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Review

A Comprehensive Review of Hidradenitis Suppurativa: From Pathogenesis to Clinical Insights & Novel Therapeutic Advancements

Jeevan Rivera-Díaz,[1] Sofía Laguna Rocafort,[1] Ingrid M. Bonilla,[2] Alejandra Figueroa Moreda,[3] Jose Rabelo Cartagena.[3]

Abstract

Hidradenitis suppurativa (HS) is a chronic, recurrent, and inflammatory follicular skin disease that profoundly impairs patients' quality of life. Pathogenesis is thought to begin with follicular occlusion, leading to painful nodules, abscesses, and sinus tract formation. Pain is a central and debilitating symptom, occurring as both acute flare-ups and persistent chronic discomfort, significantly contributing to morbidity and psychological distress. This narrative review was conducted with methodological rigor, employing the Scale for the Assessment of Narrative Review Articles (SANRA) to ensure a thorough literature search and critical appraisal of evidence. Clinical diagnosis relies on the presence of characteristic lesions, typical involvement of intertriginous areas, and recurrence at least twice within six months. A major challenge remains the diagnostic delay, averaging 6 to 10 years from symptom onset, which is linked to increased disease severity, higher healthcare costs, and loss of productivity. Disease severity is commonly evaluated using tools such as the Hurley staging system, Sartorius scoring, and the HS Physician Global Assessment. Therapeutic options include antibiotics (e.g., clindamycin, rifampin), retinoids (e.g., acitretin, isotretinoin), hormonal agents (e.g., spironolactone, oral contraceptives), intralesional corticosteroids, botulinum toxin injections, laser hair removal, and photodynamic therapy. Biologic therapies, such as adalimumab and infliximab, represent emerging treatment modalities. Under-recognition and limited diagnostic tools continue to impede timely intervention. This review aims to improve awareness of HS, highlight its varied clinical presentations, and summarize evolving management strategies for the medical community.

Introduction

Hidradenitis suppurativa (HS), or acne inversa, is a chronic, inflammatory, and recurrent follicular skin disease that significantly impairs health-related quality of life[1,2] Typically presenting after puberty, HS is characterized by painful, deep-seated, inflamed lesions in apocrine gland–bearing regions, most commonly the axillary, inguinal, and anogenital areas, as defined by the HS Foundation consensus in 2009.3 The standardized point prevalence of HS in the United States is 98 per 100,000 individuals.[4]

Historically, HS was attributed to apocrine gland inflammation; however, current evidence implicates follicular occlusion as the primary pathogenic event. Occlusion leads to cellular debris accumulation, cyst formation, and, upon rupture, triggers local immune responses, pain, scarring, and, in some cases, malodor.[5,6] Pain is a prominent complication, manifesting as both acute nociceptive pain during flares and chronic discomfort, which can progress to reduced mobility and impair work performance and daily activities.[7] Scarring and malodor further contribute to the psychological and emotional burden of HS, alongside its physical comorbidities.[8–12]

Despite its substantial impact, the average diagnostic delay is close to 10 years, typically following multiple physician consultations, according to the VOICE project.[13] This delay prolongs patient suffering, worsens quality of life, and increases the risk of comorbidities such as anxiety, depression, inflammatory bowel disease, polycystic ovary syndrome, diabetes mellitus, thyroid disorders, inflammatory arthritis, obesity, metabolic syndrome, acne, pyoderma gangrenosum, and anemia.[7]

This review aims to address the gap between clinical presentation, recognition, and management of HS by evaluating disease pathogenesis, diagnostic approaches, current pharmacological strategies, and emerging therapeutic targets.

Search Strategy and Selection Criteria

This narrative review utilized the Scale for the Assessment of Narrative Review Articles (SANRA) to ensure methodological rigor. Literature was identified through MEDLINE, Google Scholar, Web of Science, and EMBASE, focusing on peer-reviewed articles published in English between June 2014 and June 2024. FDA databases were also consulted for medication side effect profiles. Searches employed specific keywords (e.g., "hidradenitis suppurativa," "apocrine glands," "acne inversa," "skin nodules," "skin abscess") and Boolean operators ("AND," "OR") to optimize search sensitivity and specificity. Additional filters included article

[1] Third-year Medical Student. Universidad Central del Caribe, Bayamon, PR, and VA Caribbean Healthcare System, San Juan, USA.

[2] PhD. VA Caribbean Healthcare System, San Juan, PR and University of Puerto Rico, School of Medicine, San Juan, USA.

[3] MD. VA Caribbean Healthcare System, San Juan, USA.

About the Author: *Jeevan Rivera-Díaz is currently a third-year medical student of Universidad Central del Caribe, San Juan, Puerto Rico of a 4-year program.*

Correspondence:

Jose Rabelo Cartagena.

Address: Laurel, Av. Sta. Juanita, 00960, Puerto Rico.

