · Flare · Antibiotics · HS trigger · Monotherapy · Combination therapy

## Introduction

Hidradenitis suppurativa (HS) is a chronic, inflammatory skin condition that is characterized by painful inflammatory nodules, abscesses, and scarring or tunnel formation. Due to their unpredictable nature and limited guidelines on treatment, HS flare management remains a significant challenge for both patients and clinicians.

Flares are a hallmark feature of HS. In a global survey of HS patients, more than 80% of 1299 patients reported experiencing at least one episode of worsening symptoms every month [1]. Flares pose detrimental repercussions on patients' quality of life and can cause debilitating pain, psychosocial challenges, disruptions to work, and missed clinic visits [2–9]. It is imperative that dermatologists are well-equipped to manage flares in-office and provide patients with tools that can be implemented at home. These management strategies can include lifestyle modifications, complementary and alternative medicine (CAM), pharmacologic interventions, and procedures individually or in a multimodal fashion to help gain rapid control of inflammation and alleviate flare-associated pain. It is important to note that adjustments to baseline treatment regimens should be made for patients with frequent flares. In this review we aim to summarize the current evidence on medical and procedural flare interventions and provide clinicians with a structured approach

✉ Katrina H. Lee  
leekatri@usc.edu

<sup>1</sup> Keck School of Medicine, University of Southern California, California, Los Angeles, USA

<sup>2</sup> Department of Dermatology, Keck School of Medicine, University of Southern California, California, Los Angeles, USA

to managing patients who present with acute worsening of their HS symptoms.

## Challenges with Defining HS Flares

An HS flare is characterized as an acute and temporary worsening of HS with a return to baseline disease activity. The phase III adalimumab PIONEER trials introduced a clinical trial definition of HS flares as a  $\geq 25\%$  increase in the total abscess and inflammatory nodule (AN) count with a minimum increase of 2 AN relative to baseline [10]. This definition of HS flare has subsequently been used in other HS clinical trials as well [11, 12]. A 2019 Delphi consensus project involving clinicians, patients, and industry professionals defined HS flare as “new or substantial worsening of clinical signs or symptoms” [13]. In the literature, HS flares are generally described as acute worsening of disease activity compared to prior, but a standardized set of qualitative and quantitative criteria for evaluating flares is lacking. Prior to the Delphi definition, one cross-sectional study of 20 patients relied on physician ratings of erythema (0–3), patient ratings of pain (0–3), and morphologic changes on ultrasound of a single representative nodule to define flare activity [14]. A pilot study assessing vitamin D supplementation for 22 HS patients with vitamin D deficiency considered the possibility for recurrence by defining HS flares as the appearance of at least one new or reactivated nodule [15]. To further characterize physical manifestations of flares, a survey of 438 HS patients found the following symptoms to be the most common: pain (98.9%), drainage (91.8%), itch (81.8%), bleeding (78.8%), and increased lesions (66.7%) [16]. Emotional symptoms have also been described as a component of HS flares. A qualitative interview survey of 13 HS patients (ages, 31–58) found that emotional distress was often reported by patients as part of the constellation of flare symptoms [17]. Current definitions of HS flare struggle to integrate both subjective and objective descriptors and do not consider time to return to baseline disease, making it difficult to discriminate between acute and chronic manifestations.

## Healthcare Utilization from Flare Management

HS flares contribute to increased utilization of high-cost acute care settings among the HS patient population. A retrospective analysis of the National Emergency Department Sample found that annual mean expenditure for HS-related emergency department (ED) visits was \$11,507,481.30, with cost per visit averaging \$679.94 [18]. Providing

patients with anticipatory guidance and strategies for HS flare management at home as well as offering access to urgent appointments in the outpatient setting are critical to reduce ED visits and financial burden on both patients and the wider healthcare system [9].

## Triggers and Prevention Strategies

Identifying flare triggers of individual patients is a crucial step in mitigating HS exacerbations, but heterogeneous presentations of flares make recognizing their precipitating factors difficult. A survey study of 110 British patients with physician-diagnosed HS found that heat-related factors (45%), including sweat, heat, and exercise, were the leading cause of HS exacerbations, followed by stress (35%), tight clothing and friction (16%), and deodorants and cosmetics (13%) [19]. Among 742 survey participants across various Facebook support groups without a confirmed HS diagnosis, stress (81.3%), high carbohydrate diet (32.3%), exercise (31.9%), and weight gain (28.4%) were the most commonly reported exacerbators [20]. More recently, a large analysis of 1428 standardized intake forms at a single academic medical center found sweat (51.1%) and stress (47.8%) to be the most commonly reported triggers, followed by menstrual cycle (43.9%), heat (43.0%), and exercise (24.2%) [21]. While patient-reported data on HS flare triggers may be limited by recall bias, different sample populations appear to share commonalities.

