Notable Grand Rounds

of the

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Wayne State University
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Detroit, Michigan, USA

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THE CUTTING EDGE OF TREATMENT FOR HIDRADENITIS SUPPURATIVA

February 25, 2026

About Notable Grand Rounds

These assembled papers are edited transcripts of didactic lectures given by mainly senior residents, but also some distinguished attending and guests, at the Grand Rounds of the Michael and Marian Ilitch Department of Surgery at the Wayne State University School of Medicine.

Every week, approximately 50 faculty attending surgeons and surgical residents meet to conduct postmortems on cases that did not go well. That "Mortality and Morbidity" conference is followed immediately by Grand Rounds.

This collection is not intended as a scholarly journal, but in a significant way it is a peer reviewed publication by virtue of the fact that every presentation is examined in great detail by those 50 or so surgeons.

It serves to honor the presenters for their effort, to potentially serve as first draft for an article for submission to a medical journal, to let residents and potential residents see the high standard achieved by their peers and expected of them, and by no means least, to contribute to better patient care.

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The Cutting Edge of Treatment for Hidradenitis Suppurativa

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Editor's Note: This is an edited summary of a Grand Rounds talk given by Dr. Daveluy on February 25, 2026 at the Ilitch Department of Surgery, Wayne State University School of Medicine.

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Introduction

Hidradenitis suppurativa (HS) is a chronic, relapsing inflammatory disease of the hair follicle that leads to nodules, abscesses, tunnel formation, scarring, and in advanced cases, irreversible tissue destruction. Although once referred to as "acne inversa," this terminology inadequately reflects both the pathophysiology and the severity of the condition. HS is not a variant of acne; it is a complex inflammatory disease with systemic associations, profound psychosocial burden, and a frequently delayed diagnosis.

Epidemiologic estimates suggest a prevalence ranging from 1–4% of the population, though this varies geographically and methodologically depending on case ascertainment and registry data.[1,2] In Western populations, HS demonstrates a female predominance of approximately 3:1, while certain Asian cohorts show a male predominance, potentially reflecting differences in smoking exposure patterns.[3]

The mean delay in diagnosis remains approximately 7–10 years, during which patients often consult multiple providers and receive repeated misdiagnoses.[3] This delay has important implications for disease progression, structural damage, and therapeutic responsiveness.

The modern management of HS rests on several core principles:

1. Assessment must drive therapy.
2. Medical therapy addresses inflammation and prevents new lesion formation.
3. Surgical therapy addresses persistent tunnels and structural disease.
4. Combination therapy is often required.
5. HS is almost never effectively treated with surgery alone.

The purpose of this manuscript is to synthesize contemporary understanding of HS pathophysiology, clinical staging, patient experience, and integrated medical–surgical management, with particular emphasis on procedural approaches such as deroofing.

Clinical Features and Disease Spectrum

HS is characterized by recurrent inflammatory nodules and abscesses occurring predominantly in intertriginous regions, including the axillae, groin, inframammary folds, buttocks, and perineal areas.[1,2] Over time, recurrent inflammation leads to tunnel formation (previously referred to as "sinus tracts"), hypertrophic scarring, fibrotic bands, and contractures.

Fig. 1. Clinical spectrum of hidradenitis suppurativa including nodules, abscesses, and sinus tracts in intertriginous areas. Adapted from *Nature Reviews Disease Primers*, 2020; *Dermatology* 2015;231:184–190.

HS is not a primary disorder of apocrine glands. It is a follicular occlusion disorder that evolves into a complex inflammatory and fibrosing process.[1,2]

Complications include chronic drainage, malodor, functional limitation, dyspareunia, depression, and social isolation. The disease burden is disproportionate to its apparent cutaneous footprint.

Epidemiology and Associated Risk Factors

HS typically presents in the second or third decade of life but may occur in adolescence or later adulthood.[3] Certain phenotypes have been observed:

- Younger female patients with hormonally influenced disease.
- Older male patients with rapidly progressive groin and perineal involvement.
- Severe, widespread disease in a subset of thin male patients with unclear mechanistic drivers.

Studies have demonstrated increased prevalence among African American patients in U.S. cohorts.[3] Smoking and obesity are strongly associated with HS, though causality remains multifactorial.[3]

It is critical to approach these associations carefully in clinical communication. Patients frequently report being told that HS is caused by poor hygiene, smoking, or obesity, often in a dismissive or stigmatizing manner. This undermines trust and delays engagement in meaningful care.

Hurley Staging and Structural Disease

The Hurley staging system remains widely used:

Stage I: Recurrent nodules or abscesses without sinus tracts or scarring.

Stage II: Recurrent abscesses with sinus tract formation and scarring; lesions widely separated.

Stage III: Diffuse involvement with interconnected tracts and abscesses across an entire anatomic region.[4]

Fig. 2. Hurley staging with representative images. *J Am Acad Dermatol.* 2009;60:539–561.

