Hidradenitis Suppurativa: Update on Diagnosis and Treatment

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Post-Test and Evaluation Form

Faculty

Francisco A. Kerdel, BSc, MBBS
Director of Inpatient Dermatology
University of Miami Hospital
Professor of Dermatology and Vice Chairman
Florida International University
Miami, Florida

Alan Menter, MD
Chief, Division of Dermatology
Baylor University Medical Center
Clinical Professor of Dermatology
University of Texas, Southwestern Medical School
Director, Fellowship Program
Baylor Psoriasis Center
Chair, Psoriasis Guidelines Committee,
American Academy of Dermatology
Dallas, Texas

Robert G. Micheletti, MD
Assistant Professor of Dermatology and Medicine
Perelman School of Medicine at the University
of Pennsylvania
Philadelphia, Pennsylvania

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Hidradenitis Suppurativa: Update on Diagnosis and Treatment

Learning Objectives

• Describe the range of clinical presentations in patients with hidradenitis suppurativa and list the other clinical entities that should be considered in the differential diagnosis.

• Institute an improved approach to recognizing and establishing the definitive diagnosis in patients with hidradenitis suppurativa.

• Name and explain the significance of psychosocial and medical comorbidities and complications associated with hidradenitis suppurativa.

• Discuss the evidence-based approaches to treatment of hidradenitis suppurativa now available and explain the rationale for and appropriate use of newer strategies, including the use of biologic agents.

• Develop and implement improved strategies for patient education, with the goal of teaching patients to better manage the aspects of their disease that are within their control.

• Establish or improve an existing strategy for appropriate referral of patients with chronic hidradenitis suppurativa for surgical management.

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This educational activity is designed for dermatologists, emergency medicine specialists, primary care physicians, and other clinicians who diagnose and treat patients with hidradenitis suppurativa.

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Educational Needs

Hidradenitis suppurativa, a disease of the hair follicles that involves follicular occlusion and hyperkeratosis and subsequent inflammatory responses, profoundly affects patients’ quality of life. Pain is a common feature and requires effective and safe management. In severe, recurrent, and/or recalcitrant cases, depression and other psychosocial effects frequently are seen. Medical complications include anal, urethral, and rectal strictures and fistulas; contractures and limitations of limb mobility; cutaneous squamous cell carcinoma and an increased risk for other malignancies; kidney disease; and metabolic syndrome.

Specialists in dermatology are the most likely to see patients with hidradenitis suppurativa, as are women’s health practitioners (not surprisingly, because the disease seems to affect women far more frequently than men in the United States). Patients with severe cases of hidradenitis suppurativa often seek relief in hospital emergency departments, so it is important for physicians and other practitioners in emergency medicine to be able to readily recognize the manifestations of this disease, institute palliative therapy immediately, and refer patients to the appropriate specialists for prompt attention and follow-up care.

At present, patients who are diagnosed with hidradenitis suppurativa are treated according to disease features such as severity, duration, and morphology. Medical treatment options for mild or moderate cases include a variety of topical and systemic antibiotics, used alone or in combination, as well as intralesional injections of triamcinolone for early lesions. Other treatments used include oral dapsone, zinc, acitretin, hormonal therapy (eg, as well as intralesional injections of triamcinolone for early lesions. Other treatments used include oral dapsone, zinc, acitretin, hormonal therapy (eg, 4. No relevant financial relationships.

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Introduction

Hidradenitis suppurativa, also called acne inversa, is a chronic, debilitating skin disease characterized by painful, deep inflammatory lesions. These lesions, once believed to represent a disease of the sweat glands, now are thought to arise in the terminal hair follicles in the apocrine gland–bearing areas of the body. The regions typically affected are the intertriginous areas—the axillae, groin (genital, perigenital, anal, and perianal areas), infra- and intermammary skin, buttocks, and upper thighs, although other areas—such as the nape of the neck and scalp—can be involved.

Among all the chronic inflammatory cutaneous disorders, severe hidradenitis suppurativa is at or near the top of the list of those that adversely affect quality of life. Patients with the severe form of the disease live with multiple painful nodules and draining sinuses that exude what is often foul-smelling fluid, along with the sense of dread that new lesions may erupt at any time, requiring incision and drainage, corticosteroid injection, or surgical intervention. Patients also face embarrassment because the odor and drainage may pose real physical limitations related to pain or limb contracture in the setting of severe scarring and fibrosis. For these reasons, dealing with the physical and psychological burdens of this disease can be extraordinarily difficult.

The list of unmet clinical needs covers several broad categories: earlier identification and diagnosis, therapeutic options, and patient and family education and support. It is hoped that improved patient and public education about this disease will lead to more patients seeking early medical intervention. Better communication with and provision of updated education to clinicians outside the specialty of dermatology also is needed, including efforts to reach (1) family and internal medicine specialists and others in primary care, who are in an excellent position to identify this disease in its earliest stages, and (2) emergency medicine specialists, the most likely practitioners outside of dermatology to see the most severely affected patients.

The articles in this supplement discuss these unmet needs in more detail and provide an overview of current information and views based on the available evidence as well as the authors’ clinical experiences with patients with hidradenitis suppurativa.

An educational handout that may be useful to your patients is available on page S60. The handout may be freely copied by clinicians and distributed to your patients. The handout will also be found online at www.globalacademycme.com/dermatology in the Skin & Allergy News CME Library listing for this supplement: Hidradenitis Suppurativa: Update on Diagnosis and Treatment.

Francisco A. Kerdel, BSc, MBBS*
Alan Menter, MD†
Robert G. Micheletti, MD‡

*Director of Inpatient Dermatology, University of Miami Hospital; Professor of Dermatology and Vice Chairman, Florida International University, Miami, FL.
†Chief, Division of Dermatology, Baylor University Medical Center; Clinical Professor of Dermatology, University of Texas, Southwestern Medical School; Director, Fellowship Program, Baylor Psoriasis Center; Chair, Psoriasis Guidelines Committee, American Academy of Dermatology; Director, Dermatology Residency Program, Baylor University Medical Center, Dallas, TX.
‡Assistant Professor of Dermatology and Medicine, Perelman School of Medicine at the University of Pennsylvania, Philadelphia, PA.

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Address reprint requests to: Francisco A. Kerdel, BSc, MBBS, Florida Academic Dermatology Centers, 1400 NW 12 Avenue, Suite 4, Miami, FL 33136; dr.kerdel@fadcenter.com
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Hidradenitis Suppurativa: Current Views on Epidemiology, Pathogenesis, and Pathophysiology

Robert G. Micheletti, MD*

Abstract
Hidradenitis suppurativa (HS) is a disease characterized by chronic follicular occlusion and secondary inflammation of the apocrine glands. The disease is uncommon but not rare. Further epidemiologic study is required to better determine HS prevalence in various populations. Women are affected three times more often than men, although HS tends to be more severe in men. The typical age of onset is in the second and third decades; HS is rare prior to puberty, and the prevalence is lower in older adults. A number of factors (particularly cigarette smoking) and comorbid conditions (especially obesity) are associated with HS, but causative relationships have not been established. Epidemiology and Demographics

The prevalence of HS has been estimated to be as low as 0.00033% and as high as 4%. More recent studies have provided a clearer picture of those affected (Table).1

Epidemiology and Demographics
The prevalence of HS has been estimated to be as low as 0.00033%2 and as high as 4%.3 More recent studies have estimated the risk to be 1%4 or less.5 This variation may be explained by the fact that substantial differences in prevalence exist among various subpopulations, which may be under- or overrepresented in particular studies. For example, a large Danish study estimated the point prevalence of HS from a sample of 507 consecutive patients undergoing screening for sexually transmitted diseases.6 The relatively high prevalence of HS in this group (4.1%) may well be explained by the comparatively young age of those included. Attempts to estimate the prevalence of HS from claims databases and electronic health records may be limited by the potential for misdiagnosis and inaccurate coding among the various medical specialties that interact with and care for patients with HS.

