



Review

A concise clinician's guide to therapy for hidradenitis suppurativa[☆]Emily Nesbitt, BA^{*}, Stephanie Clements, MD, Marcia Driscoll, MD, PharmD

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ARTICLE INFO

Article history:

Received 23 September 2019

Received in revised form 11 November 2019

Accepted 14 November 2019

Keywords:

Hidradenitis suppurativa

Treatment in women and pregnancy

ABSTRACT

Hidradenitis suppurativa (HS) is a chronic, often debilitating, skin condition that historically does not respond well to treatment. Although there is no cure for HS, symptoms can be managed if the appropriate diagnosis is made. HS most commonly develops in postpubertal women and manifests as painful, deep-seated, inflamed lesions, including nodules, sinus tracts, and abscesses. HS flares are marked by increased pain and suppuration at varying intervals and can occur in women before menstruation. HS is commonly misdiagnosed; physicians might mistake a lesion for an infection, abscess, or sexually transmitted infection. Incision and drainage of these lesions often leads to recurrence. Given that management of this chronic disease is often difficult, we sought to outline current diagnosis and management strategies for HS.

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Background

HS is a chronic, recurrent, inflammatory disease that affects skin that bears apocrine glands, most commonly the axilla, inguinal, genital, perineal, and inframammary regions. The estimated prevalence of HS is 1% to 4% in the U.S. population (Vinkel and Thomsen, 2018). HS is more prevalent in women, with a female:male ratio of 3.6:1 (Vinkel and Thomsen, 2018). Approximately one-third of patients with HS report a family history of the disease. Other predisposing factors include smoking and obesity/metabolic syndrome (Simonart, 2010; Vinkel and Thomsen, 2018).

[☆] No human subjects were included in this study. No animals were used in this study.

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Three main clinical features that support a diagnosis of HS include the typical lesions (multiple deep-seated, inflamed nodules or sinus tracts) in the typical locations (axilla, inguinal, genital, perineal, inframammary, often bilateral). The third feature is the chronicity and relapsing nature of the lesions (Lee and Eisen, 2015). The goal of treatment is to prevent the formation of new lesions and to manage the symptoms (most commonly pain and suppuration) of current lesions.

The approach to first-line treatment of HS depends on the staging of the disease. The most commonly used staging system is the Hurley Clinical Staging System (Hurley, 1989). Stage I consists of abscess formation (single or multiple) without sinus tracts and scarring. Stage II includes recurrent abscesses with sinus tracts and scarring. Finally, a patient with Stage III HS exhibits diffuse areas of involvement or multiple interconnected sinus tracts and

abscesses across the entire area (Hurley, 1989; Lee and Eisen, 2015).

Treatment for all stages involves prevention of lesion formation, treatment of existing lesions before they develop into chronic sinus tracts, and elimination of existing nodules and sinus tracts before extensive scarring occurs (Lockwood, 2017). However, treatment regimens are tailored by the stage of the disease. Regardless of the stage, patients should be counseled on weight loss and smoking cessation if they are overweight or smoke, because obesity and cigarette smoking are strongly associated with HS. Patients should also be advised to avoid tight-fitting clothing and excessive friction to the involved areas.

Medical therapy

Medical treatment of HS has proven to be historically difficult due to a lack of pathophysiologic insight, but patients' symptoms can often be managed with medical therapies alone. Treatment is determined based on the Hurley staging system, with topical therapies used as first-line therapy for less invasive disease and systemic antibiotics or biologics, surgery, and light therapy reserved for more extensive disease.

Stage I

Topical clindamycin is often the first-line therapy for mild HS, with evidence from multiple trials supporting its efficacy, relative safety, and tolerability. Patients may experience a slight burning sensation when the antibiotic is applied to lesions. A randomized 3-month trial conducted by Clemmensen (1983) supported the efficacy and tolerability of topical clindamycin 1% solution for inflammatory abscesses. The mechanism of clindamycin in the treatment of HS appears to be associated with the drug's anti-inflammatory properties. Thirty patients with recurrent HS were enrolled in a double-blind trial to determine the effect of clindamycin versus placebo. Patient assessment, numbers of abscesses, inflammatory nodules, and pustules were the outcomes measured. For each parameter, clindamycin 1% solution was significantly superior to placebo ($p < .01$; Clemmensen, 1983).

