Management of Acute and Chronic Pain Associated With Hidradenitis Suppurativa: A Comprehensive Review of Pharmacologic and Therapeutic Considerations in Clinical Practice

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PRACTICE **POINTS**

- First-line therapies may not provide adequate pain control in many patients with hidradenitis suppurativa.
- Pain caused by inflamed abscesses and nodules can be treated with either intralesional corticosteroids or incision and drainage. Tissue-sparing surgical techniques may lead to shorter healing times and less postoperative pain.
- Long-term management involves lifestyle modifications and pharmacologic agents.
- The most effective pain remedies developed thus far are limited to surgery and tumor necrosis factor α inhibitors.

Hidradenitis suppurativa (HS), a chronic, inflammatory, recurrent cutaneous disorder of the hair follicles, is debilitating and has substantial morbidity. Hidradenitis suppurativa–related pain has a profound effect on patient quality of life, yet at present, there are no established pain management algorithms. This comprehensive review provides an update on current treatment of HS-associated pain, including a summary of existing literature surrounding pharmacologic treatments of acute, perioperative, and chronic pain. Additionally, the epidemiology, pathophysiology, and clinical features of the disease are summarized.

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Hidradenitis suppurativa (HS) is a chronic inflammatory, androgen gland disorder characterized by recurrent rupture of the hair follicles with a vigorous inflammatory response. This response results in abscess formation and development of draining sinus tracts and hypertrophic fibrous scars.[1,2] Pain, discomfort, and odorous discharge from the recalcitrant lesions have a profound impact on patient quality of life.[3,4]

The morbidity and disease burden associated with HS are particularly underestimated, as patients frequently report debilitating pain that often is overlooked.[5,6] Additionally, the quality and intensity of perceived pain are compounded by frequently associated depression and anxiety.[7-9] Pain has been reported by patients with HS to

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The eTable is available in the Appendix online at www.mdedge.com/dermatology.

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be the highest cause of morbidity, despite the disfiguring nature of the disease and its associated psychosocial distress.[7,10] Nonetheless, HS lacks an accepted pain management algorithm similar to those that have been developed for the treatment of other acute or chronic pain disorders, such as back pain and sickle cell disease.[4,11-13]

Given the lack of formal studies regarding pain management in patients with HS, clinicians are limited to general pain guidelines, expert opinion, small trials, and patient preference.[3] Furthermore, effective pain management in HS necessitates the treatment of both chronic pain affecting daily function and acute pain present during disease flares, surgical interventions, and dressing changes.[3] The result is a wide array of strategies used for HS-associated pain.[3,4]

Epidemiology and Pathophysiology

Hidradenitis suppurativa historically has been an overlooked and underdiagnosed disease, which limits epidemiology data.[5] Current estimates are that HS affects approximately 1% of the general population; however, prevalence rates range from 0.03% to 4.1%.[14-16]

The exact etiology of HS remains unclear, but it is thought that genetic factors, immune dysregulation, and environmental/behavioral influences all contribute to its pathophysiology.[1,17] Up to 40% of patients with HS report a positive family history of the disease.[18-20] Hidradenitis suppurativa has been associated with other inflammatory disease states, such as inflammatory bowel disease, spondyloarthropathies, and pyoderma gangrenosum.[16,21,22]

It is thought that HS is the result of some defect in keratin clearance that leads to follicular hyperkeratinization and occlusion.[1] Resultant rupture of pilosebaceous units and spillage of contents (including keratin and bacteria) into the surrounding dermis triggers a vigorous inflammatory response. Sinus tracts and fistulas become the targets of bacterial colonization, biofilm formation, and secondary infection. The result is suppuration and extension of the lesions as well as sustained chronic inflammation.[23,24]

Although the etiology of HS is complex, several modifiable risk factors for the disease have been identified, most prominently cigarette smoking and obesity. Approximately 70% of patients with HS smoke cigarettes.[2,15,25,26] Obesity has a well-known association with HS, and it is possible that weight reduction lowers disease severity.[27-30]