Email: jose.rabelo-cartagena@va.gov

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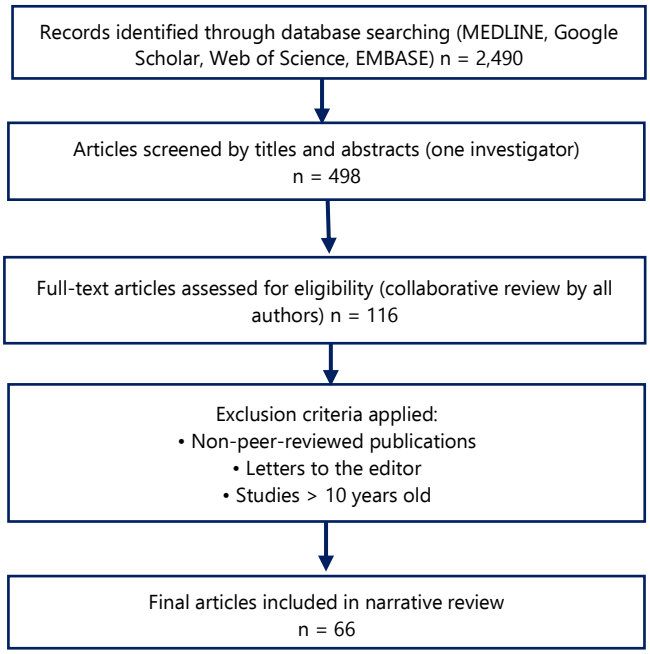
type (original research, reviews, case reports) and peer-reviewed status.

Titles and abstracts were initially screened for relevance by one investigator, with final article selection performed collaboratively by all authors in June 2024 to ensure comprehensive and unbiased inclusion. Exclusion criteria were non-peer-reviewed publications, letters to the editor, and studies older than ten years. Ultimately, sixty-six articles were included ***(Figure 1).*** This systematic and rigorous approach, guided by SANRA criteria, ensured the quality and comprehensiveness of this narrative review.

Figure 1. Literature Identification and Screening Process.



Legend: Articles were identified through MEDLINE, EMBASE, and Google Scholar between June 2014 and June 2024. Following duplicate removal and sequential screening, sixty-six studies were included in this narrative review in accordance with SANRA quality criteria.

Epidemiology

The global incidence rate of HS is highly variable, with estimates ranging from 0.00033% to 4.1%.15,16 In the United States, the prevalence rate is estimated to be nearly 10%, occurring three times more frequently in women than in men. The highest incidence is observed in women aged 20 to 29 years.4 In addition to gender, race significantly influences HS prevalence in the United States; African Americans have an adjusted prevalence of 296 per 100,000, which is more than three times higher than that of Caucasian patients, who have a prevalence of 95 per 100,000.4 Interestingly, in contrast to the gender prevalence observed in the United States, South Korean and Japanese studies found that in their population, HS occurrence was more prominent in men than women.[4]

Additionally, studies have shown that the clinical presentation of HS differs between women and men. Women more commonly develop cysts in anterior body regions, such as the groin and breasts, while men are more frequently affected in the gluteal region.17 Furthermore, studies have demonstrated that up to 42% of patients with HS have a family history of the disease.[17] These findings suggest that genetic factors may play a significant role in the development of HS. The disease typically manifests after puberty, most commonly between the ages of 18 and 19, and can have a profound impact on the quality of life in this age group.[17]

Co-Morbidities

HS is associated with an increased risk of depression, further intensifying the burden of this chronic, painful, and socially isolating disease.[18] Moreover, patients with HS have been shown to experience higher rates of anxiety and completed suicide.[19] Feelings of shame, unworthiness, and being unlovable, encompassed under the term "internalized stigma," have been reported among these patients. Feelings of shame, unworthiness, and being unlovable—collectively referred to as "internalized stigma"—are frequently reported among those affected. Predictive factors for high internalized stigma include obesity, low income and educational level, and genital involvement.[20] These findings underscore the profound negative impact of HS, affecting not only physical health but also the psychological well-being of patients.

