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Research Advances in the Treatment of Hidradenitis Suppurativa

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Abstract

Hidradenitis suppurativa (HS) is a refractory and recurrent chronic inflammatory skin disease characterized by nodules, abscesses, sinus tracts, and scarring. The etiology of HS remains unclear, rendering its treatment particularly challenging. Current therapeutic approaches encompass lifestyle modifications, topical medications, conventional systemic drugs, biologic agents, surgical interventions, and traditional Chinese medicine (TCM). A personalized treatment strategy tailored to individual patient characteristics is essential to achieve optimal clinical outcomes.

Keywords

Hidradenitis Suppurativa, Pharmacotherapy, Surgical Treatment, Physical Therapy

1. Introduction

Hidradenitis suppurativa (HS), also termed acne inversa, is a chronic inflammatory dermatosis characterized by painful inflammatory lesions predominantly affecting apocrine gland-rich regions, including the groin, axillae, inframammary folds, and anogenital areas [1]. The disease typically manifests between puberty and 40 years of age, with an estimated global prevalence ranging from 1% to 4%. While more prevalent in women, male patients often present with more severe clinical manifestations. The clinical spectrum encompasses mild inflammatory nodules to extensive abscesses, sinus tracts, and scarring in intertriginous regions [1]. The pathogenesis of HS remains incompletely elucidated; however, genetic predisposition, obesity, immune dysregulation, and environmental triggers such as smoking have been implicated as pivotal contributors to both disease initiation and per-

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sistence [2]. In the genetic context, γ-secretase gene mutations constitute a genetic determinant in familial HS [3], where impaired EGFR signaling drives pathological keratinocyte hyperproliferation and follicular hyperkeratosis—key components of HS pathogenesis [4]. Immunologically, elevated concentrations of TNF-α and IL-17 in lesional tissue implicate dysregulated innate immune responses in perpetuating the inflammatory cascade [5] [6]. Obesity exacerbates disease progression through mechanical friction in skin folds, perspiration retention, and adipokine-mediated inflammatory amplification [7]. Cigarette-derived nicotine and benzo [a] pyrene may synergistically promote epidermal hyperplasia, follicular plugging, and pro-inflammatory pathways, thereby contributing to HS pathophysiological progression [8]. China currently lacks standardized diagnostic criteria for HS, with clinical diagnosis primarily relying on comprehensive evaluation of medical history, characteristic clinical manifestations, and family history. This review synthesizes contemporary advances in HS therapeutics to inform evidence-based clinical management strategies.

2. Disease Severity Classification

Disease severity is commonly assessed using various scoring systems, including the Hurley staging system, sartorius staging system, the International Hidradenitis Suppurativa Severity Score System (IHS4), the Hidradenitis Suppurativa Severity Index (HSSI), the Dermatology Life Quality Index (DLQI), and the Visual Analog Scale (VAS) [9]. Among these, the Hurley staging system is the most widely used and classifies HS into three grades.

Hurley Stage I (Mild): Single or multiple nodules/abscesses without sinus tracts or scar. Hurley Stage II (Moderate): Recurrent nodules/abscesses with sinus tract formation and scar. Hurley Stage III (Severe): Diffuse or interconnected lesions with extensive sinus tracts and abscesses [9].

3. Treatment Strategies

3.1. General Measures

Lifestyle modifications, including smoking cessation, dietary adjustments, weight loss, and supplementation with zinc and vitamin D, have demonstrated efficacy in improving prognosis and reducing disease recurrence [10]-[13]. Additionally, maintaining proper skin hygiene—using mild cleansers, avoiding irritants, and wearing loose, breathable clothing—can minimize friction and secondary irritation in affected areas [2].

3.2. Pharmacological Therapies

3.2.1. Topical and Intralesional Agents

Topical treatments serve as first-line options for mild presentations or adjunctive therapy in moderate-to-severe cases:

- Antimicrobial agents (1% clindamycin solution, mupirocin ointment, fusidic acid cream): Reduce bacterial colonization and inflammatory responses in

pustular lesions [14].

- Keratolytics (topical retinoids, 15% resorcinol ointment): Alleviate follicular hyperkeratosis and ostial occlusion [14].
- Intralesional corticosteroids: Provide rapid symptomatic relief for acute inflammatory lesions, though contraindicated in sinus tracts with confirmed bacterial colonization [13].

3.2.2. Systemic Pharmacotherapy

(1) Antibiotic Therapy

Tetracyclines: Tetracycline-class antibiotics (tetracycline, doxycycline, minocycline) demonstrate comparable efficacy to topical clindamycin in ameliorating HS symptoms. A randomized trial involving 46 Hurley stage I/II patients receiving oral tetracyclines (100 mg doxycycline 1-2×/day, 100 mg minocycline 1-2×/day, or 500 mg tetracycline 2×/day) for ≥3 months showed clinical improvement equivalent to topical clindamycin [15]. Beyond antibacterial effects, tetracyclines exert anti-inflammatory properties beneficial for HS management [16].