Intralesional corticosteroids, such as triamcinolone 10 mg/mL, can be useful as an adjunct to reduce the symptoms of an early, painful lesion. Corticosteroids locally bind to the glucocorticoid receptor to reduce inflammation, rubor, and pain. A case series of 36 patients conducted by Riis et al. (2016) demonstrated that intralesional corticosteroids decreased erythema, edema, suppuration, and patient-reported pain ($p < .0001$).

Punch debridement of a newly inflamed nodule can be effective in eliminating a new lesion and preventing progression into an abscess or sinus tract (Danby et al., 2015). Punch debridement should be considered for only early or small acute or subacute inflammatory lesions, often involving one folliculopilosebaceous unit (Danby et al., 2015).

Finally, patients with Stage I HS may benefit from treatment with topical resorcinol, a chemical peeling agent with anti-inflammatory and keratolytic properties. According to Pascual et al. (2017), topical 15% resorcinol was associated with reductions in pain and size in both acute and long-standing lesions. Ultrasonographic follow-up was used in the study and showed that clinical resolution occurred more quickly than ultrasonographic resolution; therefore, the authors recommended continuing the use of topical resorcinol for several weeks after apparent clinical resolution (Pascual et al., 2017).

Stage II

Patients with more invasive HS may benefit from systemic antibiotics. First-line treatment is oral tetracyclines: 100 mg doxycycline once or twice daily, 100 mg minocycline once or twice daily, or tetracycline 500 mg twice daily. Systemic antibiotics are the medications most often prescribed for patients with HS and have been shown to be the most effective traditional therapy. Tetracyclines have been shown to suppress lymphocytes, neutrophils, and histiocytes and are therefore used for their anti-inflammatory properties (Alhusayen and Shear, 2015). Patients who are prescribed doxycycline should be advised to use plenty of sunscreen and wear sun protective hats and clothing because doxycycline sensitizes the skin to sun (Frost et al., 1972).

Patients who do not respond to oral tetracyclines may try the combination of clindamycin (300 mg twice daily) and rifampin (600 mg once daily). A 2009 retrospective study of 116 patients with HS who were treated with this regimen determined the efficacy on HS lesions (Gener et al., 2009). The main outcome measure was disease severity, assessed with the Sartorius score (a disease severity assessment tool) before and after 10 weeks of treatment. The Sartorius score is composed by counting involved regions, nodules, and sinus tracts. Three points are allotted per region involved, two points per nodule, four points per fistula, one point for scar, and one point each for other. Additionally, the longest distance between two relevant lesions <5 cm is awarded two points, <10 cm four points, and >10 cm eight points. If lesions are clearly separated by normal skin in each region, zero points are awarded; if not, six points are given (Table 1).

Results showed a dramatic improvement in the Sartorius score at the end of treatment ($p < .001$) (Gener et al., 2009). Although the treatment is effective, the mechanism of action of these drugs on improving HS lesions is not understood and needs further elucidation. A recent randomized control study by Caposiena Caro et al. (2019) demonstrated that oral clindamycin alone may be effective in improving HS lesions, although physicians need to be aware of the risk of pseudomembranous colitis associated with clindamycin. Rifampin may not be a necessary antibiotic, but further studies should be conducted to determine which regimen is superior. Rifampin induces the cytochrome p450 system, so a thorough medication review with the patient should be performed to ensure no drug–drug interactions.

Dapsone, an antineutrophilic and antieosinophilic antibiotic, can be effective in mild-to-moderate HS as a monotherapy. In a retrospective review of 24 patients with HS, 25% achieved significant improvement and 12.5% experienced slight improvement in their disease after treatment with dapsone (Yazdanyar et al., 2011). The drug is also relatively safe if monitored appropriately. Importantly, patients should be tested for G6PD deficiency before beginning therapy with dapsone because hemolysis is a well-known adverse effect and more likely to occur in G6PD deficient individuals (Alhusayen and Shear, 2015).

Oral retinoids may also be used for patients with stage II HS. Acitretin has demonstrated the most efficacy. Its mechanism of action is thought to include the normalization of epithelial cells through interaction with the retinoic acid receptor. According to a prospective study of 17 patients with HS, symptom and lesion improvement occurred after 2 months of treatment and persisted for several months (Matusiak et al., 2014). The mean acitretin dose was 0.56 ± 0.08 mg/kg/day. The main reasons patients decided to discontinue treatment with retinoids include intolerable side effects (redness, itching and scaling of skin, dry skin) and treatment inefficacy (Matusiak et al., 2014). Isotretinoin has typically not been effective in patients with HS (Soria et al., 2009). Retinoids are teratogenic, so their use in women of childbearing age must be

Table 1
Treatment of hidradenitis suppurativa based on Hurley staging.