Thus, the potential is high for methodologic problems in studying the epidemiology of HS. The pathophysiology of the disease is still not well characterized, so confounding variables likely exist that are not identified and controlled for. Nevertheless, there is widespread agreement, based on both available epidemiologic data and the preponderance of experience among clinicians, that HS most commonly occurs in individuals in the second and third decades of life and is seen more frequently in women than in men. Differences among ethnic populations are less clear.

Age
The disease is rare prior to puberty, and the prevalence is lower among older adults. In a study of 302 French patients with HS, the median age of disease onset was 20 years (19 years in men, 21 years in women), and the median age of evaluation for the study was 30.4 years (30.2 years in men and 33.5 years in women).7

Sex
The female-to-male ratio of patients with HS is approximately 3:1,5,7 and the highest incidence has been reported to be among women 20 to 29 years of age.1 Interestingly, the presentation of HS also appears to differ by sex in both anatomic distribution and severity, with more severe disease and more perianal and atypical disease locations seen in men. (A more detailed discussion of presentation is available in the article “Natural History, Presentation, and Diagnosis of Hidradenitis Suppurativa” on page 8 of this supplement8)

ethnicity
Some have suggested that the disease is more common and/or more severe in patients of African ancestry than in those of European descent. However, the available data do not support this notion.9

Underlying Pathogenesis in HS
Arguments supporting a number of possible pathogenetic mechanisms in HS have been proposed; behavioral, genetic, infectious, hormonal, and/or host defense factors may be involved.

Behavioral “Risk Factors” for HS
A number of factors have been associated with HS, including the most frequently cited—smoking, obesity, and mechanical and environmental factors. However, a causative relationship...
has not been firmly established for these associations. Although the term “risk factors” is commonly used, further research is needed to characterize these relationships.

Smoking
A number of studies have revealed that a high percentage of patients with HS are cigarette smokers. Some studies have shown a prevalence of cigarette smoking as high as 70% to 88.9% among patients with HS, findings that are consistent across various populations. In a multivariate analysis, Revuz et al reported the odds ratio of self-reported smoking among patients with HS to be 4.16. The quantity of cigarettes smoked did not appear to be relevant. Meanwhile, smoking cessation was not shown to improve the disease.

Obesity
Body weight has a strong positive correlation with HS. As many as 50% of those with HS are obese. The odds ratio for HS is 1.12 for each increase of one unit of body mass index. The proposed mechanisms underlying this association include follicular occlusion and trauma/friction.

Mechanical and Environmental Factors
Clinical experience and a variety of studies have implicated a number of mechanical and environmental insults that seem to have an effect on HS. Among the factors cited in this category are skin-on-skin friction in intertriginous areas (especially in obese patients), irritation from the use of antiperspirants, and shaving of axillary hair, activities that may traumatize hair follicles. Whether these effects play a role in causation or simply exacerbate the disease has not been established, and there are no data that prove the associations. However, patient reports and clinical observation suggest that all of these factors may at least worsen the symptoms of patients with HS.

Genetic Factors
Evidence exists for an autosomal dominant relationship in at least some patients with HS, possibly indicating that HS has a heritable form. In one study, 34% of first-degree relatives of patients with HS also had the disease, demonstrating autosomal dominance with variable penetrance. A potential genetic locus for the disease has also been identified. Subsequently, advances in genetic research have shown that the enzyme γ-secretase is important in skin biology and has a role in a small number of patients with HS, in whom mutations in γ-secretase genes (NCSTN, PSENEN, and PSEN1) have been identified. Further study is needed to enhance the understanding of the genetics of HS.

Infectious Factors
Cultures of samples taken from ruptured or open HS lesions commonly demonstrate a variety of bacterial species, including *Streptococcus viridians, Staphylococcus aureus, Staphylococcus epidermidis, Peptostreptococcus* and *Bacteroides* species, coryneform bacteria, and Gram-negative bacteria, including *Escherichia coli* and *Klebsiella* and *Proteus* species. Conversely, cultures from early, unruptured lesions may be sterile. To date, none of the organisms recovered from either superficial or deep sampling has predominated. Although some theorize that staphylococci or other bacteria may play an important role in HS, either by creating an occlusive biofilm or by promoting an inflammatory response, it is not possible at this time to state definitively whether bacteria play a primary or secondary role in HS.

### TABLE Demographic and Diagnostic Characteristics of Patients With Hidradenitis Suppurativa

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total (N=268)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender, n (%)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>189 (70.5)</td>
</tr>
<tr>
<td>Male</td>
<td>79 (29.5)</td>
</tr>
<tr>
<td>Age at diagnosis, y</td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>32.9 (12.6)</td>
</tr>
<tr>
<td>Median</td>
<td>30.6</td>
</tr>
<tr>
<td>Range</td>
<td>9.9–78.5</td>
</tr>
<tr>
<td>Race, n (%)</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>241 (90.3)</td>
</tr>
<tr>
<td>Nonwhite</td>
<td>26 (9.7)</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
</tr>
<tr>
<td>BMI (kg/m²) breakdown, n (%)</td>
<td></td>
</tr>
<tr>
<td>Underweight or normal (&lt;25.0)</td>
<td>49 (19.2)</td>
</tr>
<tr>
<td>Overweight (25.0–29.9)</td>
<td>66 (25.9)</td>
</tr>
<tr>
<td>WHO class I obesity (30.0–34.9)</td>
<td>49 (19.2)</td>
</tr>
<tr>
<td>WHO class II obesity (35.0–39.9)</td>
<td>49 (19.2)</td>
</tr>
<tr>
<td>WHO class III obesity (40.0–49.9)</td>
<td>33 (12.9)</td>
</tr>
<tr>
<td>Super obese (50+)</td>
<td>9 (3.5)</td>
</tr>
<tr>
<td>Unknown*</td>
<td>13</td>
</tr>
<tr>
<td>Smoking status, n (%)</td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>79 (29.8)</td>
</tr>
<tr>
<td>Current</td>
<td>153 (57.7)</td>
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<tr>
<td>Former</td>
<td>33 (12.5)</td>
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<tr>
<td>Unknown</td>
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</tr>
<tr>
<td>1st- or 2nd-degree family members affected?</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>22 (8.2)</td>
</tr>
<tr>
<td>No</td>
<td>117 (43.7)</td>
</tr>
<tr>
<td>Unknown</td>
<td>129 (48.1)</td>
</tr>
<tr>
<td>Time between symptom onset and diagnosis, y</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>156</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>5.1 (5.8)</td>
</tr>
<tr>
<td>Median</td>
<td>3.3</td>
</tr>
<tr>
<td>Range</td>
<td>0.0–30.0</td>
</tr>
<tr>
<td>Diagnosis of HS rendered by, n (%)</td>
<td></td>
</tr>
<tr>
<td>Dermatologist</td>
<td>171 (63.8)</td>
</tr>
<tr>
<td>Nondermatologist but meets 4 criteria listed</td>
<td>97 (36.2)</td>
</tr>
</tbody>
</table>

BMI=body mass index; WHO=World Health Organization.

* Of the 19 patients missing BMI, 6 patients had a description of body habitus by the physician that was recorded and abstracted.

Source: Adapted from Vazquez et al. 6 globalacademycme.com/dermatology • Hidradenitis Suppurativa: Update on Diagnosis and Treatment
Hormonal Factors
The possible role of hormones in HS pathogenesis has been proposed but not established, despite numerous studies.\textsuperscript{15-17} The age distribution of the disease in women (postpubertal and premenopausal) provides incentive for exploring a hormonal component, at least in women. Although a number of authors suggest that hyperandrogenism, specifically, may be a factor, the increased incidence of HS among women belies this claim, and the evidence available does not support this association.