Fusidic Acid (FA): A retrospective analysis by Wiala *et al.* evaluating 55 FA treatment cycles (45.5% male, 54.5% female) reported an overall response rate of 70.9% (39 cycles), with adverse events (21.8%) primarily involving gastrointestinal symptoms (27.3%, n = 15), 46.7% occurring within 4 weeks. Comparable efficacy was observed versus doxycycline controls (76.4% response rate) [17].

Moxifloxacin: This fluoroquinolone exhibits dual antimicrobial and immunomodulatory activity via interleukin-1α and TNF-α inhibition [18]. A retrospective cohort study (2021-2023) of 39 HS adults receiving 400 mg/day moxifloxacin demonstrated 20.5% excellent response, 51.3% partial response, and 28.2% non-response. Concurrent therapies included adalimumab (10.3%), metformin (12.8%), spironolactone (2.6%), and ertapenem (2.6%) [19].

Trimethoprim-Sulfamethoxazole (TMP-SMX): Combining antimicrobial and immunomodulatory actions [20], a retrospective analysis of 18 Hurley I-III patients receiving TMP-SMX monotherapy achieved excellent (50%) and partial (38.9%) response, with only one adverse event (non-pruritic rash) reported. Notably, many subjects had prior inadequate responses to rifampicin-clindamycin or doxycycline [21].

Clindamycin-Rifampicin Combination: This regimen provides broad-spectrum coverage against Gram-positive/negative bacteria, anaerobes, biofilms, and granulomas. Clindamycin targets inflammatory nodules, while rifampicin enhances penetr [22].

Piperacillin-Tazobactam: Evaluated in severe HS, a retrospective study of 10 patients receiving 6 - 21 days therapy showed 80% achieving ≥2-grade improvement on HS-Physician Global Assessment. At 3- and 6-month follow-up, 66.7% and 55.6% maintained Hidradenitis Suppurativa Clinical Response (HiSCR), with no adverse events reported [23].

(2) Retinoid Therapy

Isotretinoin: This agent normalizes keratinocyte differentiation and reduces pro-

inflammatory cytokines by attenuating Th17-mediated IL-17 production. However, current evidence remains inconsistent regarding its efficacy as monotherapy or adjunctive treatment for HS, with documented risks of disease exacerbation [24].

Acitretin: Modulating keratinocyte differentiation, acitretin demonstrates anti-inflammatory effects through suppression of IL-6, migration inhibitory factor-related protein-8 (MRP-8), and interferon-γ (IFN-γ) [24]. Notably, it downregulates IL-17A-induced IL-36β/γ expression at transcriptional and translational levels [25]. While monotherapy exhibits marked variability in therapeutic outcomes, synergistic effects are observed when combined with antibiotics, surgery, or biologics.

Alitretinoin: Exhibiting antiproliferative and proapoptotic properties, alitretinoin inhibits lipopolysaccharide-induced nitric oxide synthesis and suppresses TNF-α, IL-1β, and IL-12p40 expression [26]. Current clinical evidence supporting its application in HS management remains limited.

(3) Antiandrogen Therapy

Spironolactone: Androgen signaling contributes to HS pathogenesis. A retrospective analysis of 52 female HS patients (>18 years) receiving spironolactone (mean dose 104.3 mg/day, range 50 - 200 mg/day) demonstrated improvement rates of 84.1% (37/44) at 3 months and 81.8% (27/33) at 6 months. Discontinuation occurred in 5 patients due to adverse effects (menstrual irregularities, dizziness, muscle cramps, menorrhagia, gastrointestinal distress), with one case of unspecified causality [27]. These findings position spironolactone as a viable therapeutic option, particularly as primary or adjunctive therapy for early-stage HS in reproductive-age females, though efficacy diminishes in chronic severe presentations [28].

4. Biologic Therapies

4.1. Adalimumab

As the first biologic approved for HS patients ≥12 years, this TNF-α inhibitor mitigates underlying inflammatory processes [29]. Phase III PIONEER I & II trials (N = 633 adults) demonstrated significant reductions in inflammatory nodules/abscesses versus placebo, with favorable safety. Recommended dosing: 160 mg (four 40 mg injections) on Day 1, 80 mg (two 40 mg injections) at Week 2, followed by 40 mg every other week or 80 mg (two 40 mg injections) biweekly as maintenance [30].

