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THERAPY IN PRACTICE

Comprehensive and Updated Algorithm of Hidradenitis Suppurativa Management from the Experts

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Abstract

Management of hidradenitis suppurativa (HS) can be challenging and often requires a multimodal approach with use of on- and off-label medications. There has been a rapid expansion of available HS treatments in the years since the 2019 North American HS (NAHS) clinical management guidelines. Herein we present an up-to-date practical management algorithm based on the diagnosis and management strategies set forth by the 2019 NAHS guidelines using newly available literature. Evaluation and diagnosis of HS disease involves assessment of severity, extent of disease, and impact on patient quality of life. Initial diagnosis of HS should be shortly followed by comorbidity screening. The multimodal approach to HS treatment typically involves use of treatment stacking of topical therapies, systemic and topical antibiotics, retinoids, hormonal and metabolic therapies, biologics and small molecule inhibitors, systemic immunosuppressants, surgical treatment, pain management, lifestyle modifications, adjunctive treatment, wound care, and flare therapy. Thus, the proposed algorithm aims to guide clinicians in their implementation of treatment stacking in HS.

1 Introduction

Hidradenitis suppurativa (HS) is a chronic inflammatory dermatosis characterized by the formation of abscesses, inflammatory nodules, and tunnels. Management of HS can be challenging and often requires a multimodal approach with use of off-label medications. The North American HS clinical management guidelines (NAHS guidelines) were published in 2019 to help provide clinicians with an evidence-based algorithmic approach to treatment [1]. This was followed by the publication of a comprehensive textbook guide to HS in 2022 [2]. As of November 2024, bimekizumab (BZK), an interleukin (IL)-17A and IL-17F inhibitor, gained US Food and Drug (FDA)-approval to join adalimumab (ADA), a TNF-inhibitor, and secukinumab (SEC), an IL-17A inhibitor, as the FDA-approved agents for HS. With the recent expansion of the clinical trial landscape and a multitude of biologics and small molecule inhibitors under investigation, a new horizon of treatments for HS is approaching [3, 4]. In addition, a recent publication from the European Hidradenitis

Key Points

Management of hidradenitis suppurativa (HS) often requires complex treatment plans, including the use of on- and off-label medications. Herein, an up-to-date practical management algorithm is proposed to assist in HS treatment plan design.

Diagnosis and evaluation of HS involves the identification of typical HS lesions, assessment of disease severity and activity level, as well as use of patient reported outcome measures.

HS treatment involves treatment stacking of interventions including but not limited to topical therapies, systemic antibiotics, hormonal and metabolic treatments, biologics, pain management, wound care, lifestyle modifications, and surgical interventions.

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Suppurativa Foundation provides updated evidence on the treatment of HS compared with the 2015 guidelines [5]. Despite recent advancements in treatment options and evidence, an updated management algorithm is not yet available. Herein we present an up-to-date and practical management algorithm based on existing literature, new scientific evidence, and our expert experience (Fig. 1). This narrative review provides an updated discussion on diagnosis and management strategies set forth by the 2019 NAHS guidelines [1, 6].

2 Diagnosis and Evaluation

Diagnosis of HS is made clinically with the identification of three diagnostic criteria, including (1) presence of typical HS lesions, (2) lesions in typical locations such as intertriginous sites (e.g., axilla, inframammary regions, and inguinal folds), and (3) chronic nature of disease with relapse and reoccurrence [7–10]. It should be noted that HS can occur outside these "typical" locations anywhere there are hair follicles, including the posterior ears and trunk. HS lesions include papules, nodules, abscesses, ulcers, comedones, tunnels, and sometimes pustules and plaques. Validated consensus definitions for the abovementioned morphological HS lesions have been established to help standardize nomenclature [11]. Notably, European efforts to establish standardized nomenclature include additional terminology, including cord and bridge [12, 13].