Host Defense Factors
Dvorak and colleagues\textsuperscript{18} concluded more than 35 years ago that host defense mechanisms are not impaired in patients with HS. These investigators found that granulocyte function and cell-mediated immune mechanisms were intact and immunoglobulin levels normal; an important role for an inflammatory process was demonstrated by elevated complement levels in all patients with HS in their study. A more recent study suggested that increased production of oxygen free radicals by neutrophils may play a role in HS.\textsuperscript{19} Enhanced expression of toll-like receptors and release of proinflammatory cytokines by macrophages and dendritic cells in HS lesions has also been demonstrated.\textsuperscript{20} Within the past decade, increased tumor necrosis factor (TNF)-\(\alpha\) expression has been observed in HS, and TNF-\(\alpha\) inhibitors have demonstrated efficacy in its treatment, suggesting that that inflammatory pathway may play a role.\textsuperscript{21-23} Additionally, both activation of the interleukin-23/T-helper cell type 17 pathway and overproduction of interleukin-1\(\beta\) have been observed in HS lesions and may prove to be important therapeutic targets.\textsuperscript{24-26}

Conclusion
Ultimately, HS is probably best thought of as a disease of follicular occlusion and chronic inflammation that is multifactorial and remains incompletely understood. It is not rare in the general population and is a significant source of morbidity among those with the disease. An improved understanding of the factors that contribute to and exacerbate HS is necessary to guide advances in its treatment and management.

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13. Lapins J, Jarstrand C, Estenstad L. Coagulase-negative staphylococci are the most common bacteria found in cultures from the deep portions of hidradenitis suppurativa lesions, as obtained by carbon dioxide laser surgery. Br J Dermatol. 1999;140:90-95.
The diagnosis of hidradenitis suppurativa (HS) is a clinical one based on characteristic history and physical exam. No confirmatory laboratory tests exist, and although histologic features can support the diagnosis or rule out alternate diagnoses, histologic findings alone are not diagnostic. Therefore, skin biopsies generally are not recommended.

Natural History and Clinical Presentation

HS most commonly is seen in patients in the second and third decades of life. The disease is rare in prepubertal children, and onset of the disease is less common later in life. The most common sites of involvement are the axillae, groin, buttocks, and perianal, perineal, and mammary regions, with the axillae most commonly affected. Lesion distribution varies by sex: The most common sites of involvement in men are the inframammary, axillary, and inguinal areas; in women, the inframammary, axillary, and inguinal areas; and perianal, perineal, mammary, and inframammary areas, which rupture and drain a thick, mucopurulent, foul-smelling fluid. Later, sinus tracts form, and, over time, palpable, ropelike fibrotic subcutaneous scars often occur, which can lead to disabling contractures of the affected limbs. Clinically, the severity of disease is classified using the Hurley staging system, which provides guidance for choosing among treatment options.

Without intervention, the disease typically progresses to form more fluctuant and more painful, subcutaneous nodules that resemble furuncles. Unlike typical furuncles, these lesions may lack characteristic central rupture and drainage, instead opening laterally and draining a thick, mucopurulent, foul-smelling fluid. Deep dermal abscesses may join and progress to form chronically draining sinus tracts colonized by mixed bacterial flora. Long-standing sinus tracts form palpable, ropelike fibrotic subcutaneous scars (Figure 3). Particularly severe or extensive sinus networks can extend into deeper tissue, including muscle, fascia, lymph nodes, and other structures, depending on anatomic location (for example, the urethra or rectum).

FIGURE 1 Hidradenitis Suppurativa Lesions of Posterior Scalp and Neck. This 23-year-old male patient presented with multiple inflammatory nodules in the scalp and neck area. This atypical anatomic presentation occurs more frequently in men than in women.

Photo courtesy of Alan Menter, MD.
Establishing the Diagnosis

HS is a clinical diagnosis based on morphology and history, summarized by three key features. The diagnosis of HS can be made confidently if (1) the lesions are typical, (2) occur in the characteristic distribution, (3) and are chronic or recurrent. The onset of lesions usually is insidious, and patients typically are otherwise young and healthy.

The differential diagnosis for HS differs according to whether early or late lesions are present (Table 1). Among the list of possible diagnoses in patients with early HS lesions based on clinical appearance alone, the most common and most likely are furuncles, folliculitis, and atypically dilated comedones of acne vulgaris.

Distribution and anatomic location further narrow the clinical differential. Characteristic bilateral involvement of the axillae, perineum, or inguinal folds is suggestive of HS, whereas a single, unilateral nodule more likely represents a staphylococcal furuncle. Lastly, the appearance of characteristic lesions in characteristic locations in a recurrent or cyclic fashion, with onset, rupture, healing, and reappearance, suggests HS.

Unusual or atypical mimics of HS that could be considered include Bartholin’s abscess in female patients as well as, in either sex, pilonidal cyst, granuloma inguinale and lymphogranuloma venereum, Crohn’s disease, scrofuloderma, and other infectious or neoplastic causes of perforating lymph nodes.

Alikhan et al2 proposed an algorithm for diagnostic workup based on four questions: (1) Is there more than a single inflamed lesion? (2) Is the course chronic with new and recurrent lesions? (3) Are the lesions bilateral? (4) Are the lesions located primarily in the milk line? A positive answer to all four questions indicates a diagnosis of HS. If the answer to any of these is negative, the authors suggest a systematic strategy for further investigation according to which feature is atypical.

TABLE 1 Differential Diagnosis of Hidradenitis Suppurativa

<table>
<thead>
<tr>
<th>Early lesions</th>
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<tbody>
<tr>
<td>• Acne</td>
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<tr>
<td>• Carbuncles</td>
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<tr>
<td>• Cellulitis</td>
</tr>
<tr>
<td>• Erysipelas</td>
</tr>
<tr>
<td>• Folliculitis</td>
</tr>
<tr>
<td>• Furuncles</td>
</tr>
<tr>
<td>• Inflamed epidermal inclusion cyst</td>
</tr>
<tr>
<td>• Lymphadenopathy</td>
</tr>
<tr>
<td>• Perirectal abscess</td>
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<tr>
<td>• Pilonidal cyst</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Late lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Actinomycosis</td>
</tr>
<tr>
<td>• Anal fistula</td>
</tr>
<tr>
<td>• Cat scratch disease</td>
</tr>
<tr>
<td>• Crohn’s disease</td>
</tr>
<tr>
<td>• Granuloma inguinale</td>
</tr>
<tr>
<td>• Ischiorectal abscess</td>
</tr>
<tr>
<td>• Lymphogranuloma venereum</td>
</tr>
<tr>
<td>• Nocardia infection</td>
</tr>
<tr>
<td>• Noduloulcerative syphilis</td>
</tr>
<tr>
<td>• Pilonidal disease</td>
</tr>
<tr>
<td>• Tuberculous abscess</td>
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</table>

FIGURE 2 Severe Axillary Hidradenitis Suppurativa. The axillary region in this patient has active, draining sinus tracts that have formed ropelike, fibrotic subcutaneous scars.
Photo courtesy of Robert G. Micheletti, MD.

FIGURE 3 Severe Perineal Hidradenitis Suppurativa. Note the swelling, induration, and draining sinus tracts in the perineal region in this male patient.
Photo courtesy of Robert G. Micheletti, MD.
**Histologic Manifestations**
A biopsy is indicated to rule out an alternate disease process if HS is suspected but the clinical features are atypical. Histologic findings of HS depend on lesion age but may include follicular hyperkeratosis and plugging, follicular dilation and rupture, perifollicular lymphocytic or mixed inflammatory infiltrate, abscess formation, and sinus tracts with stratified squamous epithelium, foreign-body giant cells, and fibrosis.

**Disease Staging**
In 1989, Hurley\(^4\) proposed a clinical staging system for characterizing the extent of disease in patients with HS (Table 2). This simple system has been used in clinical trials and as the basis for choosing a particular therapy. For example, for patients with stage I disease (abscess formation without sinus tracts and scarring), a treatment regimen that includes oral doxycycline, chlorhexidine wash, and application of a clindamycin solution may be sufficient to manage active lesions. In contrast, a patient with more severe stage II or stage III disease will likely require measures more aggressive intervention to slow or arrest the progression of disease.