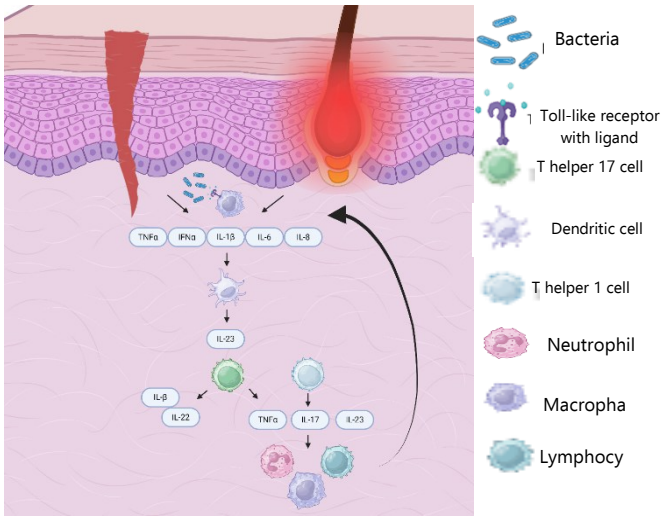
Hormones

It has been shown that patients with HS frequently have coexisting endocrine disorders, suggesting a role for hormonal imbalances in HS development. Polycystic Ovary Syndrome (PCOS) is one such condition, sharing several features with HS, including a predilection for young women and strong associations with metabolic syndrome and obesity.[18] In a cross-sectional study of 22,990 HS patients using a multi-health system analytics platform, the prevalence of PCOS among HS patients was 9.0%, compared to just 2.9% in individuals without HS ($P < 0.0001$). 21 The odds ratio for PCOS in HS patients was 2.14 (95% CI: 2.04–2.24), indicating that HS patients are more than twice as likely to have PCOS compared to those without HS.[18,21] These findings highlight the increased prevalence of concomitant PCOS in HS patients and underscore the potential role of hormonal factors in the development of both conditions.

Cardiovascular

The results of a study done in 2016 found that HS is associated with an increase in adverse cardiovascular outcomes, including ischemic stroke and myocardial infarction.[18,22] Moreover, HS patients presented a higher risk of cardiovascular-associated death than patients with severe psoriasis.[18] It is important to note that while there are no definite mechanisms outlined for the observed association between HS and cardiovascular outcomes, there are several shared risk factors that can play a role, such as obesity, smoking and metabolic syndrome, and chronic inflammation, all of which overlap with the pathophysiology of HS.

Figure 2. The Role of Immune System Dysregulation in the Pathophysiology of Hidradenitis Suppurativa.



Legend: When the skin gets injured, pathogens can enter deeper layers and areas around hair follicles. These pathogens activate toll-like receptors, which trigger the release of pro-inflammatory signals called cytokines (such as TNF α, IFN α, IL-1 β, IL-6, and IL-8). These cytokines then activate dendritic cells, which produce another signal called IL-23. IL-23 activates T helper cells, which further activate macrophages, dendritic cells, and neutrophils. Although this immune response is initially aimed at fighting infection and reducing inflammation, in HS it becomes a positive feedback loop. The continuous and excessive activation of immune cells leads to chronic inflammation, which in turn triggers more immune responses, perpetuating the cycle and causing ongoing tissue damage and painful skin lesions.

Smoking

Active smokers with HS tend to have more affected body areas than non-smokers.[17] Studies have shown that approximately 90% of individuals with HS are smokers.[23] Studies indicate that about 90% of HS patients are smokers, and a large U.S. cohort study identified smoking as a risk factor, with smokers showing twice the incidence of HS.[18] While the exact mechanism remains unclear, nicotine is linked to follicular plugging, increased cytokine production, neutrophil chemotaxis, and delayed lesion healing.[23] Additionally, nicotine may promote Staphylococcus aureus colonization, inflammatory mediator recruitment, and infundibular epidermal hyperplasia, all contributing to HS pathogenesis.[24]

Obesity

Obesity is well known to be associated with HS, with 60-77% of patients being overweight and 30% obese. Obese patients have increased friction and mechanical stress due to larger inverse skin areas.[25] The relationship between obesity and HS is characterized by the release of pro-inflammatory cytokines from adipose tissue, such as TNF α and IL-6, contributing to inflammation in HS patients. Moreover, HS patients exhibit a 50.6% prevalence of metabolic diseases, significantly higher than that of the general population.[18,26] Notably, HS is linked to hypertriglyceridemia, metabolic syndrome, and low high-density lipoprotein.[18] HS and metabolic syndrome exhibit a comparable adipokine profile, featuring increased levels of leptin, resistin, and visfatin, along with reduced serum adiponectin, which predisposes individuals to both conditions. Further illustrating the link between HS and obesity, Thomas et al. documented a case of rapid HS improvement following bariatric surgery.[27,28]

Pathophysiology

The precise pathophysiology of HS remains incompletely understood, but it is widely accepted to be multifactorial.[29] Ongoing research into its underlying mechanisms is critical for informing effective treatment strategies. Although HS was once thought to be a suppurative disorder primarily affecting the apocrine sweat glands, recent advances highlight its characterization as a chronic disease driven by follicular occlusion within the pilosebaceous units.[30] Multiple factors are implicated in disease development, including immune system dysregulation, hormonal imbalances, genetic predisposition, and environmental influences.[31]