Hurley staging was originally developed as a surgical classification and does not fully capture inflammatory burden. Patients with multiple recurrent inflammatory lesions without established tunnels may still have moderate disease requiring systemic therapy.

Pathophysiology

The current pathogenic model conceptualizes HS as a disorder initiated by follicular occlusion and dysregulated immune activation.

The disease evolves in three phases:

1. Follicular occlusion and dilation
2. Follicular rupture and innate immune response
3. Chronic inflammatory state with tunnel formation

Following rupture, keratin, commensal flora, and debris enter the dermis, triggering neutrophilic and cytokine-driven inflammation.[5] As disease progresses, tunnels develop, surrounded by tertiary lymphoid structures containing organized B- and T-cell aggregates.

This architectural transformation explains an important clinical reality: once tunnels are established, purely medical therapy often fails to eradicate them. The tunnel itself becomes a nidus of persistent immune activation.

The Patient Experience

Patients with HS frequently endure years of misdiagnosis and invalidation.[3] Common misattributions include:

- "Recurrent infection"
- "Poor hygiene"
- "Sexually transmitted disease"
- "Weight-related boils"
- "Smoking-related skin problem"

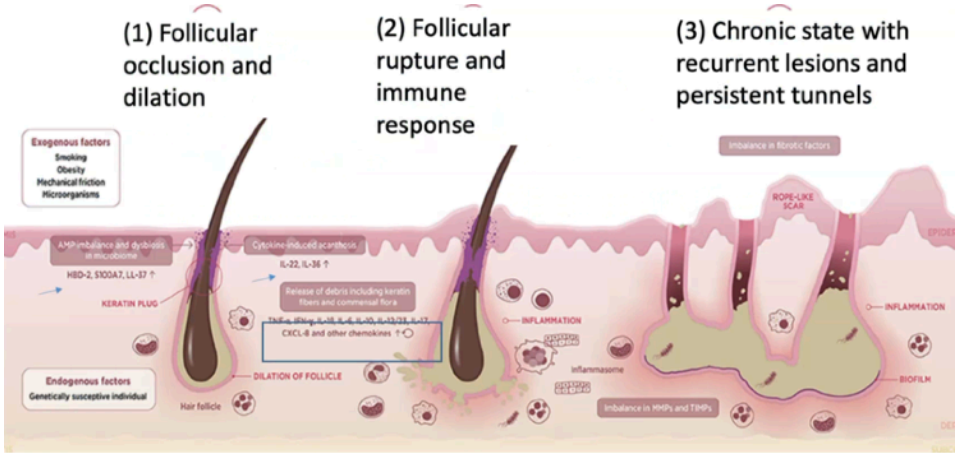

Fig. 3. Pathophysiologic model of HS. Vossen ARJV et al. *Front Immunol.* 2018;9:2965.

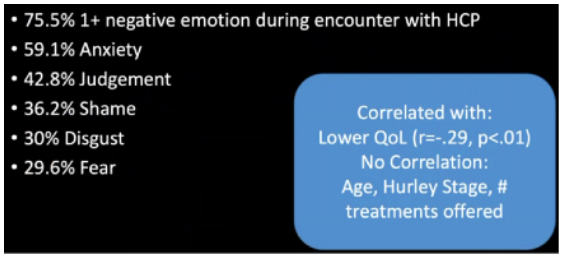

Fig. 4. Emotional responses during healthcare encounters in HS. *Int J Womens Dermatol.* 2022;8:e049.

In one study evaluating emotional responses during healthcare encounters, 75.5% of patients reported at least one negative emotion during clinical visits.[6] Anxiety (59.1%), perceived judgment (42.8%), shame (36.2%), disgust (30%), and fear (29.6%) were common.[6] These emotions correlated with reduced quality of life but not with disease severity.

The clinician must therefore pair clinical competence with deliberate empathy.

Diagnostic Approach

HS can be diagnosed reliably with two screening questions:

1. *Have you had outbreaks of boils during the last six months?*
2. *Where and how many boils have you had?*

This simple approach demonstrates 90% sensitivity and 97% specificity in patients with more than one boil in typical locations.[7]

Primary care education remains essential. Any patient with more than one boil in their lifetime, particularly in intertriginous areas, should prompt consideration of HS and referral to dermatology.

Comorbidity Screening

HS is associated with multiple systemic conditions including:

- Inflammatory bowel disease
- Spondyloarthritis
- Depression and suicidality
- Metabolic syndrome
- Dyslipidemia
- Diabetes mellitus
- Polycystic ovary syndrome

Screening recommendations are outlined in JAAD 2022 guidelines.[8]

Given the systemic inflammatory burden, HS should be approached as more than a cutaneous disease.