More recently, Sartorius and colleagues\(^5\) proposed a comprehensive staging system that yields regional and total scores based on anatomic regions affected, the number and types of lesions, and the extent of involvement (ie, the distance between lesions and the presence of normal skin between lesions). Given its complexity, the Sartorius system is better suited to quantifying disease extent and improvement in a research setting than in clinical practice but may be superior to the Hurley staging system for the former purpose.

<table>
<thead>
<tr>
<th><strong>Stage I</strong></th>
<th>Single or multiple abscesses, but no sinus tracts or scarring (cicatization)</th>
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<tbody>
<tr>
<td><strong>Stage II</strong></td>
<td>Single or multiple separated, recurrent abscesses with tract formation and scarring</td>
</tr>
<tr>
<td><strong>Stage III</strong></td>
<td>Multiple interconnected tracts and abscesses involving an entire anatomic area</td>
</tr>
</tbody>
</table>

**Importance of Early Diagnosis**
The goals of diagnosis and treatment are to reduce the burden of disease, including pain, odor, and drainage, and to prevent disease progression by healing existing inflammatory lesions and preventing the formation of new lesions, permanent sinus tracts, scars, fibrosis, and contracture, with the goal of improving quality of life. Because Hurley stage I disease can be controlled well in most patients, whereas effective control of more severe stage II or stage III disease can be elusive, early diagnosis and treatment is essential. Canou-Poitrine and colleagues\(^5\) reported that 68.2% of patients with HS have Hurley stage I disease, 27.9% have stage II disease, and 3.9% have severe, stage III, disease. Unfortunately, patients with early HS may be less likely to seek medical attention when discomfort and other symptoms are tolerable and lesions are easily hidden by clothing. Most present when lesions rupture, discomfort worsens, or discharge becomes messy, foul-smelling, or embarrassing. Some patients fail to recognize the disease for what it is and avoid medical attention until it is advanced and symptoms are severe.

Providers may similarly fail to recognize and accurately diagnose HS. It is not uncommon for patients to be seen initially in emergency departments or urgent clinics for incision and drainage of “boils.” Some patients return repeatedly to the emergency department for the temporary relief from discomfort that incision and drainage provide, until the diagnosis of HS is made and more useful and effective treatment is instituted for the disease.

Failure to diagnose HS accurately in stage I or II leads to unnecessarily prolonged patient discomfort and results in wasted expenditure of health care resources. Patient quality of life, including work and intimate relationships, may be severely affected during this time. HS is a highly distressing condition with an impact on quality of life as significant as any disease systematically studied in dermatology.\(^6\) Effective treatment options exist that can slow or arrest the disease process when lesions are mild. Progressive disease, with fibrotic scarring, contractures, and loss of limb function, such as may be seen in the presence of severe axillary disease, is much more difficult to treat and may result in permanent disability.

**Conclusion**
In most cases, a clinical diagnosis of HS can be made on the basis of presentation: The lesions of HS are typical, occur bilaterally in a characteristic distribution, and are chronic and recurrent. Routine biopsies and other laboratory tests are not generally helpful in the diagnosis of HS, except when the presentation is atypical and they are necessary to rule out other diagnoses. Early diagnosis and institution of appropriate therapy can improve both physical and emotional well-being and quality of life. The sequelae of long-standing disease can be devastating and permanently disabling.

**References**
Recognizing and Managing Comorbidities and Complications in Hidradenitis Suppurativa

Alan Menter, MD*

Abstract

The list of comorbidities associated with hidradenitis suppurativa (HS) is extensive, although these diseases do not necessarily share a common causality. Among the categories of comorbidities that are observed are obesity, other skin diseases, inflammatory conditions, and genetic disorders. Complications include scarring, restricted movement resulting from scarring and fibrosis in underlying tissue, conditions associated with obstructed lymph drainage, and psychosocial issues. Adverse effects on quality of life are common and may be severe, including unemployment, deterioration of family and other social relationships, and suicidal ideation. Clinical intervention for HS must include consideration and attention to these comorbidities and complications.

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Keywords

Comorbidities; dermatologic diseases; hidradenitis suppurativa; immune-mediated inflammatory diseases; obesity

H
dradenitis suppurativa (HS) is a disease that must be considered and treated in a context much broader than just the consideration of skin lesions and sinus tracts. Clinicians who diagnose HS also must recognize and be prepared to manage the significant comorbidities and complications associated with this chronic, debilitating disease.

Comorbidities and HS

The comorbidities associated with HS have been widely discussed in the literature and include both common (Table 1) and rarely occurring associations. However, the term “association” is used here with the caveat that, in most instances, the exact nature of the association between HS and its comorbidities is unclear. The diseases are comorbid with respect to observed coexistence, but not necessarily with respect to a common causality. As future studies reveal additional information about the genetics of all chronic diseases, genetic associations and links will become more clearly defined. Meanwhile, it is not yet known whether one or more genetic defects exist that predispose individuals to both HS and the other frequently associated comorbidities. In addition, it is important to note that associations between HS and some conditions observed to be comorbid are not strongly supported by statistics from studies of large patient populations.

Obesity

The comorbidity most commonly associated with HS is obesity, with or without the other features that characterize the metabolic syndrome (hypertension, hypertriglyceridemia, low high-density lipoprotein levels, increased fasting blood sugar concentrations). Reported percentages of patients with HS who were overweight or obese range from slightly more than 50% to 75% or more in some older studies.

Obesity certainly can exacerbate the symptoms of HS through several mechanisms, including increasing the area of skin-to-skin contact and promoting increased sweating and occlusion. In addition, hormonal changes associated with obesity—resulting in androgen excess—has been proposed as a possible inciting factor, involving changes in the hair shaft that promote follicular occlusion.

However, beyond these associations, it is not known whether obesity is linked genetically to a predisposition for HS or just serves as a trigger for expression of the disease in susceptible individuals.

Comorbid Skin Conditions

HS is associated with numerous skin diseases, especially those categorized as follicular occlusion conditions (acne conglobata, dissecting cellulitis of the scalp, and pilonidal cyst) and acne vulgaris. In addition, other, less commonly seen comorbidities include acral pustular psoriasis, acanthosis nigricans, and pyoderma gangrenosum. Recently, a report was published suggesting that HS may result in systemic amyloidosis.

<table>
<thead>
<tr>
<th>TABLE Diseases Commonly Associated With Hidradenitis Suppurativa 1</th>
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<tbody>
<tr>
<td>Diseases of follicular occlusion (follicular occlusion triad)</td>
</tr>
<tr>
<td>• Acne vulgaris</td>
</tr>
<tr>
<td>• Acne conglobata</td>
</tr>
<tr>
<td>• Dissecting cellulitis of the scalp</td>
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<tr>
<td>Pilonidal cyst</td>
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<tr>
<td>Crohn’s disease</td>
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<tr>
<td>Obesity/metabolic syndrome</td>
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*Chief, Division of Dermatology, Baylor University Medical Center; Clinical Professor of Dermatology, University of Texas, Southwestern Medical School; Director, Fellowship Program, Baylor Psoriasis Center; Chair, Psoriasis Guidelines Committee, American Academy of Dermatology; Director, Dermatology Residency Program, Baylor University Medical Center, Dallas, TX.

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Address reprint requests to: Alan Menter, MD, 3900 Junius Street, Suite 145, Dallas, TX 75246; amderm@gmail.com

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Inflammatory Conditions
Patients with HS frequently experience symptoms of arthritis and arthralgia, noted particularly during HS flares and in association with acne conglobata. Immunologic associations between these conditions have been suggested but, to date, have not been clearly established.