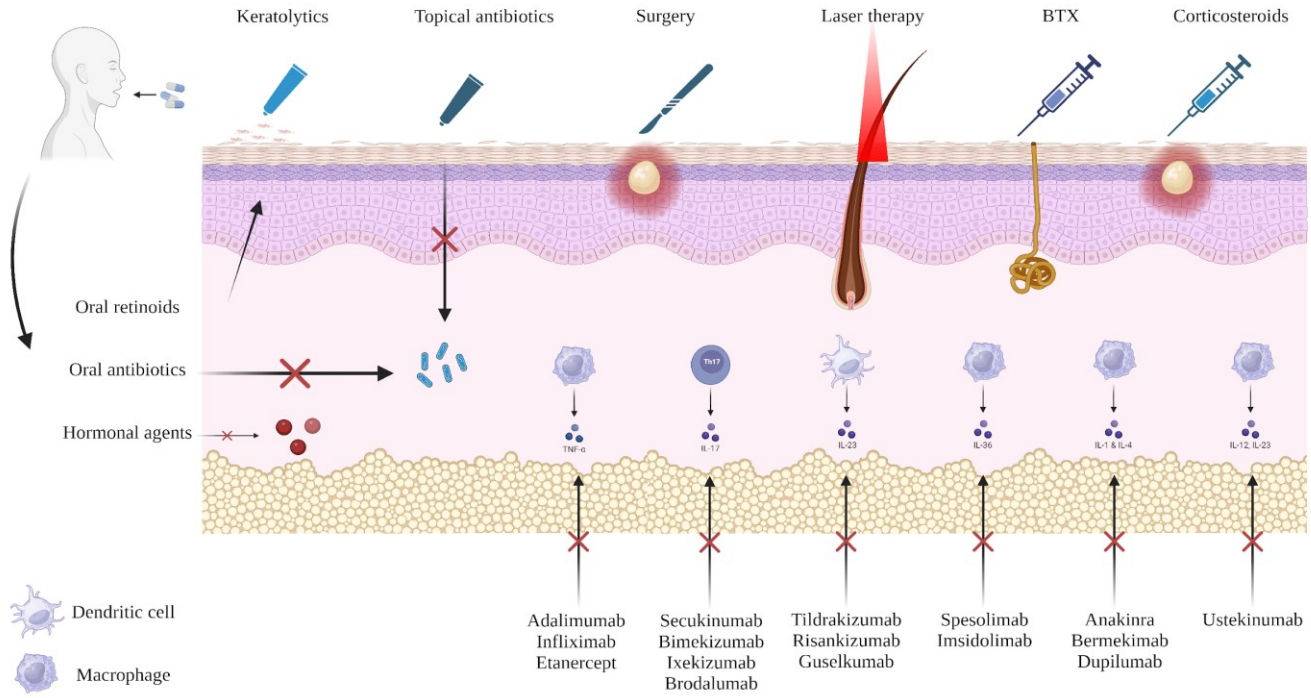
Immune System Dysregulation

Friction and microscopic epidermal injuries trigger an innate immune response, facilitating pathogen entry into the dermis and hair follicles in intertriginous areas, which leads to lesion formation.[32] The activation of the innate immune system promotes hyperplasia of the follicular epithelium, resulting in hair follicle rupture.[32] This rupture releases macrofollicular contents, such as keratin fragments, sebum, and bacteria, leading to the recruitment of additional inflammatory cells and abnormal activation of the complement system (e.g., C3a and C5a). As a result, inflammatory nodules, abscesses, and skin tunnels that open on the skin surface can form and become chronically inflamed.30 Activation of toll-like receptors by pathogen- and damage-associated molecular patterns triggers the release of pro-inflammatory cytokines such as TNF α, IFN α, IL-1 β, IL-6, and IL-8, which in turn activate dendritic cells. These dendritic cells secrete IL-23, stimulating Th17 cell activation and subsequent keratinocyte proliferation, while promoting the production of IL-17, IL-22, IL-1 β, and TNF α. 32 Both Th1 and Th17 cells release IL-17, IL-23, and TNF α, establishing a positive feedback loop that sustains chronic inflammation ***(Figure 2).***[32] IL-17 further stimulates the release of chemokines, chemokine (C-X-C motif) ligand 1 (CXCL1), CXCL8, and CC motif chemokine ligand 20 (CCL20), attracting neutrophils, macrophages, and lymphocytes to HS lesion sites.[32]

Hormonal imbalances

The interplay between PCOS, obesity, insulin resistance, and increased levels of systemic pro-inflammatory mediators—such as insulin-like growth factor 1 (IGF-1), leukotrienes, and long-chain fatty acids—further underscores the significant role of hormonal and metabolic factors in the pathogenesis of HS.[33] Androgen-receptor-mediated inflammatory pathways linked to IL-23 may provide insights into how the Th17 pathway implicated in HS correlates with hormonal dysregulation.[33] Androgens, particularly testosterone, are associated with HS because they stimulate apocrine gland growth and secretion.[34] Hormones such as androgen, estrogen, progesterone, and prolactin may contribute to infundibular hyperkeratosis, follicular occlusion, and HS disease progression.[35]

Figure 3. Current Therapeutic Approaches and Ongoing Research Biologics for Hidradenitis Suppurativa.