A daily symptom journal can guide individualized recommendations for lifestyle modifications, and a summary of commonly reported HS flare triggers and preventative strategies are outlined in Table 1. In the United States, HS-related ED visits are most common during the summer months, likely due to seasonal exacerbation of heat, sweat, and friction, and multiple preventive approaches have been proposed in the literature [18, 40–43]. For patients who experience flares secondary to shaving, alternative hair removal methods such as trimming or laser hair removal have been found to decrease lesion counts [34–36]. Dairy, foods high in sugars, and brewer’s yeast have been documented in the literature as exacerbators of HS symptoms, and dietary counseling may be considered in these patients [22–29]. Weight loss of greater than 50 pounds among patients who are female or obese has been associated with HS remission, however new onset or worsening HS has also been reported as a post-operative complication of bariatric surgery, suggesting a multifactorial relationship between diet, weight, and HS [23, 30–33]. Conversations on smoking cessation are paramount, as smoking promotes inflammatory damage resulting in infundibular hyperkeratosis and follicular occlusion characteristic to HS and has been

**Table 1** Hidradenitis Flare Triggers and Prevention Strategies

Trigger	Prevention Strategies	References
Dietary	<ul style="list-style-type: none"> <li>•Track potential associations between specific foods and HS symptoms using daily journal or the Papaya digital application</li> <li>•Consider implementing Mediterranean diet</li> </ul>	[22–24]
Dairy	<ul style="list-style-type: none"> <li>•Plant-based alternatives to milk, cheese, and other dairy products, such as grain, nut, and seed milks, cashew cheeses, and coconut yogurt</li> <li>•Vitamin D supplementation, especially in the setting of vitamin D deficiency</li> </ul>	[25]
Foods high in sugars and fat	<ul style="list-style-type: none"> <li>•Substitute intake of sugary beverages with fruit-infused water, sparkling water, tea, zero-sugar soda</li> <li>•Counsel replacing high-glycemic index snacks with similar fruits and vegetables or minimally processed alternatives</li> </ul>	[25–27]
Brewer’s yeast	<ul style="list-style-type: none"> <li>•Encourage alternative carbohydrate sources, such as whole grains, legumes, oatmeal, unleavened breads (e.g. corn tortillas, matzo, lavash)</li> </ul>	[28, 29]
Lifestyle		
Weight management	<ul style="list-style-type: none"> <li>•Counsel on importance of balanced diet and healthy-weight management once rapport has been established</li> <li>•For patients with comorbid obesity, consider starting metformin, GLP-1 agonist therapy, or referring to bariatric surgery if appropriate</li> <li>•After bariatric surgery, ensure proper nutritional intake as HS flaring has been documented as a post-operative complication even in setting of weight loss</li> </ul>	[30–33]
Shaving and waxing	<ul style="list-style-type: none"> <li>•Recommend trimming instead of a clean shave (leaving ¼ inch of hair) or depilatory cream</li> <li>•Consider laser hair removal</li> </ul>	[34–36]
Smoking	<ul style="list-style-type: none"> <li>•Engage PCP and behavioral health services for additional support</li> <li>•Consider trial of nicotine gum and patch to curb cravings</li> </ul>	[37–39]
Heat, Sweat, and Friction		
Sweat and Friction	<ul style="list-style-type: none"> <li>•Recommend loose and breathable fabrics such as cotton, cellulose-derived rayon and bamboo fibers</li> <li>•Hyperhidrosis management</li> <li>•Topical antiperspirants are first line</li> <li>•While antiperspirant use may prevent frictional irritation in axillary region, deodorants have been reported to be associated with acute worsening of HS. Carefully track HS symptoms when introducing new antiperspirant agent</li> <li>•Consider oral glycopyrrolate and/or botulinum toxin injections</li> </ul>	[18, 40–43]
Exercise	<ul style="list-style-type: none"> <li>•Consider low-impact exercise (water aerobics, swimming, walking, yoga) if limited by pain</li> <li>•Recommend moisture-wicking, loose fabrics to prevent friction in intertriginous areas</li> </ul>	[42]
Hormonal		
Menses	<ul style="list-style-type: none"> <li>•Combined OCP with 3rd or 4th generation progestin</li> <li>•Spironolactone 50–200 mg daily</li> <li>•Finasteride can be considered as second-line treatment</li> </ul>	[44–46]
Pregnancy	<ul style="list-style-type: none"> <li>•Oral antibiotics and/or prednisone may be used</li> <li>•Intralesional steroid and I&amp;D procedures (with plain lidocaine) may be used</li> </ul>	[47]
Insulin Resistance	<ul style="list-style-type: none"> <li>•Work with primary care provider to manage comorbid metabolic and endocrine syndromes</li> <li>•For patients with concomitant metabolic syndrome, polycystic ovary syndrome, or type 2 diabetes, consider starting metformin or GLP-1 agonist</li> </ul>	[48]

*GLP-1* glucagon-like peptide-1, *HS* hidradenitis suppurativa, *I&D* incision and drainage, *mg* milligram, *OCP* oral contraceptive pill, *PCP* primary care provider

associated with a higher incidence of HS [37–39]. While the exact pathogenesis is poorly understood, hormonal fluctuations related to the menstrual cycle and pregnancy [44, 47] as well as endocrine dysfunction such as insulin resistance likely modulate HS disease activity and contribute to flares

[48]. In patients who report cyclic, perimenstrual worsening of HS symptoms, oral contraceptive pills, spironolactone, and finasteride can be considered [44–46].

