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Beyond the skin: endocrine, psychological and nutritional aspects in women with hidradenitis suppurativa

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Abstract

Hidradenitis suppurativa (HS), also known as acne inversa, is a chronic inflammatory skin disorder that primarily affects body folds and the genital area, with a higher prevalence in women across Europe. The pathogenesis of HS involves a complex interplay of intrinsic and extrinsic factors, including genetics, immunity, hormones, and environmental influences. HS is frequently associated with a variety of comorbidities, such as metabolic, endocrine, and gastrointestinal conditions, as well as mental health disorders. Although the symptoms of HS are generally similar in both men and women, female patients may experience exacerbations of HS due to hormonal fluctuations during menstruation, pregnancy, breastfeeding, and menopause. These hormonal changes require special consideration by clinicians when managing HS in women. Due to its chronic nature and frequent flare-ups, HS significantly impacts patients' quality of life, affecting social interactions, emotional well-being, and psychological health. Women with HS may also experience sexual dysfunction, which is further exacerbated by emotional burdens such as shame, loss of femininity, and diminished intimacy. This review highlights key aspects of HS, extending beyond its skin manifestations to address endocrine, psychological, and nutritional aspects in the female population. It also underscores the importance of multidisciplinary collaboration in providing comprehensive care for women with this debilitating condition. Given the limited and largely off-label treatment options, a holistic approach is essential to ensure an appropriate management.

Keywords HS, Women, Psycho-dermatology, Metabolism, Nutrition and therapies

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Background

Hidradenitis suppurativa (HS) is a chronic, systemic inflammatory skin condition characterized by inflamed skin nodules, abscesses, pus discharge tunnels and scarring in areas such as axillae, under the breast, buttocks and genital region [1, 2]. HS is associated with a high comorbidity burden and the lowest quality of life (QoL) among all dermatological condition, yet it remains underrecognized and poorly understood [3]. Global prevalence is approximately 1% but may vary across countries [4]. HS can affect both genders, but in Europe the female-to-male ratio is 3:1 (varies between countries) [5, 6]. Reports also indicate that women often experience earlier onset of the disease, whereas men tend to have a more severe course [7, 8]. Hence, timely diagnosis in female patients is crucial to prevent the disease from progressing to an irreversible stage.

HS shows a multifactorial etiology, and genetic variations in γ-secretase gene can increase susceptibility to developing the disease in the presence of specific environmental triggers [9, 10]. It involves the occlusion of the pilosebaceous unit, leading to follicular rupture and subsequent inflammatory and immune responses [11]. Although the precise *primum movens* of HS pathogenesis remains unidentified, intrinsic factors such as incomplete X-chromosome inactivation in women and the influence of sex hormones are though to play a role in triggering the disease [12, 13]. In fact, certain genes located on the X chromosome, such as toll-like receptors (TLRs), can evade this inactivation and be overexpressed, leading to abnormal innate immune responses [13].

Following the initial pathogenic events, macrophages and dendritic cells (DCs) detect keratin fibers and other debris through TLRs. This detection leads to an increase in the production of tumour necrosis factor alpha (TNF-α), which recruits numerous immune cells into the skin [14]. Specifically, T helper (Th)1 and Th17 cells, which locally produce interferon gamma (IFN-γ) and interleukin (IL)-17 respectively, mainly contribute to hallmarks of HS [11, 14]. Furthermore, IL-17 promotes the production of IL-1β by keratinocytes through the activation of the inflammasome and caspase-1 [11]. Hormonal fluctuations during the menstrual cycle, pregnancy or menopause can further exacerbate these cascades and trigger HS flares [1, 15, 16].