Clinical Presentation and Diagnosis

Establishing a diagnosis of HS necessitates recognition of disease morphology, topography, and chronicity. Hidradenitis suppurativa most commonly occurs in the axillae, inguinal and anogenital region, perineal region, and inframammary region.[5,31] A typical history involves a prolonged disease course with recurrent lesions and intermittent periods of improvement or remission. Primary lesions are deep, inflamed, painful, and sterile. Ultimately, these lesions rupture and track subcutaneously.[15,25] Intercommunicating sinus tracts form from multiple recurrent nodules in close proximity and may ultimately lead to fibrotic scarring and local architectural distortion.[32] The Hurley staging system helps to guide treatment interventions based on disease severity. Approach to pain management is discussed below.

Pain Management in HS: General Principles

Pain management is complex for clinicians, as there are limited studies from which to draw treatment recommendations. Incomplete understanding of the etiology and pathophysiology of the disease contributes to the lack of established management guidelines.

A PubMed search of articles indexed for MEDLINE using the terms *hidradenitis*, *suppurativa*, *pain*, and *management* revealed 61 different results dating back to 1980, 52 of which had been published in the last 5 years. When the word *acute* was added to the search, there were only 6 results identified. These results clearly reflect a better understanding of HS-mediated pain as well as clinical unmet needs and evolving strategies in pain management therapeutics. However, many of these studies reflect therapies focused on the mediation or modulation of HS pathogenesis rather than potential pain management therapies.

In addition, the heterogenous nature of the pain experience in HS poses a challenge for clinicians. Patients may experience multiple pain types concurrently, including inflammatory, noninflammatory, nociceptive, neuropathic, and ischemic, as well as pain related to arthritis.[3,33,34] Pain perception is further complicated by the observation that patients with HS have high rates of psychiatric comorbidities such as depression and anxiety, both of which profoundly alter perception of both the strength and quality of pain.[7,8,22,35] A suggested algorithm for treatment of pain in HS is described in the eTable.[36]

Chronicity is a hallmark of HS. Patients experience a prolonged disease course involving acute painful exacerbations superimposed on chronic pain that affects all aspects of daily life. Changes in self-perception, daily living activities, mood state, physical functioning, and physical comfort frequently are reported to have a major impact on quality of life.[1,3,37]

In 2018, Thorlacius et al[38] created a multistakeholder consensus on a core outcome set of domains detailing what to measure in clinical trials for HS. The authors hoped that the routine adoption of these core domains would promote the collection of consistent and relevant information, bolster the strength of evidence synthesis, and minimize the risk for outcome reporting bias among studies.[38] It is important to ascertain the patient's description of his/her pain to distinguish between stimulus-dependent nociceptive pain vs spontaneous neuropathic pain.[3,7,10] The most common pain descriptors used by patients are "shooting," "itchy," "blinding," "cutting," and "exhausting."[10] In addition to obtaining

descriptive factors, it is important for the clinician to obtain information on the timing of the pain, whether or not the pain is relieved with spontaneous or surgical drainage, and if the patient is experiencing chronic background pain secondary to scarring or skin contraction.[3] With the routine utilization of a consistent set of core domains, advances in our understanding of the different elements of HS pain, and increased provider awareness of the disease, the future of pain management in patients with HS seems promising.

Acute and Perioperative Pain Management

Acute Pain Management—The pain in HS can range from mild to excruciating.[3,7] The difference between acute and chronic pain in this condition may be hard to delineate, as patients may have intense acute flares on top of a baseline level of chronic pain.[3,7,14] These factors, in combination with various pain types of differing etiologies, make the treatment of HS-associated pain a therapeutic challenge.