The Hurley staging system is the most widely used assessment tool [14]. Severity of disease is graded as mild, moderate, or severe on the basis of the presence of cicatrization and degree of regional involvement. However, the Hurley system was initially proposed as a surgical grading scale and does not necessarily reflect disease activity. Numerous other HS assessments tools have been proposed but many have limited practicality in the clinical setting owing to their inability to track treatment response [15]. Collaborative groups, including the Hidradenitis Suppurativa Core Outcomes Set International Collaboration (HISTORIC) and International Dermatology Outcome Measures (IDEOM), aim to develop standardized core outcome sets and standardized outcome measures for HS, respectively. At this time, selection of the assessment tool remains clinician dependent, but effort should be made to incorporate disease activity, number of anatomic regions involved, extent of tissue destruction, and patient-reported outcomes (PROs) when formulating a therapeutic approach. PROs are an important aspect of evaluation to help guide shared decision-making and can be measured utilizing tools such as the Dermatology Quality of Life Index (DLQI), Skindex-Mini, or a more specific HS Quality of Life (HiSQOL) scale [16–18]. HiSQOL is the preferred PRO measure as it offers a thorough assessment of how HS affects each patient's life. Figure 1 demonstrates the use of DLQI and HS Physician Global Assessment (PGA) to guide therapy.

3 Comorbidity Screening

The US and Canadian HS Foundations recommend comorbidity screening for dermatologic and nondermatologic comorbidities. Dermatologic comorbidities of HS include acne, dissecting cellulitis of the scalp, pilonidal disease, and pyoderma gangrenosum. Nondermatological comorbidities include depression, generalized anxiety disorder, suicide, smoking, substance use disorder, polycystic ovary syndrome, obesity, dyslipidemia, diabetes mellitus, metabolic syndrome, hypertension, cardiovascular disease, inflammatory bowel disease, spondylarthritis, and sexual dysfunction [19]. Collaboration with primary care providers, including gynecologists, is instrumental in the proper screening and management of these comorbidities. Discussing all comorbidities at the time of a first encounter may not be feasible owing to time limitations but each is important to consider for patients over time. Nondermatologic comorbidities of interest that can be screened in the dermatology clinic include anxiety, depression, smoking, inflammatory bowel disease, spondyloarthritis, and sexual dysfunction. Depression and anxiety can be screened for in the dermatology office using Patient Health Questionnaire-9 (PHQ-9) and the Generalized Anxiety Disorder-7 (GAD-7), respectively. Frequency of comorbidities and approach to screening can be found in Garg et al. [19] Of special note, all patients presenting to dermatology clinic with Trisomy 21 should be screened for HS, as patients may have a five times higher risk [20].

4 Medical and Surgical Management: Treatment Stacking

A multimodal approach and treatment stacking of both medical and surgical interventions are essential to optimize management of HS given its proposed multifactorial etiology. Treatment stacking refers to the layering of therapeutic interventions from multiple therapeutic categories [21]. Monotherapy is often insufficient to control disease. Thus, selecting multiple targeted interventions to address the various pathophysiologic contributors such as follicular occlusion, dysbiosis, metabolic and hormonal dysfunction, and immune dysregulation is recommended. Figure 1 serves as an algorithmic guide to treatment stacking.