For more than 2 decades, case reports and studies involving relatively small numbers of patients have appeared regarding a possible association between Crohn’s disease and HS. In some, Crohn’s disease occurred prior to the onset of HS; in others, HS symptoms appeared first. Church and colleagues reported that, in all such cases, HS lesions developed in the perineal and perianal areas, but patients frequently also had concomitant HS lesions in the axillae, groin, and buttocks. More generally, inflammatory bowel diseases, as a group, are commonly associated with HS.

HS also has been reported as a comorbid condition in patients with several rare syndromes, including SAPHO (synovitis, acne, pustulosis, hyperostosis, and osteitis) and PAPA (pyogenic arthritis, pyoderma gangrenosum, and acne) syndromes.

Genetic Disorders and HS
Clinicians with practices containing a large number of patients with HS have on occasion seen patients with genetic disorders including Down syndrome and KID (keratitis, ichthyosis, and deafness) syndrome. The existence of these genetic comorbidities raises the issue of possible common sources of disease expression, but also raises questions about why certain individuals with conditions such as the metabolic syndrome—a clear comorbidity for HS—develop HS, whereas others do not, and why certain patients with HS tend to have disease that progresses slowly and seems to remain at the Hurley stage II level, whereas others progress to stage III, sometimes in a rapid course.

Complications of HS
The most common complications associated with HS can be grouped into four categories: scars, restricted movement resulting from surface scarring and fibrotic changes in underlying tissue, obstructed lymph drainage, and psychosocial issues. In addition, case reports have been published describing the development of squamous cell carcinoma (SCC) and other malignancies in patients with long-standing HS.

Scarring in HS is frequently cosmetically disfiguring, contributing to the psychosocial problems so common with this disease. Strictures of the anus, rectum, and urethra may occur secondary to HS lesions in the groin and genitourinary areas. Contractures from scarring and fibrosis in the axillae and groin, a complication of a long-standing disease process, can result in limited mobility of the arms and legs, respectively.

Severe lymphedema is a late complication resulting from scarring of the lymph glands and obstruction of lymph drainage. Cases of disfiguring and disabling genital swelling also have been reported, including scrotal elephantiasis.

As described earlier, patients with HS of long duration have an increased risk for malignancy, particularly SCC. In a retrospective study involving more than 2000 patients, Lapins and colleagues found that half of the patients with HS had an increased risk for malignancy. With respect to SCC specifically, the risk for this type of cancer was increased by 4.6-fold among patients with HS; men with HS are more likely than women to develop SCC.

Psychosocial Issues
Emarrassment, social isolation, and depression are inevitable consequences of HS. The exudates—often foul-smelling—that drain from active lesions are difficult to conceal, as are the multiple sinuses, contractures, and scars of healed lesions (Figures 1–3).

FIGURE 1 Axillary Hidradenitis Suppurativa. This 53-year-old female patient has draining sinuses and scarring from previously healed tracts. Photo courtesy of Alan Menter, MD.

FIGURE 2 Multiple Lesions in Hidradenitis Suppurativa. This 32-year-old male patient has comedones, inflammatory nodules, multiple draining sinuses, and scarring in the axillary region. Photo courtesy of Alan Menter, MD.

FIGURE 3 Inframammary Hidradenitis Suppurativa. These multiple draining nodules and sinuses formed in the inframammary folds of this 23-year-old obese male patient. Significant scarring also is evident. Photo courtesy of Alan Menter, MD.
In a study of quality-of-life (QOL) impairment in 61 hospitalized patients, Wolkenstein et al. used validated QOL instruments to compare HS with other chronic skin diseases, including psoriasis, chronic urticaria, and atopic dermatitis. The reported impairment in patients with HS was substantially more severe, although the authors note that the 61 subjects were hospitalized (ie, at the severe end of the HS spectrum).

In a 1996 study, Jemec and colleagues reported that, overall, patients with HS lost an average of 2.7 workdays per year, but those with severe disease were unable to work at all because of permanent disabilities. In an early study attempting to characterize the specific psychosocial implications of HS, Anderson and coworkers reported that five of six patients in their study of patients with HS in the perineum, buttocks, and groin were unemployed and poor, and were divorced or reclusive. In an even earlier study, Anderson and Perry observed that axillary HS was associated with increased rates for unemployment, poverty, family deterioration, and suicidal ideation. More recently, Matusiak and colleagues described the adverse effects of HS on QOL and professional activity, and Kurek et al. studied sexual health in patients with HS and reported that the adverse effects were “profound.”

Intervention for Comorbidities and Complications

With the exception of obesity, it is not known whether the treatment of comorbid conditions affects the development or course of HS; conversely, effective management of HS may have no implications for comorbid diseases. Weight loss certainly can decrease the risk for disease progression and can be an essential adjunct to disease-specific treatment. The reduction of skin-fold area can decrease friction, sweating, and mechanical occlusion of follicles, all factors that contribute to the development of HS lesions. In addition, other comorbid conditions that are part of the metabolic syndrome (eg, diabetes) will also be ameliorated by appropriate diet, weight loss, and even gastric bypass surgery in the morbidly obese patient.

Early identification and effective treatment of HS can prevent or mitigate complications of scarring, fibrotic changes, obstruction of lymph drainage, and the sequelae associated with these complications.

In addition to prompt and effective medical management of the physical aspects of the disease, clinicians can and should provide education, support, and practical information that can help patients cope with the psychosocial consequences of HS. Such efforts are easier to incorporate in dermatology, gynecology, family medicine, and primary care clinician practices than in the emergency care settings. However, because many patients with HS are seen in emergency departments, specialists in emergency medicine also can provide help in this area, despite the limitations of patient exposure time inherent in the emergency room.

Clinical experience shows that acknowledging to patients that their disease can be challenging and that support is available among others who are coping with HS can be comforting. Providing information, such as a patient education handout, and website information are enormously helpful. Strategies for coping with the psychosocial consequences of HS, including practical suggestions for managing employment and social challenges, have the potential to change the negative psychosocial milieu that many of these patients endure.

Conclusion

HS has long been considered an orphan disease; however, evidence accumulated over the past decade has shown that the actual number of patients affected may be far greater than was previously known. Rather, HS has been a hidden disease, with many patients being identified only when the severity of symptoms drives them to seek the help of a medical practitioner—often in the operating room or in the hospital emergency department. Fortunately, HS has been the focus of therapeutic, academic, and research attention recently, and the unmet needs of patients with HS in both improved medical therapy and attention to psychosocial issues—now are being addressed. Patient-focused support groups exist in many areas, with access to this type of assistance continuing to grow. Meanwhile, it is imperative that clinicians counsel patients and their families appropriately and, whenever possible, provide practical information such as printed handouts and contact information for patient support resources.

References

Current and Emerging Nonsurgical Treatment Options for Hidradenitis Suppurativa

Francisco A. Kerdel, BSc, MBBS*

Abstract
Several nonsurgical strategies for managing hidradenitis suppurativa (HS) are used that are successful in many patients. The overall goals of pharmacologic therapy are to clear or reduce the number and extent of current lesions and to prevent new lesions from forming. No pharmacologic agent is universally effective in all patients with HS, and, to date, none has been approved for this indication by the US Food and Drug Administration. Among the agents most commonly used are topical and systemic antibiotics and intralesional and systemic corticosteroids. Within the past decade, clinical experience with biologic agents— principally, tumor necrosis factor inhibitors—has been described, and the results of clinical trials with these agents in patients with HS have been promising. The treatment options for patients with hidradenitis suppurativa (HS) include both surgical and medical modalities. The severity, extent, chronicity, and anatomic location of HS lesions determine which treatment—or combination of modalities—is most appropriate for a given individual case. Even a cursory scan of the medical literature on HS treatment suggests that surgery is the only curative method of choice. Articles abound reporting the use of various surgical techniques and their short-term outcomes. Certainly, patients with advanced disease may do well with surgery, but these procedures can be extensive and associated with high morbidity. In addition, depending on the operative site and the extent of dissection, surgery can result in disfigurement and loss of function. Moreover, long-term follow-up data are not available that demonstrate cure without recurrence, quality-of-life results, or patient satisfaction. Thus, surgery should not be the treatment of first choice in every case but should be considered along with medical therapy in developing an individualized treatment plan.