Assessment Drives Therapy

At each visit, the clinician must distinguish between:

- **Inflammatory disease** (erythema, swelling, drainage, new nodules)
- **Structural disease** (persistent tunnels, scars, contractures)

Tool	Comorbidity
History	Tobacco, IBD, Arthritis, Sexual Dysfunction
Physical Exam	Acne, Dissecting Cellulitis, Pilonidal, Pyoderma Gangrenosum
Height and Weight	Obesity
PHQ-2, PHQ-9	Depression, Suicidality
GAD-7	Anxiety
AUDIT-C, Opioid Risk Tool	Substance Use
Rotterdam Criteria	PCOS
Blood Pressure	Hypertension
Fasting Lipids	Dyslipidemia
Hemoglobin A1c	Diabetes

Table 1. Comorbidity screening recommendations in HS. J *Am Acad Dermatol.* 2022;86:1092–1101.

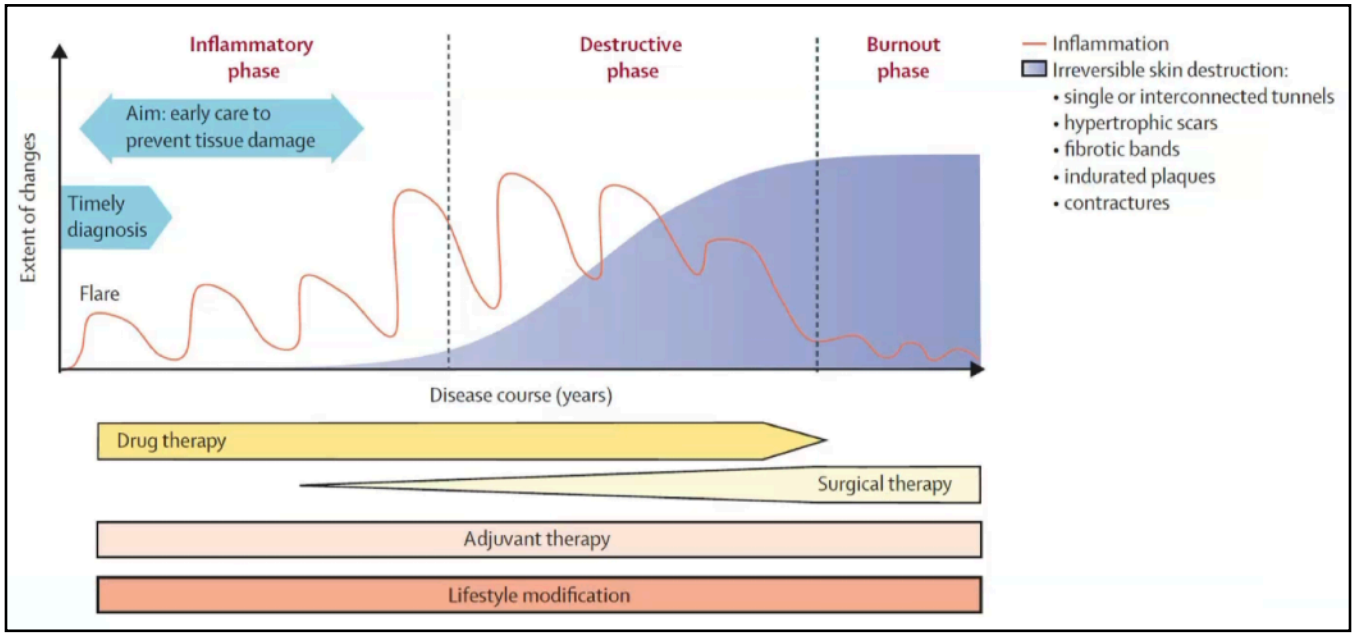

Fig. 5. Window of opportunity model in HS. *Lancet*. 2025;405:420–438.

Medical therapy reduces inflammatory burden and prevents new lesions. Surgical therapy removes persistent tunnels and fibrotic tissue that cannot be reversed pharmacologically.

This distinction forms the foundation of rational HS management.

The Window of Opportunity

Emerging evidence suggests that earlier intervention may improve long-term outcomes.[9,10]

Delayed initiation of biologic therapy is associated with reduced response rates. Patients with longer disease duration or multiple prior treatment failures demonstrate diminished responsiveness.[11]

This reinforces the importance of early diagnosis and timely escalation.

Medical Therapy: Controlling Inflammation and Preventing New Lesions

Once HS is diagnosed and staged, medical therapy aims to suppress active inflammation and prevent formation of new nodules and abscesses.

Importantly, medical therapy does not reliably eliminate established tunnels.

Topical Therapies

Topical clindamycin has demonstrated benefit in mild inflammatory disease and is frequently used as part of baseline therapy.[4] While its effect size is modest, it is well tolerated and easily incorporated into daily regimens. Adjunctive washes—benzoyl peroxide, chlorhexidine, or zinc-based cleansers—are often recommended despite limited randomized data, as they are low-risk interventions that may reduce bacterial load and inflammatory stimulation. Topical clindamycin should always be paired with an antimicrobial wash to prevent the development of bacterial resistance.

These therapies are not sufficient alone in moderate or severe HS.

Systemic Antibiotics

Oral tetracyclines and clindamycin remain commonly prescribed for inflammatory disease. Their utility derives from anti-inflammatory properties rather than antimicrobial effects.