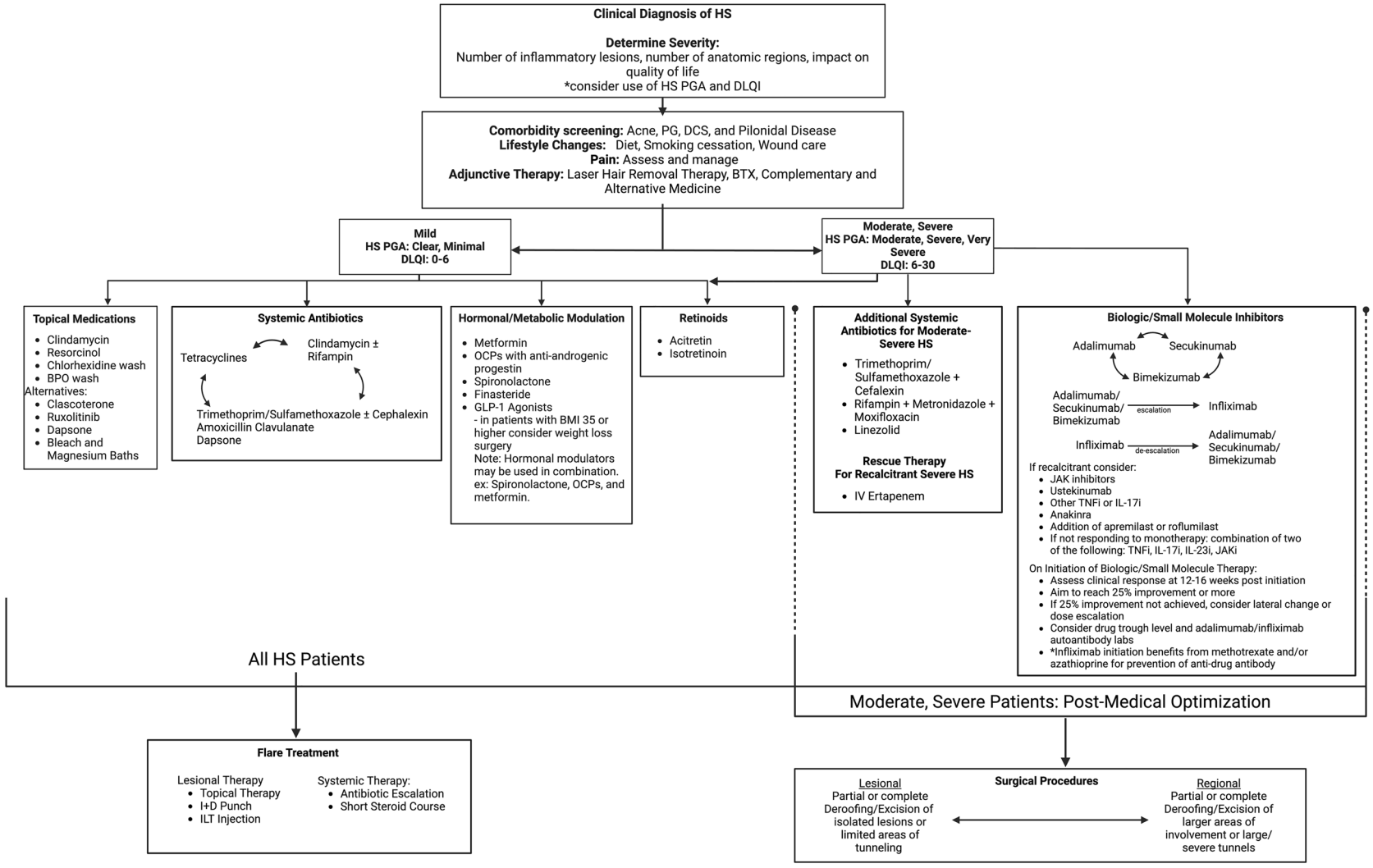

Fig. 1 Hidradenitis suppurativa treatment algorithm. *HS* hidradenitis suppurativa, *HS PGA* hidradenitis suppurativa Physician Global Assessment, *DLQI* Dermatology Life Quality Index, *PG* pyoderma gangrenosum, *DCS* dissecting cellulitis of the scalp, *BTX* botulinum toxin, *BPO* benzoyl peroxide, *OCPs* oral contraceptive pills, *GLP-1* glucagon-like peptide-1, *IV* intravenous, *I+D* incision and drainage, *ILT* intralesional triamcinolone; created with Biorender.com

5 Topical Therapies

Topical medications should be initiated for patients with HS of all severities. Topical clindamycin 1% solution to HS-affected areas twice daily is recommended on the basis of controlled trials [22, 23]. The NAHS guidelines recommend benzoyl peroxide, chlorhexidine, zinc pyrithione, resorcinol, and dapsone on the basis of limited evidence and expert consensus [1].

Newer data have emerged regarding the use of resorcinol 10% cream, clindamycin-benzoyl peroxide gel, and topical clascoterone. In a prospective, randomized, open trial, resorcinol 10% cream demonstrated clinically significant improvement of total HS lesions when compared with topical clindamycin 1% and control patients ($p = 0.017$ and $p < 0.001$, respectively) [24]. In a randomized controlled study, clindamycin-benzoyl peroxide gel had similar clinical efficacy to monotherapy clindamycin and provided the added benefit of antibiotic resistance prevention [25]. Clascoterone may be helpful in reducing HS nodule count and severity based on a small, uncontrolled retrospective chart review ($n = 12$) [26].

A phase 2 double-blind, vehicle-controlled trial for the use of topical ruxolitinib in HS was recently completed. According to preliminary data, approximately 80% of patients treated with topical ruxolitinib achieved Hidradenitis Suppurativa Clinical Response 50% (HiSCR50) (NCT05635838) [27]. Bleach and magnesium sulfate baths have yet to be studied for HS specifically.