The first-line treatments for acute pain in HS are oral acetaminophen, oral nonsteroidal anti-inflammatory drugs (NSAIDs), and topical analgesics.[3] These treatment modalities are especially helpful for nociceptive pain, which often is described as having an aching or tender quality.[3] Topical treatment for acute pain episodes includes diclofenac gel and liposomal lidocaine cream.[39] Topical lidocaine in particular has the benefit of being rapid acting, and its effect can last 1 to 2 hours. Ketamine has been anecdotally used as a topical treatment. Treatment options for neuropathic pain include topical amitriptyline, gabapentin, and pregabalin.[39] Dressings and ice packs may be used in cases of mild acute pain, depending on patient preference.[3]

First-line therapies may not provide adequate pain control in many patients.[3,40,41] Should the first-line treatments fail, oral opiates can be considered as a treatment option, especially if the patient has a history of recurrent pain unresponsive to milder methods of pain control.[3,40,41] However, prudence should be exercised, as patients with HS have a higher risk for opioid abuse, and referral to a pain specialist is advisable.[40] Generally, use of opioids should be limited to the smallest period of time possible.[40,41] Codeine can be used as a first opioid option, with hydromorphone available as an alternative.[41]

Pain caused by inflamed abscesses and nodules can be treated with either intralesional corticosteroids or incision and drainage. Intralesional triamcinolone has been found to cause substantial pain relief within 1 day of injection in patients with HS.[3,42]

Prompt discussion about the remitting course of HS will prepare patients for flares. Although the therapies discussed here aim to reduce the clinical severity and inflammation associated with HS, achieving pain-free remission can be challenging. Barriers to developing a long-term treatment regimen include intolerable side effects or simply nonresponsive disease.[36,43]

Management of Perioperative Pain—Medical treatment of HS often yields only transient or mild results. Hurley stage II or III lesions typically require surgical removal of affected tissues.[32,44-46] Surgery may dramatically reduce the primary disease burden and provide substantial pain relief.[3,4,44] Complete resection of the affected tissue by wide excision is the most common surgical procedure used.[46-48] However, various tissue-sparing techniques, such as skin-tissue-sparing excision with electrosurgical peeling, also have been utilized. Tissue-sparing surgical techniques may lead to shorter healing times and less postoperative pain.[48]

There currently is little guidance available on the perioperative management of pain as it relates to surgical procedures for HS. The pain experienced from surgery varies based on the area and location of affected tissue; extent of disease; surgical technique used; and whether primary closure, closure by secondary intention, or skin grafting is utilized.[47,49] Medical treatment aimed at reducing inflammation prior to surgical intervention may improve postoperative pain and complications.

The use of general vs local anesthesia during surgery depends on the extent of the disease and the amount of tissue being removed; however, the use of local anesthesia has been associated with a higher recurrence of disease, possibly owing to less aggressive tissue removal.[50] Intraoperatively, the injection of 0.5% bupivacaine around the wound edges may lead to less postoperative pain.[3,48] Postoperative pain usually is managed with acetaminophen and NSAIDs.[48] In cases of severe postoperative pain, short- and long-acting opioid oxycodone preparations may be used. The combination of diclofenac and tramadol also has been used postoperatively.[3] Patients who do not undergo extensive surgery often can leave the hospital the same day.

Effective strategies for mitigating HS-associated pain must address the chronic pain component of the disease. Long-term management involves lifestyle modifications and pharmacologic agents.

Chronic Pain Management

Although HS is not a curable disease, there are treatments available to minimize symptoms. Long-term management of HS is essential to minimize the effects of chronic pain and physical scarring associated with inflammation.[31] In one study from the French Society of Dermatology, pain reported by patients with HS was directly associated with severity and duration of disease, emotional symptoms, and reduced functionality.[51] For these reasons, many treatments for HS target reducing clinical severity and achieving remission, often defined as more than 6 months without any recurrence of lesions.[52] In addition to lifestyle management, therapies available to manage HS include topical and systemic medications as well as procedures such as surgical excision.[36,43,52,53]

Lifestyle Modifications

Regardless of the severity of HS, all patients may benefit from basic education on the pathogenesis of the disease.[36]

The associations with smoking and obesity have been well documented, and treatment of these comorbid conditions is indicated.[36,43,52] For example, in relation to obesity, the use of metformin is very well tolerated and seems to positively impact HS symptoms.[43] Several studies have suggested that weight reduction lowers disease severity.[28-30] Patients should be counseled on the importance of smoking cessation and weight loss.