In addition to verbal counseling, a written summary of flare management strategies may enhance patient confidence

in managing flares at home. In a pilot cross-over randomized clinical trial of 22 patients that received a verbal consultation and a HS-written action plan (HS-WAP) for home HS flare management, participants reported that the HS-WAP offered clarity on modifying treatment to accommodate changes in disease severity (18/22), presented a stepwise approach to treatment (15/22), improved understanding of the treatment regimen (14/22), and acted as a helpful daily reminder of treatment strategy (14/22) [49].

## Medical Management of HS Flares

Presently, there are limited guidelines for HS flare treatment, but monotherapy and multimodal approaches can be implemented by patients to alleviate flares at home. In Table 2, available treatments, their clinical indications, and documented adverse effects are summarized for clinician use.

## Topical Agents

### Clindamycin

Topical clindamycin has the highest rated level of evidence according to the 2019 North American (NA) guidelines for HS [50]. Clindamycin 1% can be applied to actively flaring HS-affected areas once or twice daily. Topical clindamycin performed better than placebo based on patient assessment and AN count in a double blind randomized controlled trial of 30 patients [51].

### Resorcinol

Resorcinol 15% is recommended by the NA guidelines as a rescue therapy for flares [50]. In a case series of patients with Hurley stage 1 or 2 HS, all 12 patients reported reduced pain and decreased duration of painful lesions when applying resorcinol 15% cream twice daily within hours of flare onset. Adverse effects include irritant dermatitis and reversible hyperpigmentation [52]. Notably, in a study of 134 patients, topical resorcinol 15% cream demonstrated a significant improvement ( $p < 0.001$ ) in International Hidradenitis Suppurativa Severity Score System (IHS4), pain Visual Analogue Scale (VAS) score after 12 weeks of treatment without concern for antimicrobial resistance, and Hidradenitis Suppurativa Clinical Response 50 (HiSCR50), defined as a  $\geq 50\%$  reduction from baseline AN with no new abscesses or tunnels, was achieved by significantly greater proportion ( $p < 0.001$ ) of patients on topical resorcinol 15% cream compared to clindamycin 1% gel or cream [53].

### Clascoterone

Clascoterone is a topical androgen receptor inhibitor that is FDA-approved for acne vulgaris [54]. In a case series of 12 patients with Hurley stage 1 or 2 HS treated with clascoterone cream, ten experienced improvement on clascoterone 1% cream based on patient and/or clinician evaluation after a median follow-up time of 12 weeks [55]. There were no side effects or adverse events [55], although a larger trial for facial acne vulgaris found application site irritation to be among treatment-emergent adverse events (TEAE) reported in 9 of 369 participants [54].

### Ruxolitinib

Ruxolitinib is a Janus kinase (JAK) inhibitor that has FDA-approved indications for atopic dermatitis and vitiligo [56, 57]. In phase II clinical trials of 69 adult HS patients with Hurley stage 1 or 2, 79.2% of participants on ruxolitinib 1.5% achieved  $\geq 50\%$  reduction in AN (AN50) compared to 50% of participants in the placebo group, and a greater mean reduction in IHS<sub>4</sub> was observed at week 16 in the treatment group (-4.46) compared to the placebo group (-2.66) [58]. COVID-19 (5.9%) and nasopharyngitis (5.9%) were the most reported among 38.2% of participants reporting TEAE, but only 2 participants (5.9%) discontinued treatment due to TEAE [58]. At present, phase 3 trials are pending further evaluation of ruxolitinib 1.5% cream's safety and efficacy for HS (NCT# 06959225, 06958211).

## Systemic Antibiotics

For decades, systemic antibiotics have been a mainstay treatment for HS due to their quick onset anti-inflammatory properties. The treatment paradigm regarding systemic antibiotics has shifted away from using systemic antibiotics as a long-term therapy and instead to consider them for flare therapy or as a bridge to appropriate long-term therapeutic options [89]. Limitations of long-term use of antibiotics include that disease recurrence is common after stopping treatment, and concern for the development of antimicrobial resistance [50]. Antibiotics are commonly prescribed for two-week courses for flares. Dosing recommendations and clinical considerations for specific antibiotic agents can be found in Table 2.