Legend: Therapeutic approaches for HS encompass a combination of interventions aimed at reducing inflammation, managing symptoms, and preventing disease progression. Medical treatments often begin with topical and systemic antibiotics to control bacterial infection and inflammation. Hormonal therapies, such as oral contraceptives and anti-androgens, are utilized in individuals with hormone-related HS. For moderate to severe cases, systemic anti-inflammatory drugs like retinoids and immunosuppressants are used. Recently, biologics have emerged as a significant advancement in HS treatment, with tumor necrosis factor-alpha (TNF-α) inhibitors like adalimumab and other FDA-approved biologics HS treatment. Other biologics targeting interleukin (IL)-17, IL-23, and IL-1 pathways have been recently approved by FDA while others remain under investigation, showing promising efficacy in clinical trials. Surgical interventions, including incision and drainage, deroofing, and wide excision of affected areas, are employed in certain cases.

Genetic factors

Evidence suggests a hereditary aspect of the autosomal dominant transmission pattern of HS, with approximately 33 to 40 percent of affected individuals reporting a first-degree relative with the condition.[34] Mutations in genes associated with gamma-secretase, an intermembrane protease complex that cleaves the intracellular domain of the Notch transmembrane receptor protein, affect keratinocyte differentiation in HS.[30] Notable mutations include those in presenilin-1 (PSEN1), presenilin enhancer-2 (PSENEN), and nicastrin (NCSTN).30 Additionally, variations in genes encoding IL-1, IL-12, and IL-23 are implicated in HS pathogenesis.[34]

Environmental influences

Several external factors contribute to the pathogenesis of HS, including mechanical stress, obesity, smoking, bacterial colonization, and certain medications.

Mechanical Stress

Increased mechanical stress from pressure, friction, or shear forces on intertriginous skin and areas such as beltlines and bra straps heightens the risk of follicular occlusion and rupture.[30]

Bacterial colonization

The warm and humid environment in conjunction with high concentration of pilosebaceous-apocrine units in intertriginous areas create favorable conditions for microbial growth.32 Antimicrobial peptides (AMPs) produced by epithelial and immune cells are vital for the skin's innate immune defense, preventing the overgrowth of commensal bacteria, inhibiting pathogen invasion, and facilitating cutaneous wound healing.[32] Disruption of AMPs and the complement system contributes to chronic inflammation and microbiota imbalances in HS. Early HS lesions show higher levels of commensal bacteria, while advanced lesions feature reduced commensals and increased pathogenic bacteria.[32] Sinus tracts and fistulas containing keratin debris and hair fragments can foster biofilm formation by gram-negative anaerobes, complicating treatment due to antimicrobial resistance.[32]

Medication use

Evidence suggests that androgens may exacerbate HS, as observed in female patients receiving oral contraceptives with androgenic progestins, intramuscular medroxyprogesterone acetate, or levonorgestrel-releasing intrauterine devices.[30] Additionally, HS has been reported in association with lithium

therapy and, in rare cases, as an adverse effect of anti-TNF-alpha agents and other biologics used to treat chronic inflammatory diseases.[30]

Disease Presentation and Diagnosis

HS is diagnosed clinically using three main criteria: typical HS lesions, classical locations of intertriginous areas, and two or more occurrences within six months.[19,23,36] Typical HS lesions are described as inflammatory nodules, abscesses, and comedones.[20] Nodules and abscesses often rupture, secreting sanguineous & purulent material.[19,23] Lesions may occur singly or in multiple clusters, and with chronic disease progression, often lead to significant scarring and disfigurement.[36] Disease progression may connect adjacent nodules forming sinus tracts–deep dermal tunnels lined by epithelium and associated with inflammation.[19] Patients commonly experience pruritus, malodor, and painful & burning sensations.[23] An average of three misdiagnoses are made before achieving the correct diagnosis of HS.20 Differential diagnoses include cutaneous abscess, inflamed epidermal cyst, furunculosis, cellulitis, necrotizing fasciitis, acne vulgaris, inflamed epidermal cysts, and cutaneous Crohn's disease.[20,36]

Most studies show a delay of HS diagnosis ranging from six to ten years after initial presentation, averaging ten years.[20] This diagnostic delay can lead to worse patient morbidity, higher costs to the healthcare system, and increased likelihood of patients' wage loss due to work absence.[20] The support of accurate and early recognition of the disease is needed to reduce diagnostic delays and, thus, the limitation of HS progress/management of the comorbidity burden.[37,38] Inadequate recognition and incorrect diagnosis of the disease may stem from the widespread lack of awareness about HS within the medical community and the significant variability in its clinical manifestation.[39] Research has shown that HS patients visited more than 3 different physicians on average including general practitioners, dermatologists, surgeons and gynecologists. The correct diagnosis was made by dermatologists in most cases.[37]