Although HS is not an infectious or contagious disease, bacterial colonization in skin lesions is considered a secondary pathogenic event [11]. Notably, sex-related differences in the skin microbiome have been observed, which are associated with variations in sweating rates, skin pH, and hormone fluctuations [17–19]. Bacteria can also sustain the inflammation in hair follicles, once they are already blocked and damaged due to the condition [20]. Moreover, the presence of the anti-inflammatory mediator IL-10 in HS lesions, along with limited Th22 activity, may contribute to the reduction of antimicrobial peptides (AMPs), and thereby increase susceptibility to infections [11]. Indeed, HS lesions are often colonized by a wide range of opportunistic pathogens [21–24]. For instance, *Staphylococcus lugdunensis* is frequently detected in HS nodules, along with anaerobic bacteria such as *Prevotella* and *Porphyromonas* [22, 23]. These strains can form biofilms that exacerbate chronic inflammation and complicate the management of HS lesions [11]. In parallel, the gut microbiome of HS patients exhibits a reduced diversity compared to healthy subjects [25]. Specifically, it shows similarities to bacterial profiles linked to other inflammatory conditions, including Crohn's disease and inflammatory arthritis. Interestingly, metabolites derived from this dysbiotic gut microbiota may contribute to the development of HS and its related comorbidities [26–29]. In this regard, trimethylamine N-oxide (TMAO) metabolite was significantly elevated in the bloodstream of HS patients compared to controls, with ~ 70% of HS subjects were female. Moreover, a positive correlation between TMAO concentrations and the severity of HS was observed [30]. Therefore, nutritional interventions play a crucial role in supporting the health and diversity of the gut microbiota. A balanced diet that includes prebiotics, probiotics, and essential nutrients can help improve gut dysbiosis and potentially reduce inflammation [31], which may also aid in managing the severity of HS.

Other trigger factors of HS include smoking, obesity, and tight clothing, all of which can exacerbate the condition. Smoking contributes to inflammation, whereas obesity increases friction and sweating, both of which can worsen symptoms. Additionally, tight clothing can further irritate affected areas, leading to more frequent flare-ups [32]. A simplified overview of HS etiopathogenesis in women is illustrated in Fig. 1.

In this narrative review, we explored the current understanding of HS in women, extending beyond its skin manifestations to encompass psychological, endocrine, and nutritional aspects. Addressing these dimensions is important, as HS is highly disabling condition for women. In fact, they often experience difficulties with sexual intercourse due to genital inflammation or psychological barriers related to the clinical manifestations of the disease [1, 2]. Furthermore, the possibility of conception presents additional challenges, as pregnancy complications may arise from coexisting comorbid conditions [33, 34]. The risk of pregnancy loss, including miscarriage, may also be influenced by the localization and severity of HS [35–37]. This highlights the need for tailored support for women and collaborative care within a multidisciplinary team for this unique population.