	Medication/regimen	Comments/precautions
Hurley stage I	Topical clindamycin 1% BID during flares, qd for maintenance Intralesional corticosteroids (triamcinolone 10 mg/mL)	Well tolerated Atrophy, skin hypopigmentation can occur, sterile abscess formation less frequent
	Topical 15% resorcinol BID during flares, qd for maintenance Punch debridement of newly inflamed nodule	Irritant contact dermatitis common Should be performed only on small, newly inflamed nodules without sinus tracts; recurrence is common in incised nodules
Hurley stage II	Oral antibiotics: Doxycycline 100 mg qd or BID Minocycline 100 mg qd or BID Tetracycline 500 mg BID Clindamycin 300 mg BID + rifampin 600 mg qd	Patients taking doxycycline should be advised to wear sunscreen and sun protective clothing because of photosensitization; other side effects include nausea, pseudotumor cerebri, and tissue hyperpigmentation
	Dapsone 50–200 mg qd Acitretin 0.56 ± 0.08 mg/kg qd	Clindamycin carries the risk of pseudomembranous colitis. Rifampin induces cytochrome p450, can cause red urine and nausea. Patients with G6PD deficiency can develop hemolytic anemia. Contraindicated in pregnancy; redness, itching, and dry skin common; can also cause elevated triglycerides
	Spironolactone 100 mg qd	Contraindicated in pregnancy. Gynecomastia is common in men.
	Hurley stage III	Adalimumab 40 mg weekly
	Infliximab 5 mg/kg at weeks 0, 2, and 6	Risk of infection (must test for latent tuberculosis and hepatitis before use); headache, nausea, increased alanine aminotransferase common
	Prednisone 40–60 mg for 3–4 days with a 7–10 day taper	Should only be considered in severe inflammatory cases due to unpleasant side effects and risk of infection
	Ustekinumab (45–90 mg at weeks 0, 4, 16, and 28)	Risk of infection
	Anakinra 100 mg qd	Risk of infection; headache, vomiting, and infection site reaction common

BID, twice daily; qd, one a day.

closely monitored. Acitretin must be discontinued >2 years before a woman attempts conception (Matusiak et al., 2014).

Increased androgens are thought to play a role in the development of HS lesions, so hormonal therapy can be useful in patients with mild-to-moderate HS (Kraft and Searles, 2007). Cyproterone acetate, an antiandrogen and progestin medication, has been shown to be effective in women with HS. It can be used by itself or in combination with oral contraceptive pills. In a randomized, double-blind crossover study, 24 women were assigned to take ethinylestradiol 50 µg/cyproterone acetate 50 mg or ethinylestradiol 50 µg/norgestrel 500 µg, and both treatments produced substantial improvement in disease (Mortimer et al., 1986).

Spironolactone 100 mg daily is an antiandrogen and may be used alone or in combination with cyproterone acetate or oral contraceptive pills for the improvement and prevention of HS lesions (Kraft and Searles, 2007). A retrospective chart review published in January 2019 showed that patients taking spironolactone achieved significant disease improvement with regard to pain, inflammatory lesions, and HS Physician's Global Assessment score. No change was found for Hurley stage or fistulas, and there was no difference in improvement between patients who received <75 mg of spironolactone daily and those who received >100 mg daily (Golbari et al., 2019). Hormonal therapy is contraindicated in pregnant women due to adverse effects on the fetus.

Surgical removal of lesions is a final, definitive treatment option for patients with Stage II or III disease, especially those with extensive, recurrent HS lesions. Five surgical approaches can be considered: local destruction via cryosurgery, cryoinfusion, electrosurgery, and photodynamic therapy; incision and drainage; standard, wide unroofing and debridement of individual sinus tracts; or complete surgical excision beyond all clinically apparent margins with either complete closure or partial thickness skin graft (Danby et al., 2015). Each surgical option has pros and cons, and treatment decisions should be tailored to the individual patient. Incision and drainage may provide temporary relief, but it is generally not advised due to frequent recurrence of cysts after the procedure.

Very wide unroofing and debridement of individual sinus tracts allows for healing by secondary intention and is a definitive treat-

ment for a symptomatic area, but it does not prevent new lesions or decrease inflammation. Complete excision is an effective, definitive treatment but may have negative cosmetic results. Of these techniques, a prospective study conducted by Menderes et al. (2010) found that conservative treatment methods, such as punch debridement and standard unroofing, had little or no effect, especially on gluteal, perineal/perianal, and axillary HS. The only successful surgical treatment was wide surgical excision (Menderes et al., 2010).