6 Systemic Antibiotics

Systemic antibiotics are used short-term for alleviation of flares or for longer courses as a bridge to slower-acting therapies. The NAHS guidelines recommend monotherapy for mild disease and combination therapy as second-line or as adjunctive treatment in more severe disease [1]. Tetracyclines remain first-line and can be used for a 12-week course or as long-term maintenance therapy. Historically, clindamycin has been recommended in combination with rifampin. However, recent literature suggests that clindamycin monotherapy may has similar efficacy to combination therapy [28]. For moderate-to-severe disease, combination moxifloxacin, metronidazole, and rifampin can be prescribed [1]. In addition, combination trimethoprim-sulfamethoxazole and cephalexin has been shown to be effective in Hurley stage II and III disease [29]. Dapsone is effective in some cases of mild or moderate disease as a long-term therapeutic option. A 6–16 week course of intravenous (IV) ertapenem is reserved as rescue therapy for severe disease or as a bridge to surgery or other long-term treatment [30]. If systemic antibiotics are needed longer-term, we propose cycling through first- and second-line antibiotic regimens to minimize the development of antibiotic resistance (Fig. 1).

7 Retinoids

The NAHS guidelines recommend the use of acitretin or isotretinoin as second- or third-line therapies in patients with concomitant acne [1]. Studies of retinoids in HS continue to demonstrate mixed results. A recent study of 62 patients with HS who were treated with acitretin demonstrated significant improvement in International HS Severity Score System (IHS4) scores in male ($p = 0.04$) but not female patients [31]. According to expert opinion, acitretin may be superior to isotretinoin [1]; however, comparative studies are still lacking. Oral retinoids may be considered in those with moderate to severe inflammatory acne and prescribed adjunctively with close monitoring of side effects and laboratory abnormalities.

8 Hormonal and Metabolic Therapies

Hormonal therapies, including estrogen-containing combined oral contraceptive pills (OCPs), spironolactone, metformin, and finasteride are recommended as monotherapy by the 2019 NAHS guidelines for females with mild-to-moderate disease or as adjunctive treatment for severe disease in patients with premenstrual flares or polycystic ovarian syndrome (PCOS) [1]. Both metformin and finasteride are options for male patients as they are widely used in other medical conditions, including diabetes and hair loss, respectively.

Limited evidence suggests that the use of progestin-only OCPs may worsen HS, thus combined OCPs are preferred when there are no contraindications [32, 33]. Drospirenone-containing OCPs may have greater antiandrogenic properties; however, these effects have not yet been shown in patients with HS [34]. The authors also recommend considering the drospirenone mini pill for those patients who cannot tolerate estrogen (e.g., they have migraines with aura). Metformin 500 mg to 2 g daily has been shown to improve insulin resistance, C-reactive protein, and cardiovascular disease biomarkers in patients with HS [35].

Recent commentaries and case reports have described potential therapeutic benefit with glucagon-like-peptide 1 (GLP-1) agonists, currently FDA-approved for type 2 diabetes mellitus [36, 37]. One retrospective study revealed

the use of semaglutide in 30 patients with HS reduced the frequency of patient-reported flares and DLQI [38]. While data on the use of GLP-1 agonists for HS are limited, adjunctive implementation may be considered for those with HS and concomitant diabetes or obesity.

Owing to the differing pathoetiologic targets of each hormonal/metabolic treatment, we suggest using a combination of hormonal therapies when appropriate (Fig. 1) [35]. For example, stacking an OCP, spironolactone, and a GLP-1 agonist may provide therapeutic benefit in female patients with HS with comorbid PCOS, obesity, and insulin resistance/diabetes.