4.2. Secukinumab

The 2023 SUNSHINE and SUNRISE Phase III trials reported HiSCR50 achievement in 45% (vs. 34% placebo) and 42% (vs. 31% placebo) of moderate-to-severe HS patients, respectively [31]. Secukinumab demonstrated favorable efficacy and safety profiles with rapid symptom relief sustained over one year of treatment. Prior TNF-α inhibitor exposure did not compromise HiSCR attainment [31]. Ap-

proved regimen: 300 mg subcutaneously at Weeks 0, 1, 2, 3, 4 (loading phase), then every 4 weeks. Dose escalation to 300 mg every 2 weeks is permitted for suboptimal responders [32].

4.3. Ustekinumab

Targeting IL-23/IL-12 axis dysregulation linked to Notch pathway abnormalities [33]-[35], a retrospective study of 10 patients (80% adalimumab-experienced) showed 90% HiSCR50 response within 4.72 months [36]. Current clinical evidence remains limited.

4.4. Bimekizumab

This EMA-approved humanized monoclonal antibody targeting IL-17A/F demonstrated efficacy in BE HEARD I & II RCTs (N = 505 and 509 patients). At Week 16, HiSCR50 rates were 47.8% (Q2W) and 45.3% (Q4W) vs. 28.7% placebo in BE HEARD I, and 52%/53.8% vs. 32.2% in BE HEARD II [37]. Responses persisted through 48 weeks with favorable tolerability. Recommended dosing: 320 mg (two 160 mg injections) every 2 weeks until Week 16, then monthly [30].

4.5. Brodalumab

This fully human monoclonal antibody targeting IL-17 receptor A (IL-17RA) modulates inflammatory pathways in HS [38]. A study of 16 moderate-to-severe HS patients receiving brodalumab (210 mg at Weeks 0, 1, 2, then every 2 weeks) demonstrated 50% (8/16) achieving HiSCR at Week 16, with 31% experiencing mild adverse events [39]. While promising, broader clinical validation is required.

4.6. Vilobelimab

Research has demonstrated significantly elevated plasma C5a concentrations in HS patients compared to healthy individuals, highlighting its potential as a therapeutic target [40]. A prospective open-label single-arm phase IIa study evaluating Vilobelimab's safety and efficacy in HS treatment revealed that 83% of patients achieved HiSCR50, with an 83.3% response rate maintained at the 3-month follow-up [41]. This investigational agent continues to undergo clinical development.

A network meta-analysis evaluating biologic therapies for moderate-to-severe hidradenitis suppurativa (HS), incorporating 13 studies, 14 therapeutic interventions, and 2,748 participants, demonstrated significant efficacy for adalimumab (RR: 0.37, 95% CI = 0.06 – 0.63), secukinumab (RR: 0.25, 95% CI = 0.11 – 0.47), and bimekizumab (RR: 0.38, 95% CI = –0.01 – 0.89) [42]. Therefore, among the evaluated biologics for moderate-to-severe hidradenitis suppurativa, adalimumab demonstrated superior efficacy, followed by secukinumab, with bimekizumab also exhibiting promising therapeutic potential. Brodalumab and ustekinumab, how-

ever, require further clinical validation through expanded datasets to substantiate their therapeutic profiles.

4.7. Upadacitinib

As a JAK inhibitor, upadacitinib modulates JAK/STAT-mediated inflammatory cascades. A case report described a HS patient with ulcerative colitis (UC) refractory to infliximab, ustekinumab, and antibiotics who achieved sustained HS and UC remission on upadacitinib 30 mg/day for 9 months without adverse effects [43]. The phase II trial NCT04430855 (2023) showed statistically significant improvement ($p < 0.05$) in moderate-to-severe HS patients receiving 30 mg/day upadacitinib versus placebo at Week 12 [43]. Dosing protocol: 15 mg/day initially, escalating to 30 mg/day from Week 4 if inadequate response, continued for 24 weeks [44].

5. Physical Modalities

Follicular occlusion constitutes a pathogenic factor in HS, where laser-assisted hair removal (LHR) disrupts pilosebaceous units, reduces sebum production, and induces follicular degeneration.

5.1. Nd: YAG Laser (1064 nm)

This non-ablative laser selectively targets hair shafts and follicles via melanin and water chromophore absorption [45]. Recommended for Hurley stage I patients, emerging evidence supports its application in stages II-III [46].

5.2. Alexandrite Laser (755 nm)

By targeting melanin-rich structures, this modality damages follicular bulge stem cells and dermal papillae [45]. A case-control study of 15 HS patients (24 lesions: 11 axillary, 10 inguinal, 3 inframammary) demonstrated 75% HiSCR achievement at Week 24, with site-specific efficacy: 72.73% axillary, 71.43% inguinal, and 100% inframammary [45].