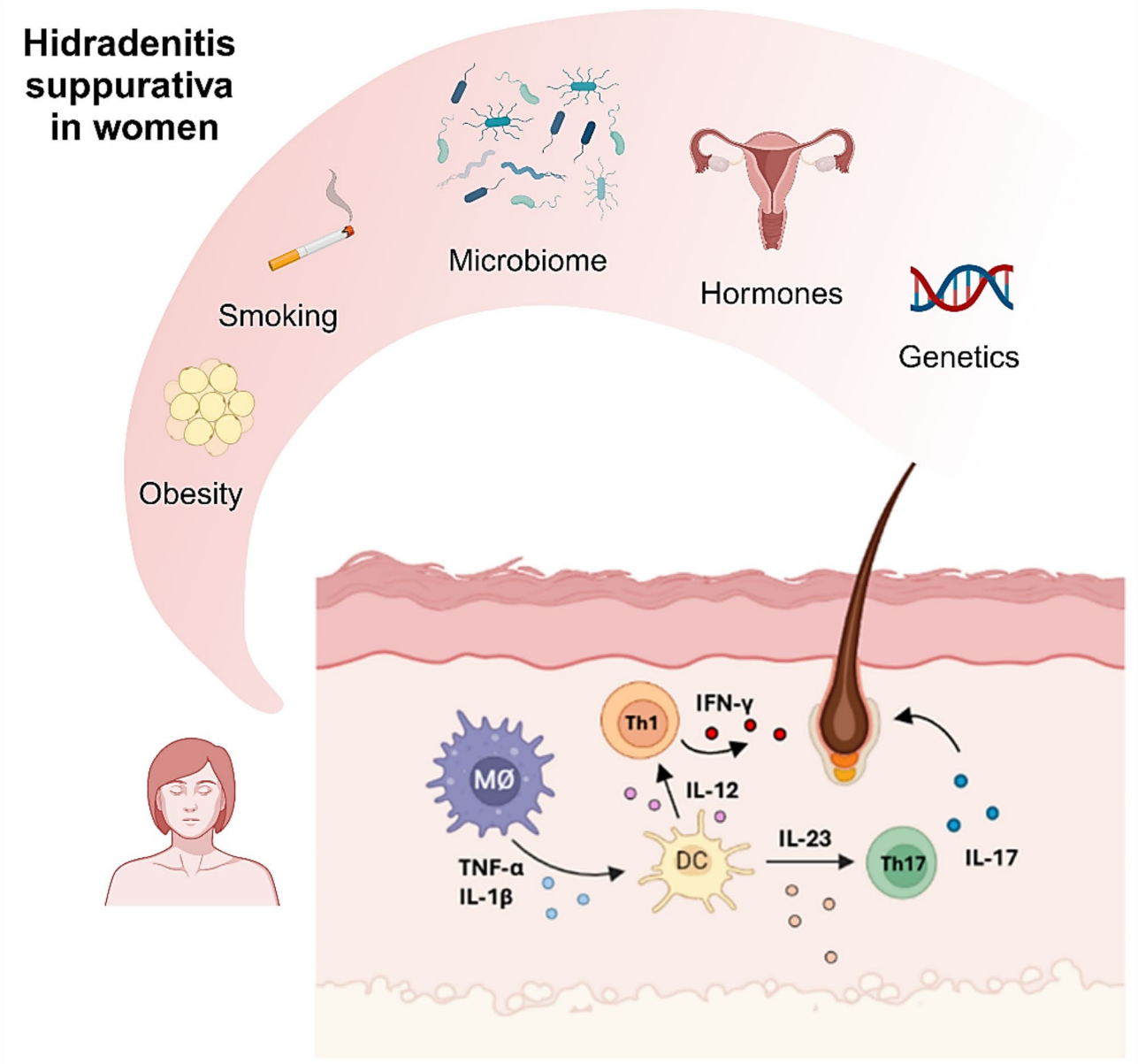

Fig. 1 A simplified overview of HS etiopathogenesis in women. Abbreviations: DC, dendritic cell; IFN-γ, interferon gamma; HS, hidradenitis suppurativa; IL, Interleukin; MØ, macrophage; Th, T helper cell; TNF-α, tumour necrosis factor alpha. Created with Biorender.com

Clinical diagnosis and drug treatment

Physical examination

The diagnosis of HS primarily relies on observations of lesions by dermatologists. Women typically exhibit greater involvement in the genitofemoral, axillary, and inframammary areas, whereas men more frequently show involvement of the axilla, perineum, perianal region, and buttocks [38]. Clinically, HS lesions may present as painful, inflamed papules and nodules, as well as abscesses, sinus tracts, and fistulas. A sex-disaggregated HS population analysis revealed that women tend to have more inflammatory nodules, while men more frequently exhibit fistulas [39]. Other lesions may also appear, including open comedones, fibrotic scars, and double-ended pseudocomedones [40]. Unfortunately, HS patients often experience fragmented care. Studies indicate an average global delay of around seven years from the onset of symptoms to an accurate diagnosis [41, 42]. Thus, increasing awareness and education about HS among healthcare providers is essential for facilitating timely diagnosis and comprehensive management of this debilitating condition. Gynecologists, in particular, can play a key role in the early HS identification in females. They may recognize the condition during pelvic and breast examinations and distinguish it from Bartholin cysts, Crohn's disease, folliculitis, granuloma inguinale,

lymphogranuloma venereum, and necrotizing fasciitis [38, 43]. To date, several clinical tools are available for assessing HS severity [44]. The Hurley staging system, which classifies HS into three stages (Fig. 2), is the most widely used tool for disease assessment during clinical practice. However, existing scoring systems for HS lack of precision and do not take into account subclinical inflammatory changes. Additional diagnostic tools, such as ultrasound to detect clinically invisible lesions and magnetic resonance imaging for pre-surgical mapping of fistulae, may also be useful [45, 46].

