Hidradenitis Suppurativa for the Dermatologic Hospitalist

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Hidradenitis suppurativa (HS) is a common chronic inflammatory skin disease characterized by purulent subcutaneous nodules, papules, abscesses, and fistula tracts that lead to scarring and fibrosis. Lesions develop primarily in the axilla, groin, and other intertriginous and hair-bearing areas.

The natural history of the disease is characterized by periods of disease flare, followed by periods of disease quiescence. Patients might have weeks or months of low disease activity but frequently develop multiple exacerbating episodes over the course of weeks or months. The condition primarily presents in adolescent and peripubescent years, continuing throughout adulthood. Some evidence suggests a bimodal disease distribution, with a second peak of incidence in middle-aged adults. Women and men are affected equally; however, the disease can be phenotypically different in men and women.

Patients frequently present in emergency and inpatient settings for evaluation because of the pain and severity of HS flares as well as associated systemic symptoms. Inpatient and emergency department (ED) care are unique opportunities for dermatologic hospitalist and dermatologic consultative services to educate other physicians about the condition and initiate aggressive treatments that are frequently necessary to control HS flares. This article aims to address best methods for treating HS in these settings.

Pathophysiology

Although the exact pathophysiology of the condition is unknown, HS is thought to begin with follicular occlusion with downstream inflammation mediating neutrophilic activity and scarring. Hyperplasia of the infundibular epithelium is observed on histology, and the resulting
occlusion, contained keratin, and follicular rupture initiate robust downstream inflammation.\textsuperscript{1,2} Follicular occlusion might be initially androgen mediated\textsuperscript{3} or might occur in combination with friction\textsuperscript{4} and genetic or acquired factors involving Notch signaling. Although HS characteristically presents in areas of high apocrine density, apocrine glands are not thought to be the primary mediator of disease activity.\textsuperscript{5} IL-17, IL-23, tumor necrosis factor $\alpha$, and IL-1$\beta$ are implicated in the pathogenesis of HS, but it is unknown if these cytokines are the driving pathologic factor in HS or if they are merely secondary sequelae.\textsuperscript{6}

**Demographics and Prevalence in Hospitalized Patients**

Although increasing treatment availability has brought more attention to HS, true prevalence is unknown. A prevalence of 1% has been reported in many European countries.\textsuperscript{7} Global prevalence has been more difficult to determine, with variable data suggesting a prevalence of 0.03% to 8%, depending on the population included.\textsuperscript{8} Most patients studied in a US-based claims database were aged 30 to 64 years, and the overall prevalence was 0.05%.\textsuperscript{9} Despite prevalence similar to psoriasis, utilization of high-cost emergency and inpatient admissions is notably higher among patients with HS. Recent claims data suggest that HS patients utilize the ED at a rate 3 times higher than psoriasis patients and are admitted as inpatients at a rate 5 times higher.\textsuperscript{10} Similar data suggest an associated increased cost of care for patients with HS vs other conditions, such as psoriasis, due to frequent ED and inpatient stays.\textsuperscript{11} Although HS frequently presents in the inpatient and emergency settings, there is little literature on best methods for managing patients in these settings.

**Pearls for Inpatient and Emergency Evaluation and Management**

**Initial Evaluation**—When dermatologic consultative services are asked to evaluate patients with HS, preliminary evaluation should reflect the acuity of the patient. Vital signs and toxicity should be reviewed to ensure that there is no evidence of severe infection necessitating critical or acute care.

**History**—History-taking should reflect assessment of the patient’s baseline disease, including date of initial onset; exacerbating factors, such as friction, smoking, pregnancy, and menses; and the current history of the patient’s flare. A history of antibiotics, immunosuppression, topical therapy, antiandrogen therapy, and vitamin A analog therapy also should be reviewed. If an initial diagnosis is made in the ED or inpatient setting, a family and personal history should focus on specific risk factors and disease associations, including inflammatory bowel disease,\textsuperscript{12} pilonidal cysts,\textsuperscript{13} polycystic ovary syndrome,\textsuperscript{14} and metabolic syndrome.\textsuperscript{15}

**Physical Examination**—As with all dermatologic consultations, a full-body skin examination, with special attention to the axilla, inframammary skin, groin, buttocks, and perineum, should be undertaken. In addition to these common areas of disease progression, examination should focus on atypical sites for disease manifestation, including the posterior auricular scalp, skin folds in the pannus and back, and the beard area in men. Evaluation of axillary and gluteal hair should note features of folliculitis and hair removal, which can exacerbate HS. Examination also should include an investigation of cutaneous manifestations of comorbid conditions, including acanthosis nigricans, contiguous or metastatic cutaneous Crohn disease, erythema nodosum, and pilonidal cysts. Caution should be exercised when diagnosing pilonidal cysts, as isolated or evolving HS in the gluteal cleft often is misdiagnosed as a pilonidal cyst.