In severe presentations, intravenous ertapenem has shown striking short-term efficacy.[12] While not a long-term solution, it can function as bridge therapy to biologics or surgery.

However, antibiotics alone do not address established structural disease.

Hormonal Therapy

HS frequently demonstrates hormonal sensitivity, particularly in female patients. Estrogen-containing oral contraceptives may be beneficial. Conversely, progestin-only contraceptives (including depot medroxyprogesterone) may exacerbate disease.

Spironolactone is widely used in women with inflammatory HS, particularly when flares correlate with menstrual cycles. Metformin may benefit patients with metabolic syndrome or insulin resistance. Finasteride can be used in select cases.

Hormonal modulation is often layered onto other systemic therapies.

Biologic Therapy

Biologics have transformed HS management. Approved agents include:

- Adalimumab
- Secukinumab
- Bimekizumab

These agents target inflammatory cytokine pathways central to HS pathogenesis.[13]

However, response may be limited in patients with longstanding disease and extensive tunnel formation.[9,11] As well, because they suppress new lesion formation but do not reliably eliminate existing tunnels, biologics are most effective in the inflammatory phase (Sup. Figs. 1A and B show the European guideline algorithm for distinguishing inflammatory and non-inflammatory HS.)

Fig. 6. Anti-inflammatory therapy versus surgical intervention paradigm. *Acta Derm Venereol*. 2017;97:412–413.

Combined Medical and Surgical Management

The evidence increasingly supports integration of biologics and surgery rather than viewing them as sequential alternatives.

A randomized study demonstrated that continuing adalimumab perioperatively improved outcomes without increasing postoperative complications.[14]

Similarly, combining infliximab with surgery significantly increased complete response rates compared with biologic therapy alone.[15]

The clinical implication is clear:

- Do not discontinue biologics for surgery in HS.
- Surgical removal of persistent tracts improves overall inflammatory control.
- Biologics reduce recurrence after surgery.

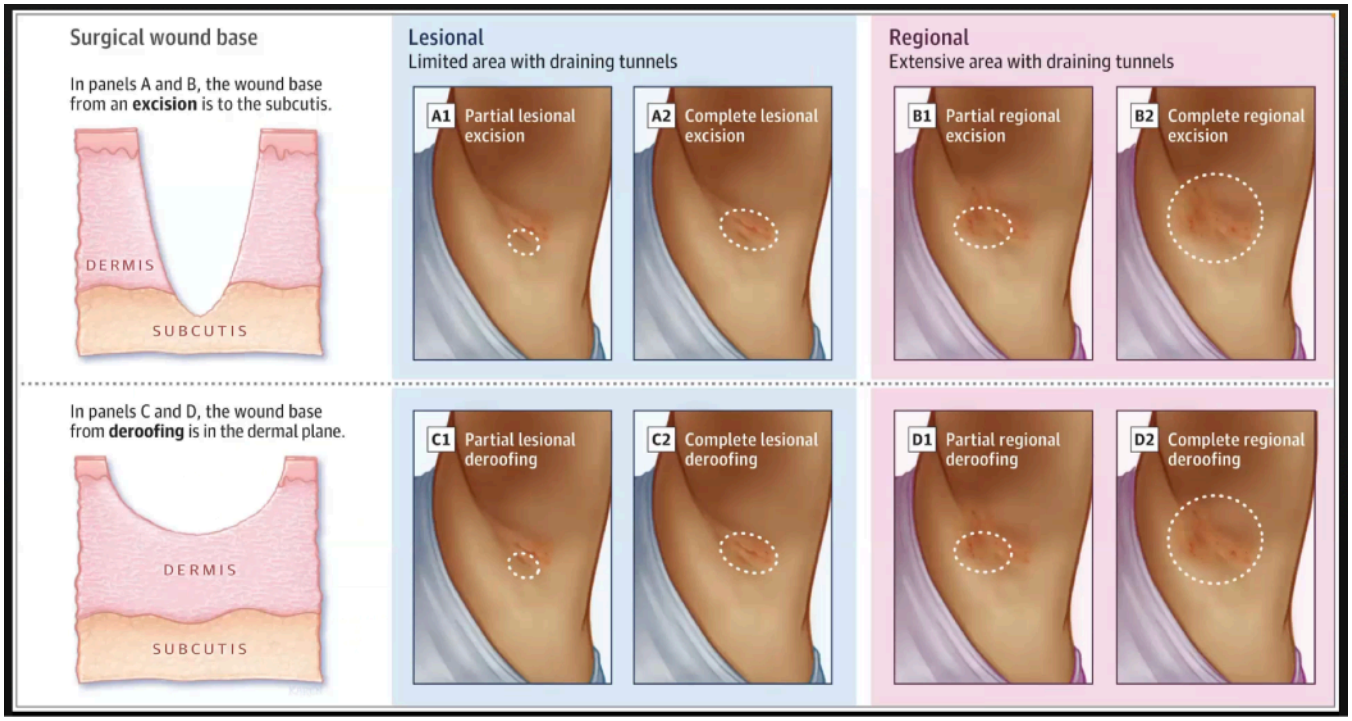

Fig. 7. Surgical classification framework: lesional vs regional; partial vs complete excision; dermal vs subcutaneous depth. *JAMA Dermatol*. 2023;159:441–447.