Metabolic, endocrine, and gastrointestinal comorbidities

HS is associated with a significant comorbidity burden beyond skin manifestations, including metabolic, endocrine, and gastrointestinal disorders [47].

Notably, nearly 50% of HS patients are classified as obese [48]. This association is thought to arise because adipose tissue functions as a hormonally active organ, releasing inflammatory mediators such as adipokines (e.g., chemerin, leptin, visfatin, omentin-1, and adiponectin), cytokines (e.g., IL-23, TNF-α, IL-6, and CCL2), and hormones [1, 49, 50]. Adipokines have various immunomodulatory effects that may influence the pathogenesis of HS [51, 52]. For instance, chemerin and leptin promote the polarization and recruitment of M1 macrophages to inflamed tissues whereas stimulating the proliferation of Th1 and Th17 cells [52–54]. Conversely, adiponectin reduces the production of TNF-α and IL-6 in monocytes/macrophages and encourages the synthesis of anti-inflammatory mediators, such as IL-10 and IL-1 receptor antagonist [55, 56]. Serological studies have shown dysregulated adipokine levels in HS patients [57–60]. Notably, serum levels of pro-inflammatory adipokines, like leptin, visfatin, and omentin-1, are significantly elevated in HS patients compared to controls [59, 60], whereas circulating adiponectin levels are lower [59], reflecting the systemic inflammation characteristic of HS patients. Although sex steroids influence adipose tissue functions [61], with estrogens promoting adipocyte proliferation and survival, studies on the relationship between adipokine levels and sex hormones across genders in HS are currently lacking.

Among endocrine disorders, the polycystic ovary syndrome (PCOS) is a common comorbidity found in HS women that increases the risk of type 2 diabetes mellitus due to its insulin-resistant nature [34, 62]. A recent Italian multicentre study involving 234 HS patients, mostly females (62%), found that 13.8% of the overall study population had PCOS (22.3% of women) [63], which is higher than the 9% prevalence observed in a U.S. population-based analysis of female HS patients [34]. Likewise, insulin resistance affects approximately 9.6% of individuals with HS [64], complicating weight loss, as excess blood sugar is stored as fat. Additional evidence of dysfunctional metabolism in HS comes from studies showing the