9 Biologics and Small Molecule Inhibitors

Initiation of immunomodulators prior to or at the first signs of scar formation and/or tunnel development is essential for mitigating disease progression and further tissue destruction. With the recent FDA approval of bimekizumab in November 2024 and secukinumab in October 2023, adalimumab, bimekizumab, and secukinumab can all be considered first-line biologics for moderate to severe HS. Adalimumab dosing is as follows: 160 mg subcutaneously at week 0, followed by an 80 mg dose at week 2, then 40 mg weekly or 80 mg at week 4 and every 2 weeks thereafter for maintenance. Escalation of adalimumab to 80 mg weekly has demonstrated efficacy in severe and recalcitrant cases or in patients with high body mass index (BMI) (BMI > 30) [39, 40]. Secukinumab is administered 300 mg subcutaneously weekly for 5 weeks, then 300 mg once every 4 weeks thereafter, with potential to escalate to every 2 weeks. Studies have demonstrated efficacy of secukinumab in those who have previously failed adalimumab, with 71.4% of patients reaching HiSCR50 by week 52 [41]. Patients treated with bimekizumab, an IL-17A/F inhibitor, every 2 weeks or every 4 weeks achieved HiSCR50 at week 16 at higher rates compared with placebo in two phase 3 trials ($p = 0.006$, $p = 0.003$, respectively) [42]. Bimekizumab is administered as 320 mg subcutaneously every 2 weeks for the first 16 weeks followed by every 4 weeks thereafter. Infliximab continues to be recommended for moderate-to-severe cases and for those with suboptimal response to adalimumab, bimekizumab, and/or secukinumab. Induction can be administered at weeks 0, 2, and 6. An additional induction dose at week 4 can be added for patients with severe disease [43–45]. However, recent data suggest that patients with HS often require higher and more frequent dosing to achieve clinical response, thus a reasonable maintenance dose of 10 mg/kg every 4–8 weeks has been proposed [35]. In the authors' experience, escalating to a maintenance dose of 10 mg/kg every 4 weeks is often required for severe disease. Biosimilar infliximab-abda has demonstrated similar efficacy to infliximab in a small, single center study [43].

Figure 1 illustrates a framework for escalation and de-escalation of biologic medications as appropriate. If adalimumab, bimekizumab, or secukinumab is found to be inadequate, one can be switched for the other. Alternatively, therapy can be escalated to infliximab. In severe cases, it may be beneficial to start with infliximab (if insurance permits) with plans to de-escalate to adalimumab or secukinumab once disease is controlled. To mitigate the development of antidrug antibodies and ensure treatment endurance, the initiation of infliximab may be accompanied by the use of methotrexate or azathioprine, and antibody and drug levels may be monitored [46].

Immunomodulators with recently reported data or those under current investigation for HS include Janus kinase inhibitors (JAK) inhibitors, ustekinumab, certolizumab, golimumab, brodalumab, and deucravacitinib. Oral JAK inhibitors with promising data include upadacitinib, povorcitinib, and tofacitinib. In phase 2 clinical trials, more patients receiving upadacitinib achieved HiSCR50 than placebo by week 12 (38.3% [$n = 47$] versus 25.0%, $p = 018$) [47]. Povorcitinib, which is not yet commercially available, demonstrated dose-dependent improvements in HiSCR50 [48]. Tofacitinib has demonstrated benefit for HS in case reports [49].

Ustekinumab, an IL-12/23 inhibitor, has shown efficacy at a maintenance dose of 90 mg every 4 weeks in severe patients with HS who have failed adalimumab or infliximab [50]. Brodalumab, an IL-17 inhibitor, has shown positive results in an open label cohort study with 100% ($n = 10$) of patients reaching HiSCR50 by week 12 and has demonstrated efficacy in a retrospective review of eight patients [51, 52]. Certolizumab ($n = 7$) and golimumab ($n = 17$), TNF inhibitors, have demonstrated efficacy in several patients who have failed other biologic and systemic treatments [53, 54]. Deucravacitinib, a TYK2 inhibitor, is currently being studied in phase 2 clinical trials.

Data on use of phosphodiesterase-4 (PDE4) inhibitors, apremilast and roflumilast, are limited. Apremilast has demonstrated clinically meaningful improvement in a small cohort randomized controlled trial, and roflumilast has demonstrated clinical improvement in a case report [55, 56]. Both risankizumab and guselkumab, IL-23 inhibitors, failed to meet their primary end points in phase 2 trials [57, 58]. In both trials, there was no significant difference found between the drug intervention and placebo groups. While not the sole reason for failure, it should be noted that high placebo response rates are a common obstacle in HS clinical trials [59].