5.3. Intense Pulsed Light (IPL)

Utilizing broad-spectrum pulses, IPL induces follicular thermolysis while exerting anti-inflammatory effects [45]. A randomized trial (N = 18 Hurley II-III patients) showed significant HS-LASI reduction in the IPL group ($p < 0.001$), with widening efficacy divergence from controls over time ($p < 0.001$) [47]. Further safety evaluations are warranted.

5.4. Photodynamic Therapy (PDT)

This low-toxicity modality effectively controls disease progression and recurrence. A meta-analysis by Alexandra Strobe *et al.* (N = 3,437) evaluated outcomes using Hurley staging, IHS4, pain-NRS, and DLQI. At Week 26: Pain reduction: 80% Hurley I, 70.6% Hurley II, 42.8% Hurley III. DLQI improvement (≥4-point

reduction): 66.4% Hurley I, 61.3% Hurley II, 52.1% Hurley III [48].

6. Surgical Interventions

Surgical approaches constitute definitive management options across HS severity grades.

6.1. Incision and Drainage

Primarily employed for acute pain relief in fluctuant abscesses. While providing immediate symptom mitigation, this approach offers only transient benefit due to high recurrence rates [49].

6.2. Deroofing

The surgical mainstay for persistent nodules/sinus tracts in Hurley I/II disease [50]. This minimally invasive technique involves excision of lesion domes while preserving underlying fibrotic tissue and scar beds [51]. Though facilitating rapid recovery, deroofing carries elevated risks of recurrence and postoperative infection.

6.3. Wide Local Excision

Indicated for recalcitrant Hurley II/III lesions, this procedure entails complete resection of affected tissue with margins extending beyond clinical lesion boundaries, ensuring en bloc apocrine gland removal [52]. While achieving superior long-term control, extensive excisions necessitate prolonged healing periods and may result in hypertrophic scarring [51].

6.4. STEEP Technique

(Skin Tissue-preserving Excision with Electrosurgical Peeling): Combines deroofing with layered tangential electrosurgical dissection to eradicate pathologic tissue while maximizing healthy skin preservation [51]. The methodology follows:

Initial deroofing

- Tangential electrosurgical peeling parallel to skin surface;
- Progressive depth adjustment until complete fibrotic tissue removal;
- This tissue-sparing strategy promotes rapid re-epithelialization, favorable cosmesis, and reduced contracture risk [51].

6.5. Carbon Dioxide Laser

This infrared laser enables precise tissue ablation through parameter-adjusted vaporization, performing deroofing, vaporization, or localized excision contingent on lesion severity [51]. Advantages include rapid postoperative recovery and reduced scar contracture compared to conventional techniques.

7. Conclusion

Hidradenitis suppurativa (HS) is a chronic inflammatory dermatosis characterized by recurrent nodules, abscesses, and sinus tracts, predominantly affecting ap-

ocrine gland-rich regions such as axillary and inguinal areas. Its pathogenesis involves complex interactions among genetic susceptibility, immune dysregulation, and microbial factors. HS presents diverse therapeutic options requiring comprehensive implementation. All patients should adhere to foundational management including smoking cessation and weight optimization. Topical therapies (antibiotic ointments), surgical interventions, and physical modalities serve as first-line management for mild HS or adjunctive roles in moderate-to-severe disease. Systemic agents (antibiotics, biologic therapies, retinoids) may be utilized individually or in synergistic combinations to maximize therapeutic efficacy. A tiered therapeutic algorithm is recommended (Table 1). Effective management requires multidisciplinary collaboration to develop personalized regimens based on disease severity, treatment history, and clinical manifestations, thereby enhancing therapeutic adherence and outcomes. Future research should prioritize elucidating molecular pathways, refining clinical trial designs for novel therapeutics, and integrating surgical and biologic approaches to achieve sustained disease control and improved quality of life.

Table 1. HS recommended graded treatment regimens.

Hurley stage	Clinical manifestations	First-line treatment	Second-line treatment	Adjunctive therapy
Stage I (Mild)	Scattered nodules, abscesses	Topical medications or oral tetracycline-class antibiotics	Clindamycin-Rifampicin, Retinoid, Antiandrogen (female)	Incision and drainage, deroof-ing, physical modalities
Stage II (Moderate)	Scattered nodules, abscesses, sinus-tracts, scar	oral tetracycline-class antibiotics	Above second-line therapies and/or biologics	Topical medications or adjunctive therapy as above
Stage III (Severe)	Diffuse interconnected abscesses and sinus tracts	Oral moxifloxacin + rifampicin combination	Biologics, Antiandrogen (female)	Topical medications, deroofing, wide local excision, physical modalities

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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