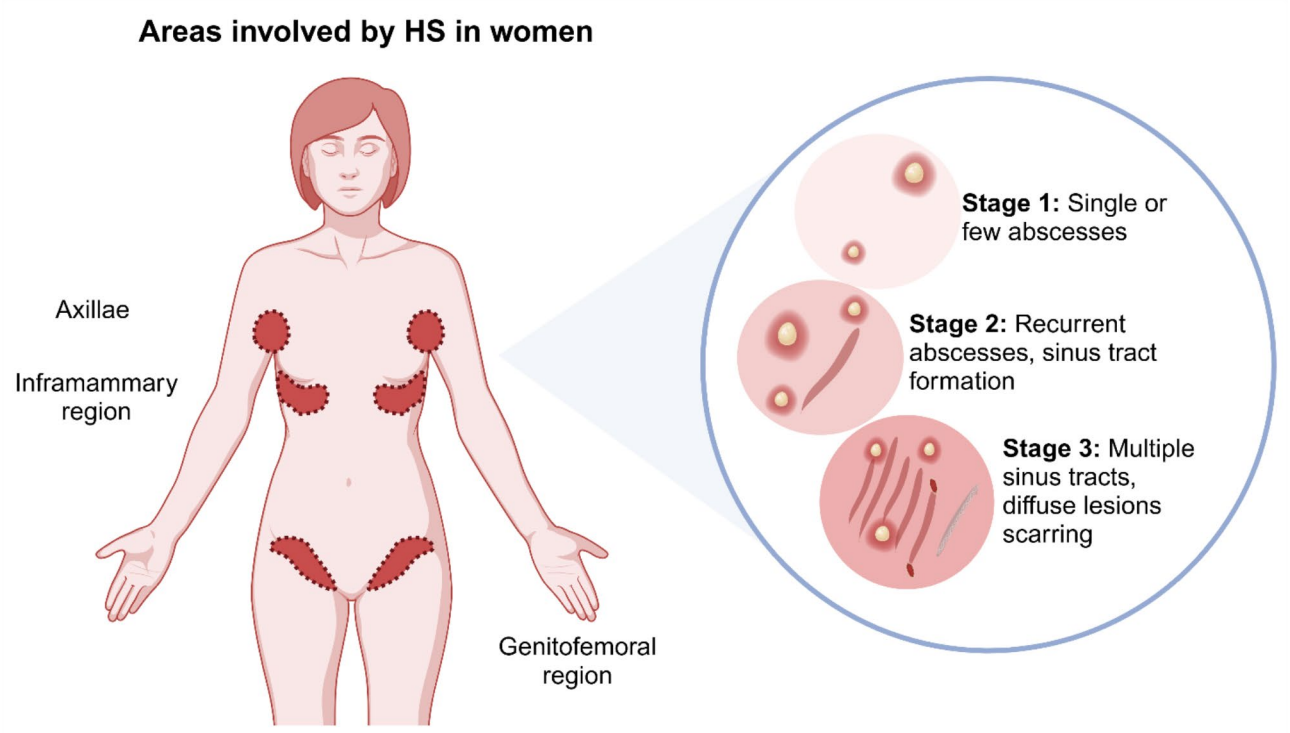

Fig. 2 Clinical localization and Hurley stage scoring of HS in women. Abbreviations: HS, hidradenitis suppurativa. Created with Biorender.com

positive effects of metformin on HS [65]. This antidiabetic drug has garnered attention for managing HS, particularly in mild-to-moderate cases [66–68]. Metformin works by inhibiting gluconeogenesis, increasing insulin sensitivity, and reducing androgen production [69, 70]. A prospective study involving 25 HS patients treated with metformin for 24 weeks found that 76% showed significant improvement, with Sartorius score decreasing from an average of 33.8 to 18.1 [71]. Similarly, a retrospective chart review of 53 HS patients treated with metformin showed a 68% clinical response, regardless of gender, even though 85% of the cohort was female [67]. To date, metformin is administered off-label in HS and is generally well-tolerated, with a safety profile that is considered acceptable for use during pregnancy and breastfeeding [72]. Long-term follow-up is crucial for refining treatment strategies and providing updated, evidence-based recommendations.

Concerning metabolic and endocrine dysfunction in HS, changes in sex hormone-binding globulin (SHBG) levels have been linked to obesity and insulin resistance [73–75], and observed in some women with long-standing HS [76]. Notably, reduced SHBG levels have been noted in obese women compared to their non-obese counterparts [77]. Since SHBG binds to circulating androgens, low levels can lead to elevated free androgen levels, potentially triggering conditions like PCOS. The precise mechanism by which obesity decreases SHBG remains unclear, though one possibility is that elevated insulin levels may inhibit SHBG production by liver [78]. Persistent high insulin levels may also directly affect the hypothalamic-pituitary-thyroid axis, impairing thyroid function and contributing to metabolic dysfunction [79]. A retrospective comparative cross-sectional study investigated thyroid function in 430 patients with HS (68% female) and 20,780 control subjects [80]. The results showed that HS is linked to hyperthyroidism, with lower levels of thyroid-stimulating hormone (TSH) and elevated levels of total triiodothyronine (tT3) compared to the control group. These results remained statistically significant even after accounting for additional factors such as body mass index (BMI), smoking, and oral contraceptive use. Indeed, smoking may impact thyroid function directly through thiocyanate, which has both pro- and anti-thyroid effects, or indirectly via sympathetic stimulation or immune disturbances [81]. Additionally, oral contraceptives can influence thyroid function by suppressing progesterone/estrogen levels [82]. Overall, these findings highlight the complex interplay between obesity, insulin levels, SHBG, and thyroid function in HS, underscoring the need for further investigation in this area.