10 Systemic Immunosuppressants

The use of systemic immunosuppressants in HS remains a matter of debate. According to the NAHS guidelines, methotrexate or azathioprine have limited evidence to support their use. [1] Recent literature has also shown minimal clinical improvement of HS with the use of azathioprine [60]. Although methotrexate may reduce number of abscesses, it has not been found to reduce number of fistulas in severe patients not on biologics [61]. While methotrexate or azathioprine may not be appropriate monotherapy agents, they have demonstrated a role in reducing immunogenicity to TNF inhibitors in chronic inflammatory diseases [46]. Thus it may be beneficial to initiate methotrexate or azathioprine in conjunction with TNF inhibitors to prevent development of antidrug antibodies. Cyclosporine may be beneficial for patients with recalcitrant moderate-to-severe HS who have failed or are not candidates for other systemic therapies [1].

11 Surgical Modalities

Deroofings and excisions remain the backbone of HS surgery. Surgical intervention should be considered for persistent or recalcitrant HS lesions despite medical optimization. Partial or complete deroofings are carried out by removing a portion or the majority of the skin overlying an HS tunnel, followed by debridement of the cavity; the base is left intact [62]. Partial lesional deroofing may be beneficial in cases where full debridement of the cavity is unachievable in an outpatient setting. Infection risk is low, and overall patient satisfaction seems to be high with deroofing procedures [63]. Full thickness excisions of the epidermis, dermis, and deeper HS-affected tissue can be done for individual lesions or regionally over an entire anatomical area [62]. In a meta-analysis, wide excision, local incision, and deroofing resulted in 13, 22, and 27% HS recurrence rates, respectively [64]. Local incision and drainage procedures are only performed for symptomatic relief, thus having a high recurrence rate of almost 100% [1]. Healing by secondary intention may be associated with lower recurrence rates and faster return to physical activities than extensive reconstructions, but patient preference for sutured wounds or clinical scenarios with extensive disease may benefit from reconstructive techniques [65]. According to the Safety and Efficacy of Adalimumab for Hidradenitis Suppurativa Peri-Surgically (SHARPS) study, biologic use should not be interrupted for HS procedures and continuation through preoperative, operative, and postoperative periods is recommended [66]. While this study observed surgical outcomes only in patients treated with adalimumab versus placebo, our expert opinion favors continuing all appropriate HS-directed biologics through surgical intervention. Additional recommendations for perioperative management include optimizing medical treatment prior to surgery, encouraging smoking cessation to improve wound healing, providing intralesional steroid injections to flared lesions prior to surgery if indicated, and continuing all appropriate HS treatments to prevent flares in the postoperative periods [67–69]. Proactive curation of a postsurgical flare treatment plan may also benefit patients.

12 Pain Management

HS disease control is the foundation of pain management. Early and consistent assessment and appropriate management of pain are essential in bridging the gap to disease control. Pharmacologic pain management modalities include over-the-counter pain relievers such as acetaminophen and nonsteroidal antiinflammatory drugs (NSAIDs), topical anesthetics, neuromodulator agents, and opioids. Savage et al. introduced a pain management algorithm to help provide clinicians guidance on treating various types of pain (acute, chronic, nociceptive, and neuropathic). Acute pain episodes can be treated with acetaminophen, NSAIDs such as ibuprofen, topical anesthetics, and tramadol. If additional pain control is needed, opioids can be prescribed for severe flare-related pain (e.g., prescribing 20 pills for a severe flare episode) [70]. Agents best suited for treatment of chronic nociceptive pain include NSAIDs, duloxetine, and nortriptyline. Gabapentin, duloxetine, pregabalin, venlafaxine, and nortriptyline may be helpful for chronic neuropathic pain. Referral to a pain specialist is recommended for patients who fail at least two pharmacologic therapies, have debilitating pain despite medical optimization, or for those using chronic opioids.

13 Lifestyle Modifications and Adjunctive Treatment

Lifestyle modifications remain an important aspect of managing HS. Clinicians should continue to screen for obesity, counsel weight loss when appropriate, and recommend smoking cessation to improve overall health. Expert recommendation is to wait until rapport is established with patients before addressing these sensitive topics.