**Laboratory Evaluation**—Testing often is misleading in patients with HS, especially in the acute setting, because the condition is a chronic inflammatory process. The C-reactive protein level as well as the absolute white blood cell and neutrophil counts often are elevated, even in the absence of acute infection.\textsuperscript{16} In fact, although patients often are treated with intravenous antibiotics by inpatient and emergency teams in the setting of these 3 laboratory abnormalities, these findings often reflect disease activity, not frank infection. Fever, especially low-grade fever, also can reflect ongoing disease activity. Thrombocytosis and anemia also are anecdotally common, though these findings have not been reported specifically in the literature.

**Bacterial Cultures**—The role of lesional and perilesional bacterial cultures is controversial in HS. Prior studies have demonstrated that biofilm formation may be associated with the chronic inflammation seen in HS.\textsuperscript{17} However, most data to date suggest that infection is not the primary driver of HS disease flares, as demonstrated by the frequency of sterile cultures and the variable response of the disease to penicillin and related antibiotics.\textsuperscript{18}

**Imaging**—Ultrasonography and magnetic resonance imaging can be conducted if there is concern about deeper abscesses that are not apparent on examination. When interpreted by nondermatologic practitioners, however, the findings of these modalities can result in unnecessary surgical intervention, given the concern for development of infectious abscess.\textsuperscript{19}

**Diagnosis**—Many patients with HS experience a notable delay in time to diagnosis, living with symptoms for 7 years on average prior to being given a name for their condition.\textsuperscript{20} Often, patients seek ED care at initial presentation because lesions can present quickly and are associated with remarkable pain. Inpatient dermatologic evaluation can provide patients with definitive diagnosis, appropriate counseling that provides an overview of the natural history of the disease, lifestyle recommendations, and expedited connection to outpatient longitudinal care.

Diagnosis is made clinically by assessment for typical lesions, such as painful or tender papules, nodules,
## Treatment Considerations for Hidradenitis Suppurativa Based on Severity

<table>
<thead>
<tr>
<th>Disease Severity Acutely</th>
<th>Acute Care</th>
<th>Longitudinal Treatment Modalities Initiated During Inpatient Stay</th>
<th>Ancillary Care</th>
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</thead>
<tbody>
<tr>
<td>Scarring without active lesions</td>
<td>Counseling, including natural history of disease; lifestyle modifications; recommendations for management of acute flares</td>
<td>Expedited outpatient follow-up; class I topical steroid ointment for use during flares; consideration of low-dose spironolactone&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Referral to primary care for management of relevant comorbidities and for smoking cessation; psychiatric and psychological services (when relevant)</td>
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<tr>
<td>Hurley stage I</td>
<td>Counseling, including natural history of disease; lifestyle modifications; recommendations for management of acute flares; oral amoxicillin–clavulanic acid for 7 d; doxycycline or clindamycin if amoxicillin–clavulanic acid is contraindicated</td>
<td>Expedited outpatient follow-up; class I topical steroid ointment for use during flares; low-dose spironolactone&lt;sup&gt;+&lt;/sup&gt;; amoxicillin or tetracycline for 3–6 mo; consider acitretin for patients with comedonal disease</td>
<td>Referral to primary care for management of relevant comorbidities and for smoking cessation; psychiatric and psychological services (when relevant)</td>
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<tr>
<td>Hurley stage II</td>
<td>Counseling, including natural history of disease; lifestyle modifications; prednisone taper beginning at 0.5–0.75 mg/kg for 7–10 d, reserving longer tapers for severe disease or when long-term anti-inflammatory therapy is not feasible; oral amoxicillin–clavulanic acid for 7 d; doxycycline or clindamycin if amoxicillin–clavulanic acid is contraindicated; laboratory screening before initiating biologic therapy</td>
<td>Expedited outpatient follow-up; low-dose spironolactone&lt;sup&gt;+&lt;/sup&gt; or full-dose tetracycline; adalimumab; consider additional treatment, as listed under Hurley stage I, pending response to adalimumab</td>
<td>Referral to primary care for management of relevant comorbidities and for smoking cessation; psychiatric and psychological services (when relevant); surgical evaluation when disease is localized; laboratory evaluation, including CBC, for evaluation of anemia and thrombocytosis</td>
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<tr>
<td>Hurley stage III</td>
<td>Counseling, including natural history of disease; lifestyle modifications; prednisone taper beginning at 0.5–0.75 mg/kg for 7–10 d, reserving longer tapers for severe disease or when long-term anti-inflammatory therapy is not feasible; IV antibiotic with anaerobic coverage; laboratory screening before initiating biologic therapy; consider infliximab infusion, 5 mg/kg, for severe disease if adalimumab fails; cyclosporine for patients in whom prednisone is contraindicated or those who have already received multiple prednisone tapers</td>
<td>Expedited outpatient follow-up; low-dose spironolactone&lt;sup&gt;+&lt;/sup&gt; or tetracycline therapy; adalimumab; infliximab infusion, 5 mg/kg, for severe disease if adalimumab fails; consider cyclosporine for patients in whom prednisone is contraindicated or those who have already received multiple prednisone tapers</td>
<td>Referral to primary care for management of relevant comorbidities and for smoking cessation; psychiatric and psychological services (when relevant); surgical evaluation when disease is localized; imaging (magnetic resonance imaging or ultrasonography) when there is concern for progressive abscesses or masses; laboratory evaluation, including CBC, for evaluation of anemia and thrombocytosis; colonoscopy when there is evidence of cutaneous Crohn disease or pyodermalike lesions</td>
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Abbreviations: CBC, complete blood cell count; IV, intravenous.