Surgical Strategy: Defining Structural Disease

When inflammation is controlled but persistent lesions remain—particularly tunnels—surgical intervention becomes necessary (see Fig. 8).

Recent efforts have standardized terminology for surgical approaches in HS.[16]

The distinction between excision (to subcutis) and deroofing (dermal plane) is critical for outcome interpretation and recurrence reporting.

Recurrence and Reconstruction

Meta-analyses suggest lower recurrence with wide excision compared with limited procedures.[17,18] However, historical data often grouped incision and drainage (I&D) with partial excisions, inflating recurrence rates.

Secondary intention healing consistently demonstrates lower recurrence than primary closure.

Primary closure may create potential spaces that predispose to tunnel reformation.

Smoking and Obesity: Surgical Outcomes

Recent systematic reviews show no statistically significant association between smoking and poor surgical outcomes in HS.[19] Similarly, obesity was not clearly linked to recurrence or repeat surgery.[20]

Therefore, surgery should not be withheld solely due to smoking or obesity in otherwise appropriate candidates.

Procedural Techniques in Detail

1. Incision and Drainage (I&D)

I&D provides temporary relief for tense abscesses but carries nearly universal recurrence.[21] As well, I&D does not remove sinus tract epithelium or inflamed tissue. It should be avoided when deroofing is feasible.

2. Deroofing

Deroofing involves removal of the "roof" of a sinus tract while preserving the floor and allowing secondary intention healing.

Technique:

1. Local anesthesia.
2. Probe sinus tract.
3. Incise overlying skin.
4. Remove roof using scissors, scalpel, or laser.
5. Curettage residual tract lining.
6. Allow healing by secondary intention.

Recurrence rates range from 4–17%.[22] [23]

Recent retrospective data demonstrate:

- 97% patient satisfaction
- 92% cosmetic satisfaction
- 3% recurrence at 1 year
- 5% recurrence at 5 years[24]

Deroofing is simple, efficient, and highly durable. It is superior to I&D and appropriate for persistent tunnels and recurrent nodules.

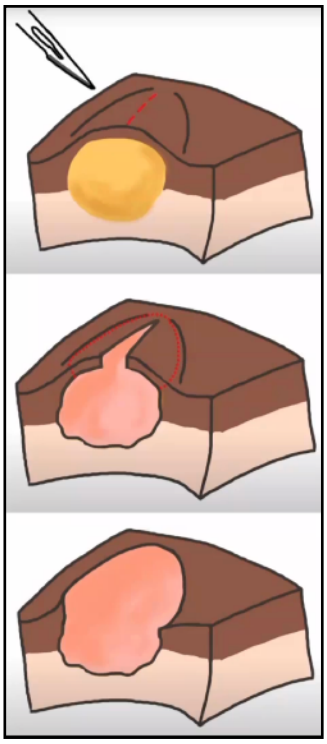

Fig. 8. Deroofing. *J Am Acad Dermatol*. 2010;63:475–480.

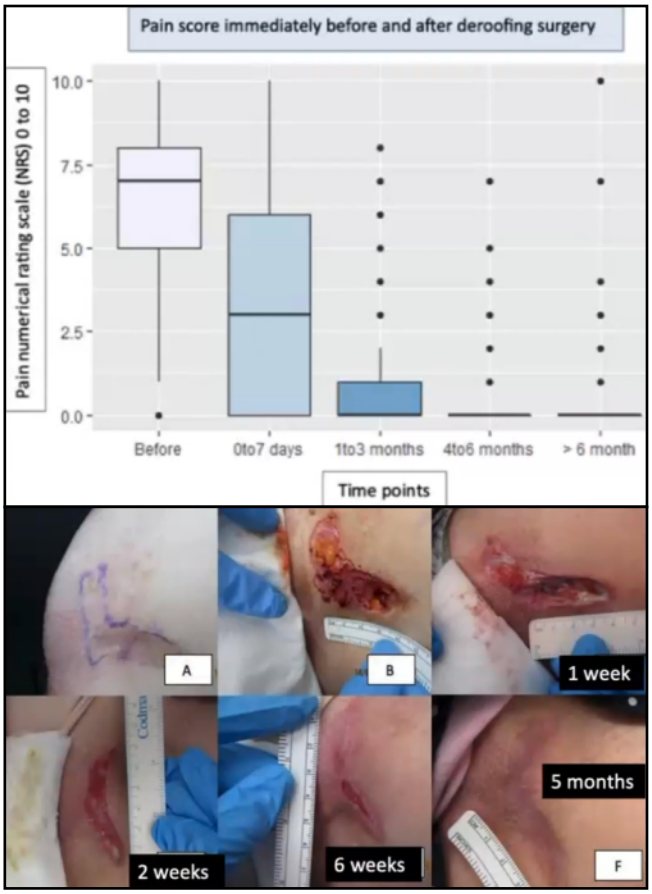

Fig. 9. Pain reduction and recurrence following deroofing. *Australas J Dermatol*. 2025.