At the time the 2019 NAHS guidelines were published, there was inadequate evidence to recommend weight loss, avoidance of brewer's yeast (*Saccharomyces cerevisiae*), and zinc supplementation [1]. Newer evidence has since emerged supporting potential benefit from these modifications. In a single institution retrospective study, patients who underwent bariatric surgery had lower mean weight and decreased number of anatomical sites affected by HS ($p < 0.001$) and

HS activity ($p < 0.001$) postoperatively when compared with controls [71]. A survey study suggested implementation of a brewer's yeast exclusion diet in a cohort of 37 patients resulted in improvement in HS symptomology in 70% of the patients [72]. In a controlled retrospective study, patients with mild-to-moderate HS treated with zinc gluconate 90 mg and nicotinamide 30 mg once daily for 90 days reported a marked reduction in mean HS visual analogue scale (VAS), DLQI, and International HS Severity Score System (IHS4) scores compared with the control group at 12 and 24 weeks ($p < 0.005$) [73]. The uncontrolled, retrospective nature of the data from these interventions is a significant limitation to the reliability. Given the low risk of these adjunctive interventions, it is reasonable to recommend them for patients interested in their use in combination with prescription medications [74].

14 Light-Based and Energy-Based Treatments

The neodymium-doped yttrium aluminum garnet (Nd:YAG) and carbon dioxide (CO_2) lasers have the most evidence supporting their use and are recommended in patients with Hurley stage II or III disease [6]. Nd:YAG, intense pulsed light, and long-pulsed alexandrite and diode lasers can be used to destruct follicles in HS affected areas [75–77]. A randomized, controlled trial demonstrated that 3 months of ND:Yag laser hair reduction in HS lesion sites improved severity by an average of 65.3% across various anatomic sites on the basis of a modified HS lesion, area, and severity index [76]. For patients with predominantly follicular-type HS or areas lacking extensive scarring, laser hair reduction is recommended by experts as it removes the source of follicular occlusion and, thus, may reduce disease flares and progression. Recent literature suggests that the combination use of intense pulsed light and nonablative radiotherapy may be effective for patients with Hurley stage I and II but less so for stage III disease [78–80]. An intense pulsed light/nonablative radiotherapy combination performed synchronously with topical clindamycin demonstrated improvement in HS severity on the basis of IHS4, HiSCR50, DLQI, and pain scales in a randomized controlled trial [78]. Use of the alexandrite laser with concomitantly antiseptic chlorhexidine wash and oral zinc gluconate 90 mg daily for 30 weeks was found to reduce pain, HS VAS ($p = 0.002$), DLQI ($p = 0.002$), and flares ($p = 0.001$) [81]. With proper selection of device and application, light and energy-based treatments can be used to treat all Hurley stages in both lighter- and darker-skinned individuals.

15 Botulinum Toxin

Both botulinum toxin (BTX) A and BTX-B have been reported for the use of HS treatment, by reducing sweat production, which may decrease the proinflammatory effect of bacteria on the skin surface [82, 83]. In a randomized controlled trial, 20 patients with HS received either one treatment of intradermal, perilesional BTX-B, or a placebo. Patients receiving BTX-B had significantly greater improvement in DLQI scores at the 3-month follow-up compared with placebo patients ($p < 0.05$). All patients, including placebo patients, then received a BTX-B treatment. At the 6-month follow-up, patients in both groups had significant reduction in number of lesions ($p < 0.05$) [83]. There are two ongoing clinical trials studying BTX for HS (NCT05403710 and NCT06237465). Thus, local BTX injection may be beneficial for patients with HS with and without hyperhidrosis.

16 Wound Care

Wound care for both primary lesions and surgical wounds in HS is based on limited evidence and must be individualized to each patient. Anatomic location of wounds, amount of drainage, and cost of supplies should all be taken into consideration [6]. According to the NAHS guidelines, negative-pressure therapy for short periods of 1–4 weeks may be beneficial for large open postsurgical wounds [1]. Expert opinion suggests that use of antiseptic washes is recommended.

Specialized adhesive-free HS-specific wound care systems may be helpful and have been shown to reduce dressing-related pain and improve DLQI scores and patient usability [84]. Absorptive and atraumatic dressings are preferred to minimize trauma to the wound and peri-wound tissue but may be difficult to access for many patients [85]. Wound care dressing absorbency should be tailored to the amount of drainage as needed. The recent Delphi consensus on routine and surgical wound care determined that no single cleanser or wound dressing type is superior to others, reaffirming the need to tailor wound care dressings and reconstructive techniques to individual patient preferences and affordability [86]. Insurance coverage for wound care dressings may be available with a prescription, potentially reducing the patient's out-of-pocket expenses.