HS usually occurs in intertriginous anatomical locations (axillae, inframammary area, inguinal folds, gluteal cleft, and perianal region) and areas of friction (posterior neck, intermammary cleft, abdominal & flank folds, and medial thighs).[19] Less frequently, lesions may present in the lower abdomen, suprapubic area, retroauricular area, nape, eyelids, and scalp.[23]

The variation in clinical disease presentation, appearance, and response to treatment pose a challenge to accurately diagnosing HS.40 Diagnosis is made by exclusion of other more common conditions, such as infectious abscess, cellulitis, cystic acne, or epidermal inclusion cyst.[41] To enhance a correct diagnosis, family history and history of other follicular occlusion diseases should be considered. Likewise, recurrence history or signs of previous episodes, along with the previously mentioned presence of comedones in typical areas, are valuable for differentiating HS.[41]

The most common tools used to assess the severity of HS are the Hurley staging, Sartorius scoring, and HS Physician Global Assessment systems.[23] The Hurley Staging System is the most used and is composed of three stages: (1) one or more lesions without sinus tract formation; (2) one or more widely separated lesions with sinus tracts and scarring; and (3) multiple lesions connected via sinus tracts and extensive scarring involving an entire anatomic area.[19] Worldwide, most HS patients are classified as Hurley stages I and II.[42] Typically, this system evaluates the entire body, and the highest stage would be applied, describing the patient overall rather than individual sites.[41] Patients classified with stage I may be treated with medical therapy, stage II with wide local surgery, and stage III with surgical excision of the entire affected region.[40]

The Sartorius Scoring System is a more accurate and precise method to define severity that was later proposed, though it is more time-consuming.[23] Meanwhile, the HS-Physician's Global Assessment is a fixed 6-point scale based on lesion counts in areas prone to inflammation.[43]

Currently, HS is not diagnosed by a definitive serum or histology test.[19] Likewise, HS can't be diagnosed utilizing a skin culture or laboratory testing.[41] Commonly, the content of lesion or drainage cultures reveals several types of microorganisms or normal skin flora.[36] Point-of-care ultrasound (POCUS) may aid in visualizing collections filled with fluid in soft tissue and may show signs of chronic disease. Still, it isn't reliable to differentiate between HS and infectious abscesses.[41] The use of ultrasound in practice is currently limited due to a lack of standardization and validation. Lastly, laser speckle contrast analysis (LASCA) and optical coherence tomography (OCT) are other techniques that have been reported for the diagnosis and treatment monitoring of HS.[39]

Management

Over 50 treatments have been investigated for HS, including topical, systemic, and surgical modalities.[34] Medical therapies comprise antibiotics, retinoids, hormonal agents, intralesional corticosteroid and botulinum toxin injections, laser hair removal, photodynamic therapy, and biologics *(Figure 3)*[7] Ultimately, clinicians use Hurley staging to evaluate disease severity and determine the most appropriate treatment option for patients.[34]

Antibiotics

The first line of therapy in HS is systemic antibiotics due to bacterial colonization and biofilm formation in lesions.[44] In mild stages of the disease, monotherapy is possible, yet lower response rates and increased recurrence are observed in more advanced stages.[45] Typical therapeutic regimens include the prescription of tetracyclines (doxycyxline or minocycline), clindamycin monotherapy, clindamycin/rifampicin, clindamycin/ofloxacin, trimethoprim/sulfamethoxazole, dapsone, and ertapenem.[34] Antibiotics are limited by bacterial resistance, requiring the implementation of other alternatives such as aseptic

washes (e.g., benzoyl peroxide, chlorhexidine, bleach, and pyrithione zinc).[34]

Biological Agents

Unlike antibiotics, biologics represent a newer class of medications designed to treat HS and are used for long-term maintenance control.[34,46] They are the preferred treatment for moderate to severe HS.44 The most common side effects for the following biologics include headache, diarrhea, nausea, infections, injection site reaction, joint & muscle pain, and tiredness.[47–58]

TNF

TNF inhibitors have seen a growing use in the treatment of HS.34 Adalimumab, the most extensively researched biologic agent for HS, is the first FDA-approved medication for this condition.59 It is a fully-humanized monoclonal antibody targeting both soluble and transmembrane TNF.[59] Infliximab is a chimeric monoclonal antibody that targets soluble and transmembrane TNF used as a second-line biological treatment for HS, particularly when Hurley stage III HS resists adalimumab therapy.[34]

Etanercept is a recombinant human TNF inhibitor that functions as a soluble TNF receptor, binding to TNF-α and TNF-β, with multiple open-label studies reporting its efficacy and safety in managing HS.[59] In four HS patients, three case reports were detailed using golimumab, a fully human anti-TNF monoclonal antibody that targets both soluble and transmembrane TNF.[59]