Stage III

Patients with refractory disease not responsive to oral antibiotics, oral retinoids, or hormonal therapy may benefit from tumor necrosis factor (TNF)-alpha inhibitors. Adalimumab and infliximab have both been shown to be helpful in reducing symptoms of current lesions and recurrence of new lesions. Adalimumab is approved by the U.S. Food and Drug Administration (FDA) for the treatment of HS based on the PIONEER I and II trials. Pioneer I and II were double-blind, placebo-controlled studies in which 307 patients received either 40 mg of adalimumab weekly or matching placebo for 12 weeks. The primary end point was clinical response, defined as at least a 50% reduction from baseline in the abscess and inflammatory-nodule count, with no increase in abscess or draining fistula counts at week 12. Clinical response rates were significantly higher for the groups receiving adalimumab weekly than for the placebo groups: 58.9% vs. 27.6% in PIONEER II ($p < .001$; Kimball et al., 2016).

Adalimumab is also FDA-approved for stage II (moderate) HS. Although infliximab has not been FDA-approved for the treatment of HS, it is often used off-label to achieve rapid control of severe disease (Grant et al., 2010). Although adalimumab is the only FDA approved biologic therapy for HS, a retrospective study was performed in 2012 to compare two cohorts of 10 adult patients with severe HS. Ten patients were treated with infliximab (three infusions of 5 mg/kg at weeks 0, 2, and 6), and 10 other patients were treated with 40 mg of adalimumab every other week. In both groups, HS severity decreased, but infliximab performed better in all aspects (Sartorius score, quality of life index; van Rappard

et al., 2012). Further research on the efficacy of adalimumab versus infliximab could help elucidate the results of this study.

Etanercept, another TNF-alpha inhibitor, has not proven to be useful in the treatment of HS (Adams et al., 2010). TNF-alpha inhibitors must be used for long-term management, and patients must be advised that the disease will likely relapse if TNF-alpha inhibitors are stopped. Acute severe flares of HS can be managed with a 3- to 4-day course of prednisone, 40 to 60 mg per day tapered over the subsequent 7 to 10 days (Nazary et al., 2011).

Emerging therapies for Hurley stage III HS include the IL-12/23 receptor antagonist ustekinumab, IL-1 receptor antagonists anakinra and canakinumab, and IL-1 alpha inhibitor MABp1. All of these biologic therapies have been reported to be helpful in the treatment of severe or refractory HS, although relapse is common after stopping treatment. A study by Blok et al. (2016) evaluated the use of ustekinumab (45 or 90 mg at weeks 0, 4, 16, and 28) in 17 patients with severe HS. Of those 17 patients, 12 completed the protocol, and 82% of these patients experienced a moderate-to-marked improvement in their disease (Blok et al., 2016).

A randomized clinical trial was performed to determine the efficacy of anakinra in treating severe HS. Twenty patients were recruited; 10 patients received placebo and the remaining 10 patients received anakinra. Seventy percent of patients in the anakinra arm showed improvement, whereas only 20% of patients in the placebo group showed improvement after 24 weeks. Extensive studies evaluating the efficacy of these biologics are limited, but as these drugs become more accessible in the future, more information on their efficacy will likely become available (Tzanetakou et al., 2016).

If multiple medical therapies have failed, patients with Hurley stage III lesions should be referred to plastic surgery or general surgery for excision of lesions. As noted earlier, the best outcomes follow wide surgical excision of the lesions.

Special considerations for hidradenitis suppurativa in pregnancy

Many women experience no relief or even clinical deterioration of their disease during pregnancy; therefore, considering possible therapeutic options for these women is important. Topical antibiotics, such as clindamycin, are safe to use during pregnancy and can be applied to lesions twice daily. Oral tetracyclines (pregnancy category D) are contraindicated in pregnant and lactating women; use of tetracyclines can lead to dental staining and enamel hypoplasia in the developing fetus (Vennila et al., 2014).

Clindamycin is considered safe during pregnancy and lactation (category B drug), and rifampin is a pregnancy category C drug. Observational studies have reported no excessive birth defects in babies of >2000 mothers who took rifampin during pregnancy (Snider, 1984). Therefore, a regimen of 600 mg clindamycin and 600 mg rifampin daily is a reasonable option for some women with moderate-to-severe HS. Dapsone is another pregnancy category C drug, and no causal relationship has been found between dapsone use and birth defects. Therefore, a dose ranging from 50 to 200 mg daily might be another reasonable option for pregnant women with moderate-to-severe HS (Perng et al., 2017).