### Monotherapy

Single antibiotics agents that can be utilized in the management of acute HS flares include tetracyclines (i.e. doxycycline, minocycline), trimethoprim-sulfamethoxazole

**Table 2** Pharmacologic, Procedural, and Complementary Alternative Medicine Approaches to HS Flare Management

Agent	Dosing and Administration	Clinical Considerations	References
Clindamycin (Topical)	1% gel, foam, liquid, or lotion Apply to affected areas BID	•Use with benzoyl peroxide or chlorhexidine wash to minimize risk of antimicrobial resistance	[50, 51]
Resorcinol (Topical)	15% cream or ointment Apply BID at flare onset	•Potential AE: irritant dermatitis, reversible hyperpigmentation •Use with caution when open wounds present	[50, 52, 53]
Clascoterone (Topical)	1% cream Apply to affected area BID	•Potential AE: application site irritation	[54, 55]
Ruxolitinib (Topical)	1.5% cream Apply to affected area BID	•Phase 3 clinical trials pending further evaluation of safety and efficacy for HS	[56–58]
Tetracyclines	Tetracycline 500 mg BID Doxycycline 100 mg BID Minocycline 100 mg BID	•Advise patients to wear sunscreen to protect against phototoxicity •Avoid in children <8 years old •Gastrointestinal symptoms are common; recommend taking with food and water, but avoid simultaneously consuming with dairy or other high-calcium foods	[50, 59, 60]
Clindamycin	300 mg BID	•GI upset, such as nausea and diarrhea, is the most documented adverse effect • <i>Clostridioides difficile</i> infection •Traditionally used as combination therapy with rifampin, however recent literature suggests clindamycin monotherapy has comparable efficacy	[50, 59–61]
Trimethoprim-Sulfamethoxazole	800–160 mg BID	•Screen for sulfa drug allergies prior to starting •Avoid in pregnancy due to risk for folate deficiency	[62]
Cephalexin	500 mg BID	•Screen for allergies to beta-lactam antibiotics due to potential cross-reactivity	[63, 64]
Fluoroquinolones	Ciprofloxacin 500 mg BID Moxifloxacin 400 mg daily	•Possible adverse effects: gastrointestinal upset, dizziness, rapid heart rate, urticaria, and musculoskeletal pain •May cause QTc prolongation, exercise caution for patients with preexisting arrhythmias or taking other pharmacologic agents that prolong QT interval •Concomitant systemic corticosteroid therapy may increase risk for tendon rupture	[59, 65, 66]
Amoxicillin/clavulanic acid	500–125 mg or 875–125 mg BID	•10-day course raises very little risk for antimicrobial resistance. •Consider concomitant 40 mg prednisone taper (decrease by 10 mg every 3 days)	[67]
TMP-SMX and Cephalexin	TMP-SMX 800–160 mg BID Cephalexin 500 mg BID	•As above for monotherapy	[68]
Rifampin-Moxifloxacin-Metronidazole	Rifampin 300 mg BID Moxifloxacin 400 mg daily Metronidazole 500 mg BID	•Recommend prophylactic probiotics to lower risk of gastrointestinal disorders and vaginal candidiasis •Tendinitis secondary to fluoroquinolone therapy has been observed in this combination •Rifampin may cause LFT elevations and have drug-drug (e.g. anticonvulsant therapy) •Metronidazole may cause a disulfiram-like reaction with ethanol intake	[50, 68, 69]
Ciprofloxacin and Metronidazole	Ciprofloxacin 500 mg BID and metronidazole 500 mg daily may be considered	•Not trialed in HS patients but demonstrated anti-inflammatory effect on perianal abscesses of CD patients. •As above with RMoM therapy and fluoroquinolone monotherapy	[70]
Ertapenem	1 g IV (or IM) × 6–13 weeks	•Complications can occur due to PICC line (e.g. infection, DVT) •Use with caution in patients ≥65 years, with renal insufficiency, with lower seizure thresholds, or on anticonvulsant therapy, as convulsions and confusional states are commonly reported	[50, 59, 71–74]
Piperacillin-tazobactam	Three doses of IV piperacillin-tazobactam (12 g/1.5 g) as tolerated or until clinical remission reached	•Very limited data	[75]