IL-17

Secukinumab, a monoclonal antibody inhibitor of IL-17, is the latest FDA-approved medication for treating adult patients with moderate to severe HS.[34,48] It has demonstrated efficacy as a potential treatment for HS since 2017.[59] The recommended dosage is 300 mg administered by subcutaneous injection weekly for five consecutive weeks, then every 4 weeks. However, its safety and effectiveness in pediatric patients with HS have not been established.[48] The most promising biologic in phase III trial is bimekizumab, a humanized IgG1 monoclonal antibody that specifically targets IL-17A and IL-17F used to treat plaque psoriasis in patients.59

Ixekizumab is a monoclonal antibody that inhibits IL-17 and is currently approved by the FDA for treating psoriasis and psoriatic arthritis.[59] A few studies have demonstrated its potential efficacy in treating HS, with two case reports indicating its use in patients with concomitant HS and psoriasis, and a small case series presenting four out of five patients that achieved Hidradenitis Suppurativa Clinical Response (HiSCR).32 Likewise, brodalumab is a fully human IgG2 monoclonal antibody that binds to the IL-17RA subunit of the IL-17 receptor, thereby interfering with the signaling of various IL-17 isoforms, including IL-17A, IL-17C, and IL-17F. Three case reports have documented the promising use of brodalumab in HS patients.[59]

IL-23

Tildrakizumab is a humanized IgG1 monoclonal antibody which targets the p19 subunit of IL-23 and is approved to treat moderate-to-severe plaque psoriasis. Literature reviews also reveal reports of tildrakizumab-asmn being used in the management of HS.60 Only two case series and two case reports have been published regarding its use as treatment in HS. Yet they provided encouraging results (e.g., abscess and nodule count reduction, improved quality of life, HiSCR achievement, etc).[61]

Risankizumab, a monoclonal antibody that selectively blocks IL-23 by binding to the p19 subunit, is approved to treat Crohn's disease, psoriasis, and psoriatic arthritis.[59,60] Three case reports documented it was successfully administered to four HS patients. Similarly, guselkumab is a IL-23 inhibitor approved for treating psoriasis and psoriatic arthritis, which has been identified as a novel biologic being studied for HS management with several case reports and case series documenting its effectiveness in patients with moderate-to-severe HS that is refractory to other systemic treatments.[59,60]

IL-12/23

Patients with severe HS who failed treatment with adalimumab and infliximab have been treated off-label with ustekinumab—a human monoclonal antibody against the p40 subunit of IL-12 and IL-23.34,45 Several literature reports have shown its efficacy in treating HS, though it's currently approved for treating Crohn's disease, ulcerative colitis, plaque psoriasis, and psoriatic arthritis.[60]

IL-1 & IL-4

Though reports on its efficacy present fluctuating results, anakinra, a recombinant IL-1 receptor antagonist approved for rheumatoid arthritis, has been noted as a possible therapeutic agent for HS. Bermekimab, a fully human recombinant IgG1 monoclonal antibody that also inhibits IL-1α currently in phase II trials, shows promising results.[62] Likewise, Dupilumab—a monoclonal antibody targeting the IL-4a receptor presently approved for treating moderate-to-severe atopic dermatitis—has been used effectively in numerous case reports involving patients with both HS and concomitant atopic dermatitis.[60] In contrast, canakinumab is a human monoclonal antibody against IL-1β. No significant clinical trials have investigated its safety and efficacy in HS, and results from single case reports or case series remain ambiguous.[62]

Other agents

The upregulation of IL-36RA has been shown in HS lesions compared to healthy skin. Therefore, two phase II trials are currently underway for the anti-IL-36 receptor monoclonal antibodies spesolimab and imsidolimab in the treatment of HS.[32] Other biological agen62ts that have shown varied results include CD20 inhibitors (Rituximab), complement C5a inhibitors (Vilobelimab, Avacopan), CD40 inhibitors (Iscalimab), leukotriene

A4 inhibitor (LYS006), phosphodiesterase-4 inhibitors (Apremilast), CXC receptors (LY3041658), and janus kinase inhibitors (INCB054707, Tofacitinib, Upadacitinib).62

Keratolytics

Retinoids are used in HS treatment due to similarities with acne vulgaris pathogenesis. [44,45] Oral retinoids, including acitretin and isotretinoin, have also been used to treat HS.[34] Oral retinoids such as acitretin and isotretinoin have been employed, though isotretinoin has shown mixed results in HS despite its anti-inflammatory and keratinocyte-modulating effects. Acitretin, which inhibits epidermal growth and differentiation, has demonstrated overall response rates around 50% and high recurrence with monotherapy, making its efficacy controversial. [60, 62,63] Evidence for topical retinoids in HS is limited to a single case report of successful use alongside chlorhexidine wash, clindamycin solution, and oral doxycycline.[63] Additionally, topical resorcinol, valued for its keratolytic, antimicrobial, and anti-inflammatory properties, appears safe and effective for long-term management of mild-to-moderate HS.[44]