The safety and efficacy of biologic therapy for the treatment of HS in pregnant women is unclear and controversial. Adalimumab and infliximab appear to be the safest biologic treatments for women with no other option because there have been no increased risks of adverse birth outcomes to date (Vinet et al., 2009; Yarur and Kane, 2013). Because the human placenta is most permeable to maternal IgG antibodies during the third trimester, it is recommended that biologic therapy be stopped in the third trimester to avoid placental transfer (Androulakis et al., 2015). The safety of

Table 2
Treatment of hidradenitis suppurativa IN pregnancy.

	Medication	Comments/precautions
Safe IN pregnancy	Topical clindamycin 1% BID	
	Clindamycin 300 mg BID + rifampin 600 mg qd	Clindamycin is a pregnancy category B drug and considered safe in pregnancy; rifampin is pregnancy class C and has not been associated with increased birth defects (evidence is limited)
	Dapsone 50–200 mg qd	Presumed safe in pregnancy (evidence is limited)
	Adalimumab 40 mg qd	No increased risk of adverse birth outcomes
Contraindicated in pregnancy	Infliximab 5 mg/kg at weeks 0, 2, and 6	No increased risk of adverse birth outcomes
	Oral tetracyclines	Pregnancy class D; can cause dental staining and enamel hypoplasia in developing fetus
	Spirolactone	Antiandrogen effects can cause feminization of a male fetus
	Retinoids	Absolutely contraindicated in pregnancy due to severe birth defects
	Surgical management	Although not completely contraindicated, surgical management of lesions should be addressed after pregnancy.

BID, twice daily; qd, one a day.

other biologics (e.g., etanercept, ustekinumab, and anakinra) during pregnancy is unclear.

Hormone-based and retinoid therapies are contraindicated in pregnant women. Furthermore, surgical procedures should be avoided whenever possible during pregnancy (Table 2).

Emerging therapies

Laser and light therapy have been used in recent years as adjunctive therapy for HS lesions. Laser and light therapy work to reduce the occurrence of painful HS flare-ups by decreasing the number of hair follicles, sebaceous glands, and bacteria in affected areas and by ablatively debulking chronic lesions (Hamzavi et al., 2015). In a study conducted by Hamzavi et al. (2015), the severity of the patient's disease determined the laser/light therapy they received. Those with less extensive disease (Hurley stage I and II) benefitted from hair follicle and bacterial load reduction with Nd:YAG laser and photodynamic therapy. Those with more advanced disease (advanced Hurley stage II or III) demonstrated a better response with CO₂ laser vaporization and excision of sinus tracts. Both therapies appear to be effective with low complication rates. The most common side effect of laser/light therapy is pain in the treated area (Hamzavi et al., 2015). A retrospective study by Mikkelsen et al. (2015) reported that patients felt their lesions greatly improved and 91% would recommend laser surgery to other patients with HS.

Metformin is an emerging therapy that may be helpful in the treatment of HS. The exact mechanism of action in the treatment of HS is currently unknown, although it has been proposed that metformin acts via an antiandrogenic mechanism to improve HS. The efficacy of treatment with metformin has been demonstrated in multiple studies, with Verdolini et al. (2013) completing the pilot study. The recommended starting dose is 500 mg once daily with the maximum dose at 500 mg TID. Minimal side effects are typically experienced by patients, and the most significant side effect is nausea (Verdolini et al., 2013). Diarrhea is also a common side effect in patients taking metformin. Metformin appears to be

an excellent alternative to high-dose, long-term antibiotics for the treatment of HS.

In a pilot study by Brocard et al. (2007), patients with Hurley stage I or II HS benefitted from treatment with zinc salts. All patients received 90 mg of zinc gluconate per day and noted a clinical response. Many saw partial remission of the lesions, and approximately one quarter of patients experienced complete remission. Patients did tend to relapse after tapering to <60 mg per day, and a small percentage of patients experienced gastrointestinal side effects from the medication (Brocard et al., 2007). Zinc salts have anti-inflammatory and antioxidant properties, so zinc cannot cure the condition but instead can stop HS from progressing and prevent flares. More research needs to be conducted on their efficacy, but these agents appear to be helpful supplemental medical therapies in the treatment of HS.

Conflict of interest

None.

Funding

None.

Study Approval

N.A.

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