<sup>a</sup>For female patients.
or abscesses in the axillae, inframammary region, groin, thighs, and perineal and perianal regions. Cordlike scarring often is seen in the absence of active inflammatory lesions.21 Double-headed open comedones and prominent follicular occlusion are seen in some phenotypes but are not required for diagnosis.22

Multiple scoring modalities are in use23; the Hurley staging system, initially developed for surgical staging, has become a commonly used method in the clinical setting24:

- Hurley stage I: isolated nodules or abscesses;
- Hurley stage II: widely separate lesions and sinus tracts or scarring are suggestive; and
- Hurley stage III: multiple lesions with near-diffuse involvement and formation of sinus tracts and scarring.

Other scoring modalities, such as the Hidradenitis Suppurativa Clinical Response (HiSCR), are more commonly used in the clinical trial setting and quantitatively capture lesion count improvement while the patient is being treated.25

Treatment—Evaluation in the ED might necessitate recommendations for inpatient admission. Dermatologic consultation can be helpful in providing ED physicians with context for interpretation of laboratory results and clinical findings. Specifically, dermatologic evaluation can help differentiate presentations consistent with a primary infection from a more common presentation of HS flaring and associated bacterial colonization. Indications for inpatient admission are severe pain; concern for systemic infection, including high fever or sepsis; and need for surgical intervention. Patients with severe disease who do not have a longitudinal care plan or who lack the ability to care for lesions at home also are candidates for inpatient admission, where they can receive more intensive nursing and wound care as well as outpatient logistical management.

Acute care should be aimed at treatments that work quickly and aggressively and have both anti-inflammatory and antimicrobial effects. Severe flares require aggressive initial treatment to ensure more long-term remission. Adalimumab, maintained at 40 mg/wk after a loading dose, is the mainstay of evidence-based treatment for moderate to severe HS in patients 12 years or older; however, this treatment might not be easy to initiate in the inpatient setting because of its cost and availability and the fact that it is not as fast acting as other therapies.26 For patients with severe disease flares, prednisone,27 infliximab,28 or cyclosporine29 can be used in combination with antimicrobial therapy in the inpatient setting to quickly control active flaring. Intravenous antimicrobial therapy might be necessary in severe disease and should include coverage of gram-positive30 and anaerobic organisms.31

Although management of acute flares is critical, especially for hospitalized patients, initiating longitudinal treatment modalities while the patient is an inpatient will help prevent future readmissions, facilitate better outcomes, and enable longer periods of disease-free progression. Specific treatments, stratified by disease severity, are listed in the Table.

Postdischarge Lifestyle Modification—All disease management should include recommendations for lifestyle modification, including counseling on terminal hair removal (ie, avoid shaving, plucking, and waxing) and recommendations for daily and weekly decolonization with chlorhexidine or other antimicrobial soap, a weekly vinegar bath, and antiperspirant use in the groin and axilla. Avoiding tight clothes and humidity might also be helpful.

Other beneficial postdischarge strategies include smoking cessation and weight loss, which often are beneficial but difficult for many patients to achieve on their own; connecting patients with a primary care provider, which can facilitate better long-term outcomes; informing patients of the natural history of the disease and providing strategies for them to implement for acute flares to help avoid readmission and ED visits; and writing a “pill-in-pocket” prescription for a course of an antibiotic that provides good staphylococcal and anaerobic coverage, which can be helpful for patients who are prone to infrequent flares.

Lastly, appropriate postdischarge maintenance therapy also can be initiated during the inpatient stay, including maintenance antibiotic therapy, spironolactone32 for female patients, and acitretin33 for comedonal-predominant patients.

Final Thoughts

Hidradenitis suppurativa is a common dermatologic condition that frequently presents in emergency and inpatient settings, given its association with painful and acutely indurated lesions that often appear concerning for infection. Elevated inflammatory markers and fever are common in HS and are not necessarily suggestive of infection. As such, while antibiotics may be part of acute management of HS, care also should address the inflammatory component of the disease. Longitudinal outpatient care coordination with a dermatologist and primary care physician is imperative for limiting ED and inpatient care utilization.

REFERENCES