**Table 2** (continued)

Agent	Dosing and Administration	Clinical Considerations	References
Prednisone	0.5 mg/kg and 1 mg/kg tapered over 1–4 weeks	<ul style="list-style-type: none"> <li>•Reported side effects include hyperglycemia, mild psychomotor agitation, and sleep disturbances</li> <li>•Long-term use can predispose patients to adrenal insufficiency and avascular necrosis</li> <li>•Consider 40 mg prednisone taper (decrease by 10 mg every 3 days) with a 10-day course of amoxicillin-clavulanic acid (dosing as above)</li> </ul>	[50, 63, 67, 76]
Triamcinolone	40 mg IM	<ul style="list-style-type: none"> <li>•May consider in place of prednisone taper</li> </ul>	[77]
Topical NSAID	Diclofenac 1% gel $\leq$ 2 g per site $\leq$ 4 times daily up to 32 g per day	<ul style="list-style-type: none"> <li>•Potential AE: application site reaction</li> </ul>	[78, 79]
Topical lidocaine	4–5% ointment $\leq$ 4 times daily	<ul style="list-style-type: none"> <li>•Potential AE: application site reaction</li> </ul>	[78, 79]
Topical menthol	Apply $\leq$ 4 times daily	<ul style="list-style-type: none"> <li>•Potential AE: application site reaction</li> </ul>	[78, 79]
Acetaminophen	325–1000 mg every 3 hours; up to 4 g daily	<ul style="list-style-type: none"> <li>•Exercise caution in elderly patients and those with preexisting liver disease</li> </ul>	[78, 79]
Systemic NSAID	Ibuprofen 200–800 mg every 4–8 hours, up to 2400 mg per day Naproxen sodium initial dose 550 mg Ketorolac initial dose of 20 mg then 10 mg every 4–6 hours thereafter	<ul style="list-style-type: none"> <li>•NSAIDs should be avoided in patients with bleeding risks and renal disease</li> <li>•Consider supplementing with proton pump inhibitors to protect gastric lining</li> </ul>	[78, 79]
Tramadol	50–100 mg every 4–6 hours as needed	<ul style="list-style-type: none"> <li>•Opioid medications should be given in small quantities (~20 pills) with no refills</li> <li>•Potential AE: GI disturbances, most notably constipation</li> <li>•If longer-term treatment with opioids is required, consider connecting patient to pain management specialist</li> </ul>	[78, 79]
Warm compresses	Soak washcloth in warm water and apply to HS lesion for 10 minutes as needed	<ul style="list-style-type: none"> <li>•Both dermatologists and HS patients report that warm compresses are more helpful than cool compresses for flares.</li> </ul>	[63, 80, 81]
Intralesional corticosteroid injections	Triamcinolone 10–40 mg/mL injected intralesionally, volume variable	<ul style="list-style-type: none"> <li>•May cause hypopigmentation, atrophy, and telangiectasias at the injection site</li> <li>•High-dose ILTAC (20–40 mg/mL) may provide superior outcomes compared to ILTAC 10 mg/mL</li> </ul>	[82–85]
Punch I&D	Consider using a punch tool (4–6 mm) instead of a #11 blade to better control depth of incision and allow continued drainage post-procedure	<ul style="list-style-type: none"> <li>•Ensure adequate anesthesia prior to incision</li> <li>•Avoid packing lesion after I&amp;D</li> </ul>	[86–88]

*AE* adverse event, *BID* twice daily, *CD* Crohn's Disease, *DVT* deep vein thrombosis, *g* gram, *GI* gastrointestinal, *HS* hidradenitis suppurativa, *I&D* incision and drainage, *ILTAC* intralesional triamcinolone *IM*, intramuscular, *IV* intravenous, *kg* kilogram, *LFT* liver function test, *mg* milligram, *mL* milliliter, *mm* millimeter, *NSAID* non-steroidal anti-inflammatory drug, *PICC* peripheral intravenous central catheter, *QTc interval* heart rate-corrected QT interval, *RMoM* rifampin-moxifloxacin-metronidazole, *TMP-SMX* trimethoprim-sulfamethoxazole

(TMP-SMX), clindamycin, cephalosporins, and amoxicillin-clavulanate. A 2024 survey of 35 HS experts found that top prescribed antibiotics for HS flares included tetracyclines (91.4%), TMP-SMX (62.9%), and amoxicillin-clavulanate (60%) [63].

Oral tetracycline therapy for 12 weeks is recommended by the NA and European S2k clinical management guidelines as a first-line therapy for mild to moderate HS (HS stages 1 and 2) [50, 59]. A large international cohort study ( $n=283$ ) found oral tetracycline therapy (tetracycline 500 mg twice daily, doxycycline 100 mg daily, or minocycline 100 mg

daily for 12 weeks) resulted in a statistically significant ( $P<0.001$ ) decrease in  $IHS_4$  and achievement of  $HiSCR_{50}$  in 40.1% of patients [60].

In a retrospective case series of 18 HS patients on varied regimens of TMP-SMX, 50% of achieved an excellent response, defined as a clear or minimal Hidradenitis Suppurativa Physician Global Assessment (HS-PGA) score, and 38.9% demonstrated a decrease in HS-PGA but not to clear or minimal levels [62].