Gastrointestinal comorbidities are also prevalent in patients with HS. A retrospective study found that younger, predominantly African American females with HS were more likely to have inflammatory bowel disease (IBD) [83]. Similarly, a study of 1,489 HS patients (77.3% female) revealed a significantly higher likelihood of IBD, with an odds ratio (OR) of 2.16 (95%CI, 1.40–3.34) [84]. A meta-analysis by Chen and Chi [85] further demonstrated an increased risk of ulcerative colitis (UC) in HS patients (pooled OR, 1.51; 95% CI, 1.25–1.82). Likewise, pediatric patients with HS had an approximately 1.8-fold higher incidence of UC [86]. Additionally, a cross-sectional study by Garg et al., involving over 51,340 HS patients (68.2% female) reported a notably higher likelihood of Crohn's disease, with an OR of 3.05 (95% CI, 2.87–3.25) [87].

HS and IBD share several overlapping features, including clinical symptoms such as fistula formation, as well as common genetic and immune system factors. Both diseases are associated with genetic loci like SULT1B1 and SULT1E1, and exhibit cytokine imbalances, including elevated levels of IL-1, IL-6, IL-17, IL-23, and TNF-α [88–90]. Consistently to their association, both conditions respond to TNF-α inhibitors [91–94].

Table 1 Therapeutic management of HS

Disease severity	Medical Treatment	Invasive Procedures
Mild	Antiseptic washes, topical treatments, short-course oral antibiotics, hormonal therapies, magnesium sulfate salt bath, oral vitamin D, oral zinc	Deroofing, intralesional triamcinolone, incision and drainage, local excision, laser treatment
Moderate-to-severe	Biologics, long-course oral antibiotics systemic immunosuppressants and immunomodulators, retinoids	Wide surgical excision (± acellular dermal matrices)

NB. All the treatments (either medical or surgical) performed in milder forms, can also be used for moderate-to-severe HS, either alone or in combination. At the same time, first-line treatments for more severe forms should be considered also for milder HS when refractory. Patient education is essential in all stages (hygiene measures, smoking cessation, laser hair removal, loose cotton clothing, and weight loss). Abbreviations: HS, hidradenitis suppurativa

Current pharmacological therapy and surgical procedures

The most common medications used for women with HS include topical and systemic treatments, antiseptics, and corticosteroids (Table 1). However, some treatments may be changed, especially when planning for pregnancy. Topical antibiotics, such as clindamycin 1%, metronidazole 0.75%, and erythromycin 2%, are considered safe for use during pregnancy due to their minimal systemic absorption, which limits their excretion into breast milk [95]. Similarly, antimicrobial washes like benzoyl peroxide and chlorhexidine are also deemed safe.

Regarding systemic antibiotics, particular attention must be paid to tetracyclines. These drugs are classified

as category D by the U.S. Food and Drug Administration (FDA) and are therefore absolutely contraindicated during pregnancy and lactation. In contrast, clindamycin, metronidazole, erythromycin, and rifampin are considered relatively safe for patients of childbearing potential and lactating women. Dapsone is another oral antibiotic which can be used during pregnancy but is usually stopped at least a month before delivery to reduce the risk of jaundice and anaemia in the baby [96, 97].

Topical and intralesional corticosteroids can be used for local management of the disease and are regarded as the best choice for managing disease flare-ups. Isotretinoin and hormonal therapies, such as oral contraceptive pills and spironolactone, are often employed in the treatment of HS but are contraindicated during pregnancy and lactation due to their potential risks to fetal development and nursing infants [98].