Finally, the emotional impact of HS is not to be discounted, both the physical and social discomfort as well as the chronicity of the disease and frustration with treatment.[51] Chronic pain has been associated with increased rates of depression, and 43% of patients with HS specifically have been diagnosed with major depressive disorder.[7] For these reasons, clinician guidance, social support, and websites can improve patient understanding of the disease, adherence to treatment, and comorbid anxiety and depression.[52]

Topical Therapy

Topical therapy generally is limited to mild disease and is geared at decreasing inflammation or superimposed infection.[36,52] Some of the earliest therapies used were topical antibiotics.[43] Topical clindamycin has been shown to be as effective as oral tetracyclines in reducing the number of abscesses, but neither treatment substantially reduces pain associated with smaller nodules.[54] Intralesional corticosteroids such as triamcinolone acetonide have been shown to decrease both patient-reported pain and physician-assessed severity within 1 to 7 days.[42] Routine injection, however, is not a feasible means of long-term treatment both because of inconvenience and the potential adverse effects of corticosteroids.[36,52] Both topical clindamycin and intralesional steroids are helpful in reducing inflammation prior to planned surgical intervention.[36,52,53]

Newer topical therapies include resorcinol peels and combination antimicrobials, such as 2% triclosan and oral zinc gluconate.[52,53] Data surrounding the use of resorcinol in mild to moderate HS are promising and have shown decreased severity of both new and long-standing nodules. Fifteen-percent resorcinol peels are helpful tools that allow for self-administration by patients during exacerbations to decrease pain and flare duration.[55,56] In a 2016 clinical trial, a combination of oral zinc gluconate with topical triclosan was shown to reduce flare-ups and nodules in mild HS.[57] Oral zinc alone may have anti-inflammatory properties and generally is well tolerated.[43,53] Topical therapies have a role in reducing HS-associated pain but often are limited to milder disease.

Systemic Agents

Several therapeutic options exist for the treatment of HS; however, a detailed description of their mechanisms and efficacies is beyond the scope of this review, which is focused on pain. Briefly, these systemic agents include antibiotics, retinoids, corticosteroids, antiandrogens, and biologics.[43,52,53]

Treatment with antibiotics such as tetracyclines or a combination of clindamycin plus rifampin has been shown to produce complete remission in 60% to 80% of users; however, this treatment requires more than 6 months of antibiotic therapy, which can be difficult to tolerate.[52,53,58] Relapse is common after antibiotic cessation.[2,43,52] Antibiotics have demonstrated efficacy during acute flares and in reducing inflammatory activity prior to surgery.[52]

Retinoids have been utilized in the treatment of HS because of their action on sebaceous glands and hair follicles.[43,53] Acitretin has been shown to be the most effective oral retinoid available in the United States.[43] Unfortunately, many of the studies investigating the use of retinoids for treatment of HS are limited by small sample size.[36,43,52]

Because HS is predominantly an inflammatory condition, immunosuppressants have been adapted to manage patients when antibiotics and topicals have failed. Systemic steroids rarely are used for long-term therapy because of the severe side effects and are preferred only for acute management.[36,52] Cyclosporine and dapsone have demonstrated efficacy in treating moderate to severe HS, whereas methotrexate and colchicine have shown little efficacy.[52] Both cyclosporine and dapsone are difficult to tolerate, require laboratory monitoring, and lead to only conservative improvement rather than remission in most patients.[43]

Immune dysregulation in HS involves elevated levels of proinflammatory cytokines such as tumor necrosis factor α (TNF-α), which is a key mediator of inflammation and a stimulator of other inflammatory cytokines.[59,60] The first approved biologic treatment of HS was adalimumab, a TNF-α inhibitor, which showed a 50% reduction in total abscess and inflammatory nodule count in 60% of patients with moderate to severe HS.[61-63] Of course, TNF-α inhibitor therapy is not without risks, specifically those of infection.[43,53,61,62] Maintenance therapy may be required if patients relapse.[53,61]