A prospective observational HS flare study investigated outcomes from a combination of 875 mg–125 mg

amoxicillin-clavulanate twice daily for ten days and 40 mg prednisone taper (10 mg decrease every 3 days) in 44 patients [67]. Significant improvements at day 3, day 7, and day 14 of treatment were seen in patient Dermatology Life Quality Index (DLQI), patient-global-impression-of-severity (PGI-S), and pain scores [67]. This study had promising results for amoxicillin-clavulanate and prednisone combination therapy as an effective, fast-acting strategy for flare management with minimal risk for the development of antimicrobial resistance [67].

There are no studies assessing the efficacy of cephalosporin monotherapy in HS flare management, but in the HS expert flare survey, 34.3% and 14.3% of HS experts reported prescribing cephalexin and cefdinir, respectively [63]. For first-generation cephalosporins like cephalexin with identical R1 side chains to an aminopenicillin, there is around a 16.5% risk of cross-reactivity in patients with penicillin allergy, but this risk is estimated to be lower for third generation cephalosporins like cefdinir [64].

Clindamycin is traditionally administered as a combination therapy with rifampin (RC therapy), but a nonrandomized retrospective cohort study of 60 patients with mild-to-moderate HS found that clindamycin monotherapy (150 mg, four times daily) may have similar efficacy to RC therapy (150 mg clindamycin, four times daily with 300 mg rifampin, twice daily) across all Hurley stages [61].

The European S2k guidelines classify all fluoroquinolone regimens as experimental [59]. Once daily dosing of 400-mg moxifloxacin makes it a more convenient option for monotherapy compared to other fluoroquinolones. In a retrospective cohort study of 39 patients, 20.5% and 51.3% achieved an excellent or partial response after taking 400-mg moxifloxacin for a median course of 25.9 weeks, respectively [65]. An “excellent response” was defined as a clear to minimal HS-PGA score, and all other HS-PGA improvements were graded as a “partial” response [65]. Among the 11 participants who did not respond to moxifloxacin monotherapy, disease worsening and adverse effects, including dizziness, tachycardia, urticaria, and gastrointestinal upset, were observed in 4 and 7 participants, respectively [65]. Risk factors for serious adverse effects should be screened prior to initiating therapy [66].

## Combination Therapy

According to the NA clinical guidelines, RC may be prescribed 8–12 weeks and can be reintroduced for acute symptom management or as adjuvant therapy in more severe disease [50]. The S2k guidelines recommend RC therapy as first-line treatment for moderate to severe HS because its efficacy is supported by multiple studies [59]. A prospective cohort study of 283 participants on 300-mg

clindamycin twice daily and 600-mg rifampin daily for 12 weeks reported significant decreases in average DLQI (15.1 to 9.8), median numeric rating score (NRS) pain (7 to 3), median NRS pruritus (4 to 1.0), and median IHS<sub>4</sub> (13.0 to 6.0) after treatment [60].

The NA guidelines recommend rifampin-moxifloxacin-metronidazole (RMoM) triple therapy as second or third line for severe HS [50]. In a 2023 prospective study of 28 patients with Hurley stage 1 disease on rifampin (10 mg/kg once daily), moxifloxacin (400 mg daily), and metronidazole (250–500 mg three times daily) for 6 weeks followed by a 4-week course of rifampin and moxifloxacin alone, 75% of patients reached clinical remission, defined as a Sartorius score of 0 [68]. Lower baseline Sartorius scores were associated with clinical remission ( $p=0.049$ ), corroborating the results of a previous study associating mild disease with better clinical response to RMoM therapy [68, 69]. Notably, patients reported a median of 21 flares annually (range, 12–52) at baseline and reported 1 flare (range, 0–16) per year at 1-year follow-up [68]. Ciprofloxacin and metronidazole have not been evaluated for efficacy in HS treatment but have been shown to facilitate clinical remission in Crohn’s Disease (CD) and decrease perianal abscesses in the setting of CD [70].

One case series of 16 patients treated with a three-month course of TMP-SMX (400–80 or 800–160 mg twice daily) and cephalexin (250–500 mg twice daily) showed promise as an alternative dual therapy antibiotic regimen to clindamycin-rifampin [90]. At the end of the treatment period, 75% and 68.8% of patients showed improvement per provider assessment and self-assessment, respectively [90]. Diarrhea and pruritic rash were reported by two patients [90].

## Intravenous (IV) Antibiotics

IV ertapenem therapy is recommended for severe or recalcitrant HS as a third-line rescue therapy or for medical optimization prior to surgery [50, 59].

In the context of flare management, IV ertapenem may be considered in an inpatient setting to mount rapid, rigorous response to flare activity with systemic involvement prior to initiating long-term bridge therapy or curative surgery. A 2018 case study of a 21-year-old male with Hurley stage 3 HS presenting with recurrent fever, chills, and rigors concerning for sepsis was admitted then put on IV ertapenem (unknown dose regimen) for 3 days, after which his fever had resolved prior to transitioning to a 6-week course of IV ertapenem at home [71].