In addition to conventional surgical modalities (including deroofing and excision procedures) and photodynamic therapy, the use of laser therapy—especially the long-pulse neodymium yag laser—has shown promise in some patients, resulting in clearance of nodules and sinususes, including deep lesions. A variety of pharmacologic treatments have been used, with varying degrees of success (Figure). To date, no medical treatment has been approved by the US Food and Drug Administration (FDA) specifically for the treatment of HS. This article provides an overview of the medical therapies currently in use, including the most recent addition to the roster of options, the biologic anti-inflammatory agents.

The overall goals of pharmacologic therapy are to clear or reduce the number and extent of lesions and to prevent new lesions from developing. Theoretically, success in achieving these goals also should result in reduced scarring and other complications and sequelae. Some of the medications commonly used are antibiotics (usually with topical chlorhexidine or similar skin washes), retinoids, hormones, corticosteroids, immunosuppressants, metformin, and, most recently, biologic anti-inflammatory agents.

Antibiotics
For many clinicians, the mainstay of initial therapy for mild to moderate HS comprises topical or systemic antibiotics, a strategy initially based on the clinical similarities between HS lesions and acne conglobata. Antibiotics do not clear HS lesions, but they are administered to treat and prevent secondary infection and the associated inflammation in existing lesions, and to prevent new breakouts. Very few studies have been done in recent years to assess the efficacy of antibiotics in HS, and published double-blind and comparative trials are even more sparse.

Clindamycin is commonly used. The benefit of topical clindamycin was demonstrated in an early, small, double-blind placebo-controlled study in HS in which the medication was statistically superior (P<0.01) to placebo in reducing the number of abscesses, inflammatory nodules, and pustules. A study of topical clindamycin and oral tetracycline failed to demonstrate superior efficacy of the systemic medication.

Oral clindamycin plus rifampicin was evaluated in two retrospective studies published in 2009. One involved 34 patients who took 600 mg/day of clindamycin and 600 mg/day of rifampicin. Total remission was seen in 16 patients (47%) at 10 weeks; an additional 12 patients experienced at least some improvement. Thus, in this study, a total of 28 patients—or 82%—had at least some benefit from using this combination. In the other study, patients had taken 300 mg of clindamycin...
twice daily and 600 mg/day of rifampicin. At 10 weeks, the 70 patients (of the original 116) for whom data were available had significant improvement \((P < 0.001)\) in disease severity (measured by the Sartorius scale) and quality of life (measured by the Dermatology Life Quality Index [DLQI]).

Other antibiotics, including dapsone, also have been tried in patients with HS, although studies of these agents are limited and have yielded mixed results.

**Retinoids**

Because of the clinical resemblance between HS and nodular cystic acne, as well as their similar pathophysiologic mechanism—namely, follicular occlusion—isotretinoin has been studied in HS, but without good results. For example, in a retrospective study of 358 patients, Soria and colleagues showed that only 16.1% of patients experienced an improvement; most of the patients (77%) had no improvement, and 6.9% had a worsening of their HS.

**Hormones**

In some women with HS, symptoms seem to correlate with hormonal fluctuations during the menstrual cycle. Indeed, a hormonal connection with HS is suggested by the gender and age distribution pattern—HS is three times more common among women than men, and onset rarely is seen after menopause. Anecdotally, some patients with HS have reported symptom improvement during the use of combination estrogen/progesterone oral contraceptive use. Also, some clinicians have used spironolactone, although no studies of this agent in HS have been reported.

In men with HS, finasteride has been used with some success, and recently Randhawa and colleagues reported good results with this agent in three children and adolescent patients with HS.

**Corticosteroids**

Intralesional injection of a topical corticosteroid such as triamcinolone acetonide commonly is done to redue the pain and swelling of individual lesions and to achieve drainage of an abscess. Systemic corticosteroids such as prednisone reduce inflammation and may help clear existing HS lesions and prevent additional lesions from forming. Because of the increased risk for side effects with sustained use over time, corticosteroids are not a long-term therapeutic option.

**Immunosuppressants**

Amelioration of HS—presumably, through reduction of inflammation—has been reported with the use of methotrexate or cyclosporine in patients using these medications for other reasons (such as prevention of rejection of a transplanted organ). These agents have not been widely studied in HS.

**Metformin**

Metformin, the glucose-lowering biguanide agent approved for the treatment of type 2 diabetes mellitus, has shown some benefit in female patients with HS. No formal studies have been done to evaluate the safety and efficacy of metformin in HS.

**Biologic Agents**

The study of inhibitors of tumor necrosis factor (TNF) for HS was prompted by the finding that some patients with HS who were treated with the anti-TNF agent infliximab for Crohn’s disease experienced improvement in HS lesions. More than 20 articles have been published reporting this benefit in the clinical context of Crohn’s disease therapy.

As has been demonstrated in the treatment of numerous immune-mediated inflammatory diseases—including Crohn’s disease, rheumatoid arthritis, and psoriasis—interruption of the underlying inflammatory processes can yield significant long-term therapeutic benefits. It is postulated that, although the inciting event in HS is follicular occlusion (albeit from an as-yet-unidentified underlying cause), the resulting inflammatory response may be the process responsible for the disease progression, chronicity, associated morbidity, and, ultimately, the permanent tissue damage and associated disability that many patients with HS experience. Therefore, use of potent anti-inflammatory biologic agents seems a rational approach to control or prevent these inflammatory responses and their disfiguring and disabling sequelae in HS.

In a study of the long-term effects of one course of treatment with infliximab in 10 patients with severe, recalcitrant HS, Mekkes and Bos found that all patients improved within 2 to 6 weeks on both an acne score and the DLQI. After 2 years of follow-up, three of these patients had no recurrence of HS lesions and maintained substantial improvements. The other seven patients experienced recurrence within 4.3 to 13.4 months (mean, 8.5 months).

More recently, the first double-blind prospective study of infliximab in moderate to severe HS (N=38 patients) was published. The trial consisted of three phases: the first was an 8-week double-blind phase, in which patients were given infliximab, 5 mg/kg (n=15), or placebo (n=23) at weeks 0, 2, and 6. After 8 weeks, the study was unblinded, and patients in the placebo group were offered infliximab treatment (induction therapy was given at weeks 8, 10, and 14, and then two additional doses every 8 weeks—ie, at weeks 22 and 30). During this second, open-label phase, patients who had received infliximab since the start of the study received infliximab every 8 weeks through week 22. The third phase involved observation without additional treatment through week 52, during which patients were assessed monthly for adverse events and signs of relapse.

A post hoc analysis of composite response on the Hidradenitis Suppurativa Severity Index (HSSI) showed that 60% (n=9) of patients treated with infliximab had improvements in the HSSI ranging from 25% to 50% compared to 5.6% in the placebo group \((P < 0.001)\). Most patients treated in the placebo group (88.9%) had decreases from baseline in the HSSI of less than 20%.

**FIGURE Severe Axillary Hidradenitis Suppurativa.** This patient has severe disease, with friable, ulcerated tissue, scarring, and draining sinus tracts. The goals of pharmacologic therapy include clearing or reducing the number and extent of lesions and preventing new lesions from developing.