Fig. 10. Punch debridement technique. *J Am Acad Dermatol.* 2015;73:S62–S65.

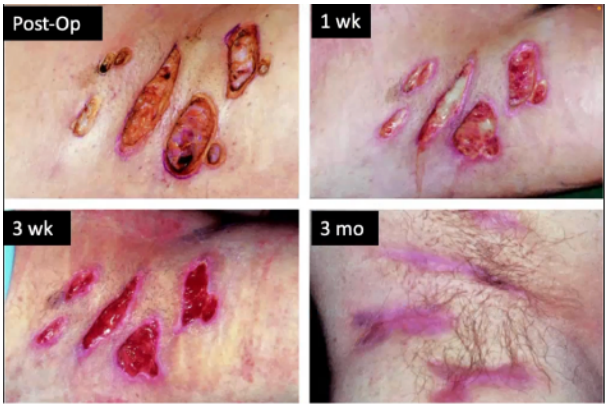

Fig. 11. CO_2 laser excision and postoperative healing timeline. *J Am Acad Dermatol*. 2002;47:283; Dermatol Surg. 2010;36:208–213.

3. Punch Debridement ("Mini-Deroofing")

For smaller nodules or abscesses without obvious openings:

1. Local anesthesia.
2. 5–8 mm punch into lesion.
3. Express contents.
4. Curettage lining.
5. Secondary intention healing.[25]

This technique converts transient abscess drainage into a definitive procedure.

4. CO_2 Laser Excision

CO_2 laser allows excision with simultaneous hemostasis. Cure rates are reported between 89.9–98.9% depending on technique and follow-up.[26-28] Laser is not mandatory but offers visualization advantages.

Fig. 12. Cryoinsufflation technique. *JAMA Dermatol*. 2014;150:765–766.

5. Cryoinsufflation

Cryoinsufflation involves insertion of a cannula into a tunnel followed by pulsed liquid nitrogen delivery.

Initial case series demonstrate resolution in selected tunnels.[30-32]

An alternative involves punch access followed by cryotherapy delivery.

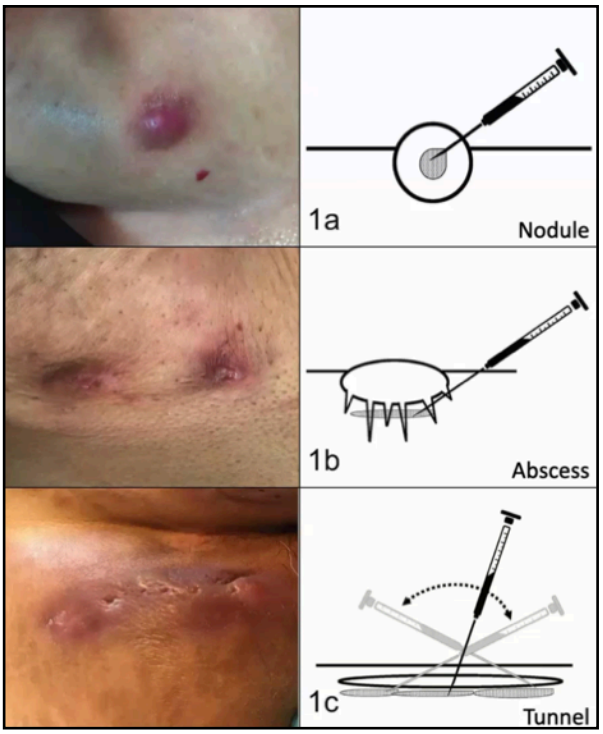

Fig. 13. Intralesional corticosteroid outcomes. *Sci Rep*. 2020;10:13363.

6. Intralesional Corticosteroid Injection

High-dose triamcinolone (40 mg/mL) injected intralesionally or perilesionally can result in:

- 81% nodule resolution
- 72% abscess resolution
- 53% tunnel improvement[33]

This may serve as bridge therapy or in select cases obviate surgery.

7. Hypertonic Saline and Botulinum Toxin

Emerging interventions include hypertonic saline injection into tunnels[34] and botulinum toxin for hyperhidrosis-associated disease.[34] These remain adjunctive strategies.

8. Tumescent Triamcinolone (Fig. 14, p. 10)

Case reports describe large-volume tumescent triamcinolone injections for refractory disease.[34] This concept reflects expanding procedural pharmacotherapy.