To our knowledge, IV antibiotics for HS flare control have primarily been assessed as a home regimen while its role in rapid HS flare control in an inpatient setting remains largely unstudied. A 2024 retrospective cohort study assessed 98

patients with suboptimal responses to past pharmacologic interventions and severe disease activity on 1 g IV ertapenem daily at home for a mean duration of 13 weeks, and significant decreases ( $p < 0.001$  for each of the following) in HS-PGA score, numerical rating scale for pain, C-reactive protein (CRP), interleukin-6 (IL-6), and leukocyte counts were observed between baseline and post-therapy follow-up (6–8 weeks after end-of-course) [72]. Nine patients required peripheral intravenous central catheter (PICC) replacement due to dislodgement, impaired flow, or PICC-associated infections, but none of these patients required treatment cessation [72].

A small cohort of 5 patients were given 1 g intramuscular (IM) ertapenem daily, which led to significant post-therapy reduction in IHS<sub>4</sub> and pain VAS scores at 6 weeks ( $p = 0.018$  and  $p = 0.0014$ , respectively) and 12/16 weeks ( $p = 0.011$  and  $p = 0.020$ , respectively) [73]. IM ertapenem may reduce costs and PICC-related risks associated with IV ertapenem and merits further study though daily injections and injection pain may be a limiting factor [73]. Confusional state and convulsions are well-documented adverse effects of ertapenem, which should be used with caution in patients  $\geq 65$  years old, with renal insufficiency, and with lower seizure thresholds [74].

Preliminary data on piperacillin-tazobactam also suggests it has potential as a third-line therapy for refractory, severe disease. Among nine patients with Hurley stage 2 or 3 disease refractory to standard systemic antibiotic and biologic treatment and associated with flares, three doses of IV piperacillin-tazobactam (12 g/1.5 g) were administered during hospitalization over a mean duration of 9.1 days (range, 6–21 days) [75]. HiSCR50 was achieved by 6 patients at 3-month follow-up and 5 patients at 6-month follow-up, and there were no reports of adverse effects [75].

## Systemic Corticosteroids

### Oral Prednisone

The NA guidelines recommend short-term oral corticosteroid pulse therapy for flare management and bridge to long-term therapy or surgery [50]. Prednisone in combination with amoxicillin-clavulanate as flare therapy was described above. Using a systemic steroid alone may be particularly advantageous in patients who are unable to tolerate systemic antibiotics. A 2016 case series found that 11 of 13 patients experienced improvement in their PGA score while taking 2.5–10 mg prednisone daily as an adjuvant to other systemic therapies, most commonly adalimumab, RC therapy,

doxycycline monotherapy, and dapsone monotherapy [76]. The HS expert flare survey found that 20/33 (60.6%) experts typically start oral prednisone dosing between 0.5 mg/kg and 1.0 mg/kg, and 66.7% typically treat for a duration of treatment of 2 weeks or less [63].

### Intramuscular Steroid Injections

Intramuscular triamcinolone acetonide (IMTAC) is an alternative treatment modality for widespread flare. According to a retrospective analysis and survey of 45 HS patients who were treated with one session of IMTAC (40 mg/ml,  $\leq 160$  mg per session), significant reductions in HS-PGA scores ( $p < 0.001$ ), C-reactive protein (CRP) levels ( $p = 0.03$ ), and subjective pain scores ( $p < 0.001$ ) were observed at follow-up (mean, 6.77 weeks; standard deviation, 4.45 weeks), and 93% of patients reported being willing to try IMTAC again [77].

## Procedural Management

### Intralesional Steroids

Intralesional triamcinolone acetonide (ILTAC) has been widely studied as a procedural intervention for acutely inflamed nodules or tunnels. Dosing (10–40 mg/mL) and volume injected into each lesion varies based on the degree of inflammation and anatomical region affected [82]. In a prospective case series of 36 patients who received mean volume 0.75 mL (range, 0.2–2.0 mL) ILTAC 10 mg/mL, significant reductions ( $p < 0.0001$ ) in physician-assessed erythema, edema, suppuration, and size were noted at 7-day follow-up [83]. Reductions in self-reported pain measured using pain visual analog scale scores were most significant one ( $P < 0.005$ ) and two days ( $P < 0.002$ ) post-injection [83]. A 2020 prospective open-label study of 46 HS patients who received a single injection (mean: 0.49 mL, range: 0.2–1.0 mL) of ILTAC 40 mg/mL for single fistulous tracts found that mean length measured by clinical and ultrasound measurement decreased from 17.0 to 5.1 ( $p < 0.0001$ ) and 16.0 to 8.6 ( $p < 0.0001$  at 90-day follow-up, respectively) [84]. However, adverse effects included pigmentation changes (54.3%) and skin atrophy (37.0%) [84]. A more recent retrospective chart review of 54 HS patients who received high-dose ILTAC (20 mg/mL and/or 40 mg/mL) found that 76.9% were satisfied or very satisfied with treatment, 92.6% of patients saw improvement of symptoms, 75.9% of patients reported improved QOL, which may suggest superior outcomes compared to ILTAC-10; there were no reported adverse events [85].