Photo courtesy of Robert G. Micheletti, MD.
25%; only 13.3% of patients in the infliximab group had decreases in the HSSI of less than 25% (P<0.001). In addition, substantial improvements were seen in secondary endpoints, including the DLQI, physicians’ global assessment, and pain. Significant improvement was seen with infliximab treatment on a visual analog scale (VAS) of self-reported magnitude of pain. The mean VAS at baseline was 53.3 in the infliximab-treated group; at week 8, the mean change from baseline was 39.8 (down to 13.5). In contrast, the mean VAS at baseline was 49.7 in the placebo group, and, after 8 weeks, the mean VAS was 49.2 (P<0.001 versus the infliximab group).10

Etanercept also had been reported to have some benefit in HS. However, two prospective studies of etanercept versus placebo failed to demonstrate significant improvement.11,12

A third TNF inhibitor, adalimumab, also showed promise in early studies. Although early case reports and series demonstrated mixed results, a phase II parallel, randomized placebo-controlled trial demonstrated a clear benefit in a group of 154 adult patients with moderate to severe disease who had failed a trial of oral antibiotics.13 At the beginning of the study, patients were randomized (1:1:1) to receive adalimumab 40 mg/week, adalimumab 40 mg every other week, or placebo for 16 weeks (the blinded period), after receiving loading doses of 160 mg of adalimumab at week 0 and 80 mg at week 1. At the beginning of period 2 of the study (the 36-week open-label period), all patients were given adalimumab 40 mg every other week; those with a suboptimal response at week 28 or 31 were switched to weekly dosing. At week 16, 9 of 51 (17.6%) patients in the weekly-dose active treatment group had achieved a clinical response compared with 5 of 52 (9.6%) patients in the group who received adalimumab every other week and 2 of 51 (3.9%) patients who received placebo. Importantly, substantial improvements in pain intensity also were seen with adalimumab use.

Subsequently, two large phase III clinical trials were launched in 2013 to evaluate adalimumab in HS. The multicenter, multinational phase III trials (PIONEER I and PIONEER II) involved about 600 patients with moderate to severe HS. Enrollment was limited to patients who had had moderate to severe HS for at least 1 year, with stable disease for the 2 months prior to beginning the study; a total abscess and inflammatory nodule count of three or more, the location of lesions in two distinct anatomic areas, and an inadequate response to a trial of an oral antibiotic agent for a minimum of 3 months were also inclusion criteria. The trials were completed in early 2014, so results were not available at the time of publication of this article.

Two other biologic agents, the interleukin (IL)-1 receptor antagonist anakinra and the IL-12/23 inhibitor ustekinumab, also have been reported to be helpful in HS. An open-label, nonrandomized phase II efficacy study of anakinra in HS showed promising results;14 a randomized, placebo-controlled phase II study of a similar compound is nearing completion at this time. A proof-of-concept phase II study of ustekinumab in HS currently is under way.

**Disease Severity Affects Treatment Choices**

Patients with mild disease (Hurley stage I) often respond to topical therapy. In addition to topical antibiotics such as clindamycin or mupirocin, topical treatment includes reducing skin bacteria populations with soaps, detergents, and antibacterial skin washes such as those used for acne vulgaris. In addition, overweight or obese patients should receive education about how excess weight corresponds to increased HS activity and should be counseled about the benefits of weight reduction in managing HS. Also, as cigarette smoking is associated with HS, smoking cessation should be strongly encouraged.

For patients with more severe disease (Hurley stage II), the topical measures described above should be employed, but long-term oral antibiotic regimens—such as clindamycin plus rifampicin or a tetracycline (minocycline or doxycycline)—should be considered. Patients with chronic outbreaks of HS in a particular anatomic area may want to consider laser hair removal in those regions as a preventive measure.

In severe disease, biologic agents should be considered. As noted above in the section on biologics, the TNF inhibitors infliximab and adalimumab (although not etanercept) have shown benefit in some patients. In addition, surgical intervention may be required to halt progression of the disease and mitigate scarring and subsequent disability.

**Conclusion**

Because HS is a relatively rare disease, large clinical trials of many of the treatments currently in use have not been conducted. Therefore, clinicians must rely on what data are available and use their best medical judgment in determining treatment strategies. Ideally, earlier diagnosis and treatment of HS will become more common, and the most severe stages of the disease and its comorbidities and sequelae will be prevented in more patients. In addition, the availability of evidence demonstrating efficacy of at least one anti-TNF agent will allow earlier effective management of moderate to severe disease, making potentially disabling surgery unnecessary.

**References**


**Additional Reading**


What You Should Know About Hidradenitis Suppurativa (HS)
Information for Patients

What is HS?
Hidradenitis suppurativa (pronounced “high-drad-en-eye-tis/sup-your-uh-fee-vah”) is a chronic skin disease that is characterized by occlusion (blockage) of the hair follicles and subsequent inflammation of the sweat glands.

The lesions occur most commonly on areas of skin-to-skin contact: under the arms (axillary area), in the groin, around the buttocks, in the region around the anus and genitals, and on the skin between and under the breasts. In women, the underarms, groin, and breast areas are most commonly affected. Men most often have HS lesions around the anus and under the arms and may also have HS at the back of the neck and behind and around the ears.

What does HS look and feel like?
The first thing that someone with HS notices is a tender, raised, red bump that looks like an under-the-skin pimple or boil. Sometimes HS lesions have two or more “heads.” These lesions often tingle and burn and may be associated with increased sweating.

Without medical attention, HS usually becomes more severe over time. It becomes more painful, and the lesions become larger and may open, oozing a thick, foul-smelling fluid possibly mixed with blood. Later, deeper abscesses develop and may connect with each other under the skin to form tunnel-like tracts (sinuses). Bacteria grow within these sinuses, which then drain fluid to the surface of the skin. In people who have had sinus tracts for some time, scars form that feel like ropes under the skin. In the very worst cases, networks of sinus tracts can form deeper in the body, including the muscle and other tissues. Many people with severe HS have scars that can limit their ability to freely move their arms or legs.

Clinicians usually classify or “grade” HS using the Hurley staging system according to the severity of the disease:
• Hurley stage I: one or more abscesses are present, but no sinus tracts have formed and no scars have developed
• Hurley stage II: one or more abscesses are present that resolve and recur; sinus tracts and scarring are seen
• Hurley stage III: an entire area of the body is involved; multiple abscesses and interconnecting sinus tracts are present.

What causes HS?
The cause of HS is not yet known. It is clear that some people are more prone than others to develop this disease, also for unknown reasons. HS most commonly occurs in people in their 20s and 30s; it is rarely seen in children and adolescents and is not common in older adults. Women are three times more likely than men to develop HS.

Finally, certain activities and conditions seem to be associated with HS. Although there is no evidence that these factors actually cause HS, controlling seems to reduce the number of HS flare-ups that patients have. The factors most commonly associated with HS include:
• Cigarette smoking
• Overweight/obesity
• Mechanical and environmental factors, such as skin-on-skin friction in the skin folds, irritation from antiperspirants, and trauma to the hair follicles from shaving

It is very important to know that HS is not contagious, and it is not caused by poor hygiene, poor nutrition, or being overweight.

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How do clinicians treat HS?
Clinicians use both medication and surgery to treat HS. The choice of treatment—or combination of treatments—is made according to an individual patient’s needs. Clinicians consider several factors in determining the most appropriate plan for therapy:
• Severity of disease
• Extent of disease
• Chronicity (how often the lesions recur)
• Location of the lesions

A number of different surgical methods have been developed that are useful for certain patients under particular circumstances. In addition, many medical treatments have been tried—some with more success than others. No medication is effective for all patients, and you and your clinician may have to try several different agents or combinations of agents before you find the treatment plan that works best for you.

The goals of therapy with medications that are either topical (used on the skin) or systemic (taken by mouth) are:
1. to clear the lesions or at least reduce their number and extent, and
2. to prevent new lesions from forming.

Some of the types of medications commonly used are antibacterial skin washes and the topical antibiotics to prevent secondary infections and corticosteroid injections into the lesions to reduce inflammation.

Other medications that may be used include retinoids, hormones, immunosuppressive agents (such as methotrexate), the antidiabetic medication metformin, and biologic anti-inflammatory medications such as infliximab and adalimumab.