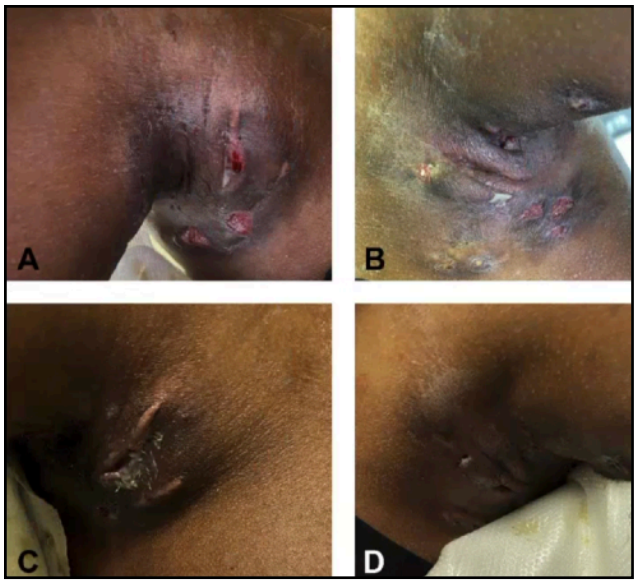

Fig. 14. Tumescent triamcinolone case illustration. *JAAD Case Rep*. 2020;6:1310–1312.

Surgical Healing Principles

Secondary intention healing is preferred:

- Avoids creating closed spaces
- Reduces recurrence
- Produces acceptable cosmetic results

Primary closure is associated with higher recurrence and is generally discouraged in HS.

Integrated Management Model

HS management requires dynamic assessment:

- Predominantly inflammatory disease → escalate medical therapy
- Persistent tunnels → surgical intervention
- Both present → combination therapy

The clinician must reassess at each visit and adjust accordingly.

Conclusion

Hidradenitis suppurativa is a chronic inflammatory disease that transitions into structural destruction over time. Effective management requires early diagnosis, biologic therapy in appropriate patients, and timely surgical elimination of persistent tracts.

Deroofing stands out as a high-value, durable, underutilized technique that bridges dermatology and surgery.

Integrated—not rigidly sequential—care is the modern standard.