## Punch Incision and Drainage (Punch I&D)

Generally, I&D is intended to provide symptomatic relief for expanding, painful abscesses. In a survey of patients with HS who had received more than one I&D procedure, approximately one-fourth of 233 respondents reported full resolution of lesions after I&D procedures, but 66.1% described either lesions that “failed to improve” or recurrence that occurred >1 week after initial improvement [86]. Adequate pain control and gentle incision were associated with greater benefit [86]. Utilizing topical and/or intral-lesional anesthesia, and icing the area, may improve patient experiences with I&D procedures [86, 87]. In place of a scalpel, the use of a 4–8 mm punch tool provides more depth control, allows for continued drainage post-procedure, and leaves a circular scar less likely to close prematurely compared to the linear scar left by a scalpel [87]. Although I&D cavities are traditionally filled with packing, a recent meta-analysis of 8 randomized controlled trials evaluating outcomes of cutaneous abscesses status post I&D discerned no significant difference in risk of recurrence, fistula in-ano, or need for second intervention with or without packing [88]. Therefore, it is not currently recommended to pack HS abscesses after I&D.

## Acute Pain Management and CAM

### Pharmacologic Approaches to Acute Pain Management

Acute pain management strategies for HS have been published in the literature, incorporating pain management principles from other conditions and are summarized in Table 2. First-line medications for mild pain are acetaminophen and topical analgesics, such as diclofenac, topical lidocaine, and menthol. Moderate pain may be managed with acetaminophen and non-steroidal anti-inflammatory drugs (NSAIDs), such as aspirin, ibuprofen, and naproxen [78]. A short course of immediate-acting opioids, such as tramadol, hydromorphone, or oxycodone can be considered for patients with severe pain, but a referral to pain management should be made prior to starting regular opioid therapy [79].

### Complementary and Alternative Medicine (CAM)

Use of common CAM therapies are well-documented for other dermatologic conditions, such as psoriasis and dermatitis but are not as well-studied in HS patients. Among 50 surveyed dermatologists, 19 of whom self-identified as

HS experts, the following home remedies for flare were recommended: warm compresses (76%), bleach baths (50%), zinc oxide cream (28%), Epsom salt baths (18%), and Vicks VapoRub (12%) [80]. In the 2024 HS expert flare management survey, warm compresses (rank: 6/13, 68.6%) and cold compresses (rank: 12/13, <25%) were among the top 13 medical, procedural, and CAM flare management approaches by experts [63]. Another 2023 survey of 900 self-reported HS patients, found that CAM (rank 2/12), including but not limited to oral supplements, medicated baths, and massages, was perceived as more helpful for flares compared to warm compresses (rank 3/12) and cold compresses (rank 10/12) among all flare management strategies [81].

## Conclusion

HS is an inflammatory skin disease with debilitating symptoms such as pain, drainage, and itch that acutely worsen in the setting of a flare. Educating patients about possible triggers and encouraging careful monitoring of events leading up to HS flares can help with flare prevention. Longitudinal monitoring of patients with HS is important for medical optimization, especially to evaluate whether a patient’s symptoms are indicative of intermittent flaring or persistent decline. Because flares can have prohibitive effects on activities of daily living, educating patients on strategies for flare management at home and improving accessibility to care via telemedicine appointment and urgent appointment slots should be a priority. The multimodal approach to HS treatment should ensure good control of underlying disease activity supplemented by flare interventions when needed. Further investigation is needed to study efficacy of HS flare treatments.

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## Declarations

ABC has served as an investigator for Incyte and AstraZeneca. JLH is on the Board of Directors for the Hidradenitis Suppurativa Foundation and has served as an advisor, investigator, and/or speaker for AbbVie, Amgen, Aclaris, AstraZeneca, Boehringer Ingelheim, Galderma, Incyte, Navigator Medicines, Novartis, Pfizer, Sanofi, Regeneron, UCB. KHL has served as an advisor and/or investigator for Novartis, Incyte. The rest of the authors have no conflicts to declare.

**Human and Animal Rights** This article does not contain any studies with human or animal subjects performed by any of the authors.

**Competing Interests** ABC has served as an investigator for Incyte and AstraZeneca. JLH is on the Board of Directors for the Hidradenitis Suppurativa Foundation and has served as an advisor, investigator, and/or speaker for AbbVie, Amgen, Aclaris, AstraZeneca, Boehringer Ingelheim, Galderma, Incyte, Navigator Medicines, Novartis, Pfizer, Sanofi, Regeneron, UCB. KHL has served as an advisor and/or investigator for Novartis, Incyte. The rest of the authors have no conflicts to declare.

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