What self-help measures are useful?
A number of measures seem to help many individuals with HS. Your clinician can help you determine which are likely to be best for you. However, two of these probably apply to most patients with HS:
1. if you smoke cigarettes, quit and
2. decrease your body weight.

Although there are no studies showing that quitting smoking and losing weight improve HS, both of these factors have a negative effect on overall health. Also, weight loss may help prevent HS from worsening—the smaller the area of skin-to-skin contact (and, therefore, of sweating and rubbing), the smaller the target for the development of HS lesions.

Some other self-help measures are:
• Avoid skin trauma (such as shaving in areas, such as the armpits, where breakouts occur)
• Wash your skin gently, using a cleansing agent recommended by your clinician; cleansers such as benzoyl peroxide wash, used by patients with acne, may be appropriate for many patients
• Apply topical medications as directed and as often as prescribed
• Avoid tight-fitting or irritating clothing or bandaging
• Follow your clinician’s guidance about antiperspirants or deodorants
• Keep your skin cool (becoming overheated and sweating can contribute to an HS flare)
• To reduce the pain of cysts or nodules, apply hot compresses for 10 minutes at a time (use a clean washcloth or a teabag soaked in hot water)

Finally, know that you are not alone. Coping with the pain and other symptoms of HS can be very difficult, so it may be helpful to connect with others who live with HS. Patient groups and networks can be sources of important information and support. Some Internet resources for information and connections are provided below.

Resources for Information
American Academy of Dermatology
http://www.aad.org/dermatology-a-to-z/diseases-and-treatments/e--h/hidradenitis-suppurativa/signs-and-symptoms

National Library of Medicine

NORD: National Organization for Rare Disorders, Inc
https://www.rarediseases.org/rare-disease-information/rare-diseases/byID/358/viewAbstract

Trials of new medications for HS
https://www.clinicaltrials.gov
Hidradenitis Suppurativa: Update on Diagnosis and Treatment Post-Test

CME Questions: For each question or incomplete statement, choose the answer or completion that is correct. Circle the most appropriate response.

1. Which one of the following statements is true concerning hidradenitis suppurativa?
   A. Hidradenitis suppurativa is characterized by apocrine gland dysfunction.
   B. Hidradenitis suppurativa is a disease of chronic follicular occlusion.
   C. Apocrine gland inflammation is the primary inciting event in hidradenitis suppurativa, causing follicular occlusion.
   D. Obesity is the underlying cause of hidradenitis suppurativa in many patients.

2. Hidradenitis suppurativa is seen most commonly in__________.
   A. African-Americans
   B. Individuals in the second and third decades of life
   C. Men
   D. Menopausal women

3. A number of factors have been associated with hidradenitis suppurativa, although none has been established as a causative factor. The most common factor associated with the disease is ____________.
   A. Autosomal dominant inheritance
   B. Cigarette smoking
   C. Hyperandrogenicity
   D. Obesity

4. Hidradenitis suppurativa is a clinical diagnosis based on morphology and history, summarized by three key features. Which one of the following is not a key feature?
   A. The lesions are chronic or recurrent.
   B. The lesions are typical.
   C. The lesions occur in the characteristic distribution.
   D. The lesions occur in patients who have a history of immune-mediated inflammatory diseases.

5. A single, unilateral axillary abscess likely represents a diagnosis of ____________.
   A. Acne vulgaris
   B. Early hidradenitis suppurativa
   C. Folliculitis
   D. Furuncle

6. Patients with hidradenitis suppurativa of long duration have an increased risk for malignancy, particularly ____________.
   A. Lymphoma
   B. Melanoma
   C. Squamous cell carcinoma
   D. Tumors in the anogenital region

7. Which one of the following statements is true concerning hidradenitis suppurativa comorbidities or associated “risk factors”?
   A. Addressing known comorbid conditions, including diabetes mellitus, has been shown to have important implications for the management of hidradenitis suppurativa.
   B. Disease progression risk in obese patients can be decreased by weight loss.
   C. Effective management of hidradenitis suppurativa has been shown to have important implications for comorbid conditions.
   D. Smoking cessation has been shown to have important implications for the management of hidradenitis suppurativa.

8. Which one of the following statements accurately describes the role of surgery in hidradenitis suppurativa?
   A. Data show that surgery should be considered an option of last resort because most procedures are extensive and associated with high morbidity.
   B. Long-term follow-up data demonstrate good rates of cure without recurrence.
   C. Surgery is the curative method of first choice for patients with hidradenitis suppurativa.
   D. Surgery should be considered along with pharmacologic therapy in developing an individualized treatment plan in any patient with hidradenitis suppurativa.

9. Which one of the following statements about pharmacologic intervention in hidradenitis suppurativa is most appropriate?
   A. No pharmacologic treatment has been approved by the US Food and Drug Administration to date.
   B. No pharmacologic treatment that is approved by the US Food and Drug Administration is indicated for severe disease.
   C. The overall goal of pharmacologic therapy is to prevent the need for surgery.
   D. The overall goals of pharmacologic therapy are to reduce scarring and other complications and sequelae.

10. Which one of the following statements concerning pharmacologic therapy for hidradenitis suppurativa is accurate?
    A. Antibiotics are given to clear lesions.
    B. Because of the clinical resemblance between hidradenitis suppurativa and nodular cystic acne, isotretinoin has been shown to be effective in several large clinical trials.
    C. Because they target the inflammatory response to follicular occlusion, biologic agents such as infliximab and adalimumab have been shown to be effective in several controlled clinical trials.
    D. Cyclosporine and methotrexate have been shown in a number of large clinical trials to be effective in managing hidradenitis suppurativa.
We would appreciate your answering the following questions in order to help us plan for other activities of this type. All information is confidential. Please print.

Name: ___________________________________________________

Specialty: ________________________________________________

Degree: [ ] MD [ ] DO [ ] PharmD [ ] RPh [ ] NP [ ] RN
[ ] BS [ ] PA [ ] Other __________________________

Affiliation: ________________________________________________

Address: _________________________________________________

City: ___________________________ State: __________ ZIP: _______

Telephone: ________________________ Fax: ______________________

E-mail: ___________________________________________________

Signature: ________________________________________________

CME CREDIT VERIFICATION
I verify that I have spent _____ hour(s)/_____ minutes of actual time working on this CME/CNE activity. No more than 3.0 CME credits will be issued for this activity.

COURSE EVALUATION: GAPS
This activity was created to address the professional practice gaps listed below. Please respond regarding how much you agree or disagree that the following gaps were met:

• Recognizing hidradenitis suppurativa and properly diagnosing this skin disease.
• Utilizing best practice methods for diagnosing and treating hidradenitis suppurativa.
• Utilizing evidence-based treatment options for hidradenitis suppurativa.

Did participating in this educational activity improve your KNOWLEDGE in the professional practice gaps that are listed above?

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Please elaborate on your answer. ____________________________________________________________

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Did participating in this educational activity improve your COMPETENCE in the professional practice gaps that are listed on the left?

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Please elaborate on your answer. ____________________________________________________________

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Did participating in this educational activity improve your PERFORMANCE in the professional practice gaps that are listed on the left?

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Please elaborate on your answer. ____________________________________________________________

________________________________________________________

Please identify a change that you will implement into practice as a result of participating in this educational activity (new protocols, different medications, etc).

________________________________________________________

________________________________________________________

How certain are you that you will implement this change?

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What topics do you want to hear more about, and what issue(s) in your practice will they address?

________________________________________________________

________________________________________________________

Were the patient recommendations based on acceptable practices in medicine?  ☐ Yes ☐ No

If no, please explain which recommendation(s) were not based on acceptable practices in medicine. __________________________________________________________

________________________________________________________

________________________________________________________

Do you think the articles were without commercial bias?  ☐ Yes ☐ No

If no, please list the article(s) that was/were biased. __________________________________________________________

________________________________________________________

________________________________________________________

The University of Louisville thanks you for your participation in this CME activity. All information provided improves the scope and purpose of our programs and your patients’ care.

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