Hormonal agents

Monotherapy with hormonal agents (e.g. spironolactone, metformin, finasteride, and ethinylestradiol) may be beneficial for female patients with mild-to-moderate HS, especially those reporting increased flares during menstruation or have features of Polycystic Ovarian Syndrome (PCOS).[34,44,45]

Surgery

Usually, surgical intervention is performed when pharmacologic care doesn't control the disease.[45] Incision & drainage (I&D) is recommended only upon abscesses, and though it can provide symptom relief, it doesn't alter the condition's recurrence rate or long-term prognosis.[46] I&D is associated with recurrence rates close to 100%.[45] Deroofing is another surgical approach that consists in opening sinus tract formations to both drain and heal lesions by secondary intention, and is preferred to simple drainage.45,46 Lastly, wide local excision (WLE) consists of a wide margin excision of all affected hair-bearing areas.46 It has been the cornerstone of conventional surgery, leading to no disease reoccurrence in the excision-performed areas.[45] Thus, it's considered the only potentially curative therapy for HS, with low recurrence rates ranging from 10-20%.[46]

Intralesional corticosteroid injections

Intralesional corticosteroid injections are recommended, either as a standalone treatment or as an adjunct to systemic therapies, for managing acute flares, refractory nodules, and sinus tracts in HS.[60] This therapy targets isolated HS nodules by activating glucocorticoid receptors and blocking pro-inflammatory cytokine production.[44] While its efficacy for acute HS flares is well-established, high doses and prolonged oral steroid regimens are not recommended due to the risk of flare-ups after tapering. However, the optimal dosages and volumes for intralesional steroid administration still need to be determined.[62]

Botulinum toxin injections

Botulinum toxin (BTX) injections are a beneficial treatment for HS.64 All patients across the pertaining literature tolerated the treatment well, with most experiencing clinical improvement and remission lasting between six to twelve months.[64,65] Notable benefits included significant reductions in HS lesions and patient-reported pain, healing of sinus tracts, and improvements in DLQI scores. The treatment was applied to areas such as the axillary, inframammary, groin, and gluteal regions, with repeated applications every three to ten months.[7] Although the role of BTX in HS pathogenesis remains unclear, it´s believed to aid by decreasing moisture, follicular rupture, and the spread of follicular material though the dermis.[64]

Lasers

Laser and phototherapy have shown effectiveness in preventing future outbreaks by targeting hair follicles and causing thermal damage to bacteria.19 Carbon dioxide (CO2) lasers were the first to be employed in HS treatment–utilized for excision, marsupialization, and vaporization of the affected skin–and appear to be linked to low recurrence rates. However, they may result in prolonged healing times.45

Prevention

Primary prevention involves proactive measures to prevent disease onset and is a widely implemented strategy in many well-characterized conditions. However, as of the time of writing this review, preventive approaches for HS are not feasible due to the incomplete understanding of its pathophysiology. In contrast, secondary prevention aims to limit disease progression and prevent complications after diagnosis.[25] In the context of HS, secondary prevention is effective and focuses on preventing the development of new nodules or fistulas following the onset of infundibulofolliculitis. It also involves interventions to prevent patients with Hurley stage I disease from advancing to more severe stages, such as stage II or III. [25]

A study assessing the impact of secondary preventive measures in HS patients demonstrated their effectiveness in reducing disease severity and progression.[25] Additionally, two patients with concurrent gluten-sensitive enteropathy experienced significant improvement in HS symptoms following the adoption of a gluten-free diet. Similarly, seven patients reported notable symptom relief after implementing a low-dairy, low-carbohydrate diet.[25] It is also important to note that shaving can irritate the skin and trigger the inflammatory cascade in HS, and should therefore be avoided.[25]

Additional lifestyle modifications recommended to reduce HS flare-ups include smoking cessation, engaging in physical activities that minimize excessive sweating, and adopting a Mediterranean diet. Supplementation with zinc (90–100 mg for 3–4 months), vitamin D, and myo-inositol has also been suggested to improve HS outcomes. [7]

Study Limitations

The limitations of this review include potential lack of generalizability due to variability in study populations and designs among the included articles. Selection bias may be present, as some relevant studies could have been missed, and publication bias may influence conclusions since studies with positive results are more likely to be published. These factors should be considered when interpreting the findings of this review.

Conclusion

Future research should prioritize evaluating the safety, efficacy, and optimal dosing of current off-label therapies, as well as novel agents targeting pathways such as IL-1, IL-12, IL-10, IL-36, C5a, IFN γ, and JAK. Further investigation into the pathophysiology of HS is also needed to clarify its etiology and molecular mechanisms. Improved recognition of HS is essential for timely diagnosis and treatment. This review aims to enhance awareness of the disease, its diverse clinical presentations, and available therapeutic options within the medical community.

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