Various interleukin inhibitors also have emerged as potential therapies for HS, such as ustekinumab and anakinra.[36,64] Both have been subject to numerous small case trials that have reported improvements in clinical severity and pain; however, both drugs were associated with a fair number of nonresponders.[36,64,65]

Surgical Procedures

Although HS lesions may regress on their own in a matter of weeks, surgical drainage allows an acute alleviation of the severe burning pain associated with HS flares.[36,52,53] Because of improved understanding of the disease pathophysiology, recent therapies targeting the hair follicle have been developed and have shown promising results. These therapies include laser- and light-based procedures. Long-pulsed Nd:YAG laser therapy reduces the number of hair follicles and sebaceous glands and has been effective for Hurley stage I or II disease.[36,43,52,53,66]

Photodynamic therapy offers a less-invasive option compared to surgery and laser therapy.[52,53,66] Both Nd:YAG and CO_2 laser therapy offer low recurrence rates (<30%) due to destruction of the apocrine unit.[43,53] Photodynamic therapy for mild disease offers a less-invasive option compared to surgery and laser therapy.[53] There is a need for larger randomized controlled trials involving laser, light, and CO_2 therapies.[66]

Conclusion

Hidradenitis suppurativa is a debilitating condition with an underestimated disease burden. Although the pathophysiology of the disease is not completely understood, it is evident that pain is a major cause of morbidity. Patients experience a multitude of acute and chronic pain types: inflammatory, noninflammatory, nociceptive, neuropathic, and ischemic. Pain perception and quality of life are further impacted by psychiatric conditions such as depression and anxiety, both of which are common comorbidities in patients with HS. Several pharmacologic agents have been used to treat HS-associated pain with mixed results. First-line treatment of acute pain episodes includes oral acetaminophen, NSAIDs, and topical analgesics. Management of chronic pain includes utilization of topical agents, systemic agents, and biologics, as well as addressing lifestyle (eg, obesity, smoking status) and psychiatric comorbidities. Although these therapies have roles in HS pain management, the most effective pain remedies developed thus far are limited to surgery and TNF-α inhibitors. Optimization of pain control in patients with HS requires multidisciplinary collaboration among dermatologists, pain specialists, psychiatrists, and other members of the health care team. Further large-scale studies are needed to create an evidence-based treatment algorithm for the management of pain in HS.

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APPENDIX

eTABLE. Suggested Algorithm for the Treatment of Pain in Hidradenitis Suppurativa

Pain Severity		
Mild localized/mild disseminated	**Moderate**	**Severe**
Lifestyle		
Weight loss, smoking cessation, zinc gluconate, metformin		
Pain management		
Loose clothing; ice packets; topical analgesics; scheduled NSAIDs or acetaminophen; refractory: opioids or anticonvulsants		
Psychosocial		
Disease education, referral to support groups, consider treatment for anxiety		
Procedures		
Localized and recurrent abscesses: drainage, excision, CO_2 or long-pulsed Nd:YAG laser ablation	Sinus tracts: deroofing, STEEP, excision, CO_2 or long-pulsed Nd:YAG laser ablation	Radial wide excision
Topical medications		
Management of new lesions, flares, and preoperative inflammation: clindamycin, resorcinol peels, 2% triclosan; intralesional steroids; botulinum toxin		
Systemic medications		
Antibiotics (12-wk course): tetracycline, doxycycline; refractory: clindamycin and rifampin; antiandrogens: spironolactone, finasteride[a]	Antibiotics; TNF-α inhibitors: adalimumab, infliximab; retinoids: acitretin	Antibiotics; TNF-α inhibitors; retinoids; immune suppressants: cyclosporine, prednisone course

Abbreviations: NSAIDs, nonsteroidal anti-inflammatory drugs; STEEP, skin tissue-sparing excision with electrosurgical peeling; TNF, tumor necrosis factor.

[a]Topical therapy may be adequate for localized disease.

Adapted from Saunte and Jemec.[36]