17 Flare Therapy

Flare treatment plans are an important aspect of the HS therapy toolbox that should be implemented for all patients with HS. It is important that patients with HS are equipped with

tools to help manage their flare symptoms. Interventions that can be implemented at home include warm compresses, sitz baths, over-the-counter analgesics (ibuprofen, etc.), and application of topical medications such as clindamycin 1% or resorcinol 10%. Initiation or escalation of oral antibiotics may be needed for more persistent flares. Intralesional triamcinolone at doses of 10–40 mg/ml can also be injected in the clinic setting for inflamed nodules and tunnels [87]. For expanding or painful abscesses, incision and drainage can provide great relief. Incision with a 6 mm punch tool after achieving adequate anesthesia has been proposed as an alternative to puncturing with an 11-blade [88]. The use of the punch tool allows for control over depth of incision, reduces chance of premature wound closure, and eliminates the need for postprocedure wound packing, which may delay healing and cause significant discomfort when being removed. In severe HS flares with multiple areas of involvement, short courses of systemic steroids may be beneficial. A short steroid taper may be appropriate if the patient is experiencing an acute flare while on stable medical management, including biologics or immunosuppressants. A short taper should start at 1 mg/kg ideal body weight and taper by 10 mg every 1–2 days. A long taper may be appropriate for a flare in the setting of high baseline disease activity while the patient is being bridged to the initiation of a biologic. A long taper starts at 1 mg/kg ideal body weight and tapers by 10 mg weekly.

18 Special Populations: Pediatrics

The onset of HS was historically thought to occur in a patient's early to mid-20s. New evidence suggests a bimodal distribution of onset, with one peak in the late teens and the other in the mid-40s [89]. The NAHS guidelines recommend screening for precocious puberty in patients with HS presenting at 11 years of age or younger. A recent case-control study demonstrated precocious puberty was associated with higher odds of HS [90]. Diagnosis and early treatment of HS in the pediatric population is essential to minimize scarring and negative impact of disease on quality of life. Recent reviews of medical and procedural therapies for pediatric HS have been published, including a figure summary of recommended treatments in Cotton et al. [91–93].

19 Limitations

Limitations of this study include the narrative review methodology, which may have resulted in the unintentional exclusion of conflicting or new publications since the time of this manuscript.

20 Conclusions

Treatment of HS is often complex and commonly requires the use of multiple medical and surgical modalities to optimize disease control. Herein, we presented a review of both established HS treatment modalities according to the NAHS guidelines and those on the horizon. This update on recent literature alongside our proposed algorithm aims to guide clinicians in their implementation of treatment stacking in HS. It is imperative that clinicians are empowered to create individualized, multimodal HS treatment plans that involve shared decision-making to optimize patient care.

Declarations

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Conflict of interest V.Y.S. is on the board of directors for the Hidradenitis Suppurativa Foundation (HSF); is an advisor for the National Eczema Association; and is a stock shareholder of Learn Health and has served as an advisory board member, investigator, speaker, and/or received research funding from Sanofi Genzyme, Regeneron, AbbVie, Genentech, Eli Lilly, Novartis, SUN Pharma, LEO Pharma, Pfizer, Incyte, Dermavant, Boehringer Ingelheim, Almirall, Alumis, Aristea Therapeutics, Menlo Therapeutics, Dermira, Burt's Bees, Galderma, Kiniksa, UCB, Ceraclere, Bain Capital, Target-PharmaSolutions, Castle Bioscience, Altus Lab/cQuell, MYOR, Polyfins Technology, Gp-Skin, and Skin Actives Scientific. J.L.H. is on the board of directors for the Hidradenitis Suppurativa Foundation and has served as an advisor for AbbVie, Aclaris, Boehringer Ingelheim, Incyte, Novartis, Sanofi, UCB; as a speaker for AbbVie, Galderma, Novartis, Sanofi Regeneron, and UCB; and as an investigator for Amgen, Boehringer Ingelheim, and Incyte. C.J.S. reports being an investigator for AbbVie, ChemoCentryx, Incyte, InflaRx, Novartis, Astrazeneca, and UCB Pharma; received consultancy fees from AbbVie, Sanofi, Alumis, Astrazeneca, InflaRx, Sandoz, Incyte, Logical Images, Sonoma Biotherapeutics, and UCB Pharma; and is a speaker for AbbVie and Novartis. Authors K.H.L. and C.B.D. have no disclosures.

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