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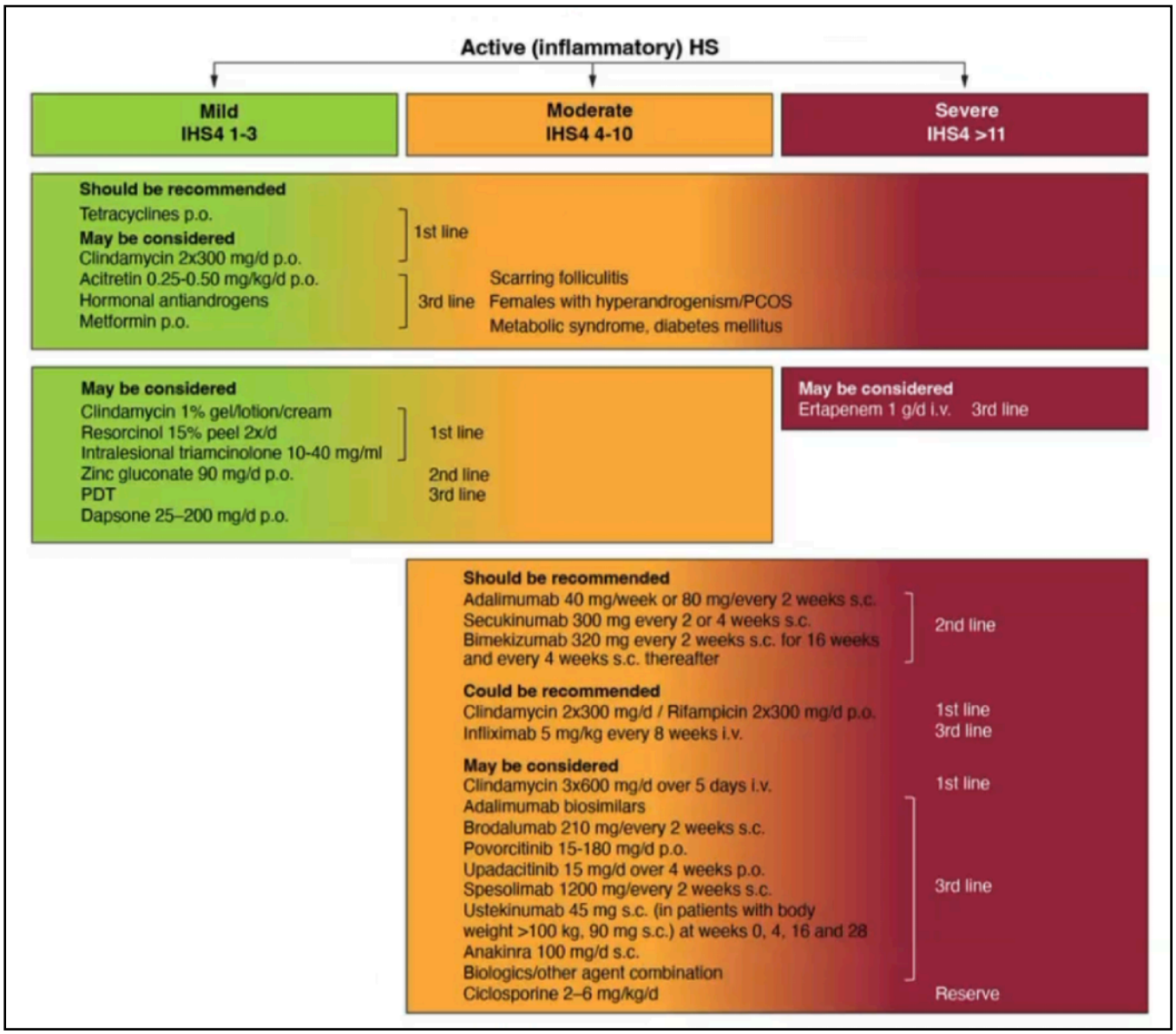
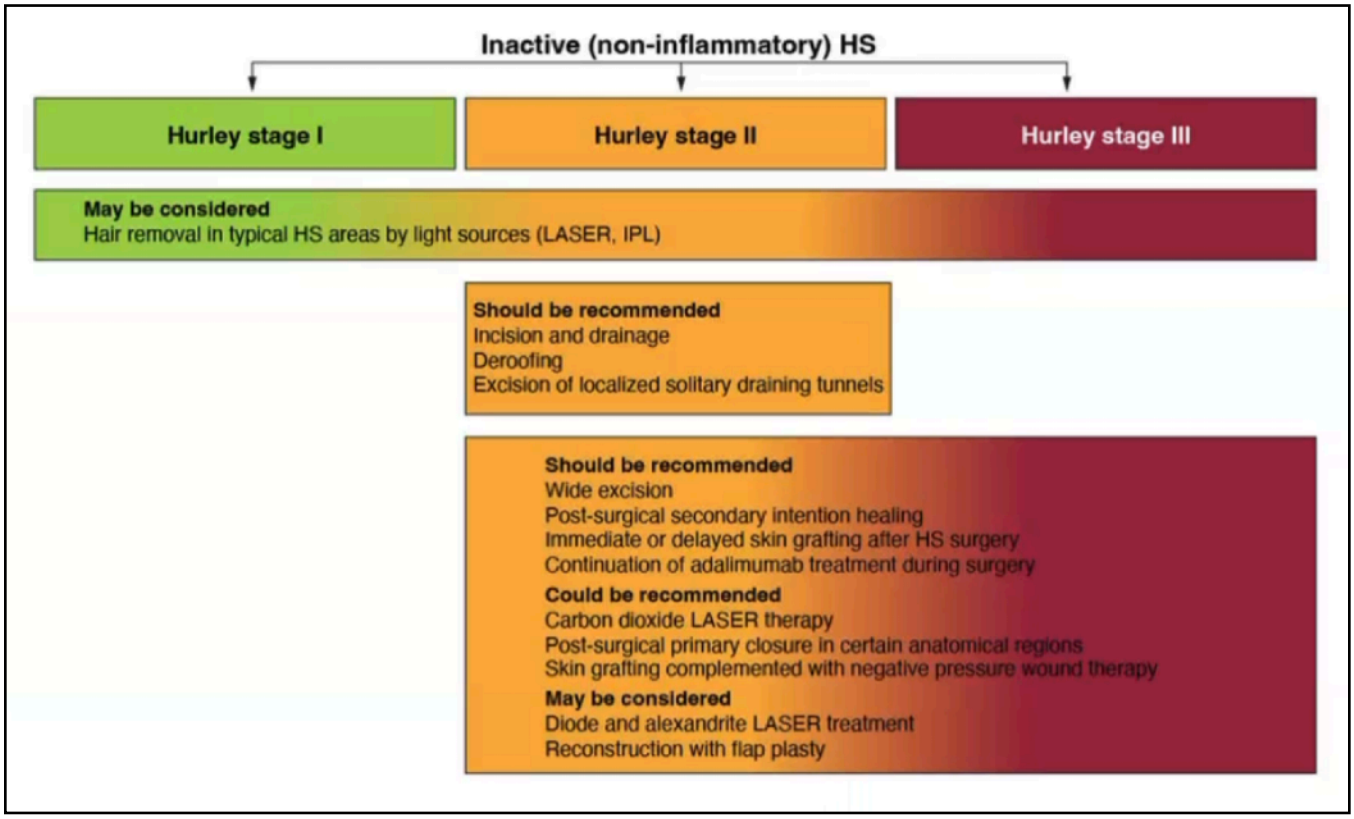
Supplemental Figures



Sup. Fig. 1A. European guideline algorithm distinguishing inflammatory and non-inflammatory HS: (A) Inflammatory. *J Eur Acad Dermatol Venereo*l. 2024;39:899–941.



Sup. Fig. 1B. European guideline algorithm distinguishing inflammatory and non-inflammatory HS: (B) Non-inflammatory. *J Eur Acad Dermatol Venereol*. 2024;39:899–941.

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