So far, the safety of newer biological therapies during pregnancy is less established compared to antibiotics. These drugs, including etanercept, adalimumab and infliximab are categorized as FDA pregnancy category B, indicating a lack of well-controlled human studies. These agents may be safely used before the third trimester, as maternal IgGs are typically transferred via the placenta only later in pregnancy. Breastfeeding while using TNF-α inhibitors is likely safe due to negligible transfer into breast milk and the drug's breakdown by the infant's gastrointestinal tract. Additionally, certolizumab pegol is considered relatively safe during pregnancy due to its minimal placental transfer, making it a preferred biologic for pregnant patients requiring treatment for inflammatory conditions [99]. It is important to note that biologics can be safely and effectively employed in patients with HS who are of childbearing potential, but extensive counselling before drug administration and adequate planning for a potential pregnancy are essential. Of note, biologics, such as TNF-α and IL-12/23 inhibitors, have shown promise in improving both the skin manifestations and metabolic comorbidities, including obesity, insulin resistance, and dyslipidemia [100, 101]. TNF-α inhibitors reduce systemic inflammation, which is often linked to metabolic dysfunction [102, 103]. Studies suggest that treatment with adalimumab may also improve metabolic profile in the setting of inflammatory dermatoses [102, 104, 105]. These data underscore the potential for biologics in the setting of HS and HS-related comorbidities.

As for non-pharmacological therapeutic strategies, surgical treatment is generally reserved for severe, persistent cases that do not respond to medical therapies such as antibiotics and biologics [106]. With specific regards to HS in women, the timing of surgery should be carefully considered for patients planning pregnancy, emphasizing safer alternatives and collaborative care to ensure the best outcomes for both mother and child [107]. Wide excision is required for extensive chronic lesions, as it removes affected tissue and minimizes the risk of recurrence. Incision and drainage are performed for abscesses to alleviate pain, although this procedure is not curative. Necrotic tissue debridement may also be necessary to aid healing and control infection. In complex cases, reconstructive surgery, such as skin grafts, may be required [108]. While surgical interventions can provide significant relief, they carry risks, including infection, scarring, and complications with wound healing [109]. Given that HS is a chronic condition, recurrence is still possible after surgery. Also, childbirth can present unique challenges in women with HS. Scar tissue in the genital area can make vaginal delivery more challenging, and a caesarean section could increase the risk of forming new nodules nearby, leading to additional discomfort afterward [110, 111]. Thus, a multidisciplinary approach involving dermatologists, surgeons, and plastic surgeons is essential for an optimal management.

Endocrine aspects

Endocrine dysfunction is a key aspect of HS, closely linked to both dermatological and psychological domains.

The skin, as the body's largest organ, functions not only as a target for various hormones but also as an endocrine gland itself. The pilo-sebaceous unit, composed of hair follicles and sebaceous glands, plays a pivotal role in producing and metabolizing steroid hormones, including those related to stress responses and sex [112]. Additionally, the overall endocrine dysfunction may also result in elevated levels of TSH, thyroid-releasing hormone (TRH), and adrenocorticotropic hormone (ACTH), sustaining vicious cycles that underlie chronic stress and metabolic disorders in HS patients [80, 113].

Concerning chronic stress, it can trigger neuroendocrine responses, increasing serum corticotropin-releasing hormone (CRH) and cortisol levels, which exacerbate HS symptoms [114–116]. In parallel, metabolic dysfunctions are strongly sustained by cortisol, which increases gluconeogenesis and glycogenolysis, two processes that elevate blood glucose levels [117].

On the other hand, sex hormones, both male (primarily androgens) and female hormones (e.g., estrogens, and progesterone), are also believed to contribute to HS disease. Accordingly, Gauntner [118] and Zouboulis et al. [119] reported increased androgen receptor (AR) transcriptional activity in HS lesions and upregulation of androgen-controlled genes in women. Mechanistically, androgens have been suggested to activate the inflammasome, leading to the activation of caspase 1 and thereby the expression of pro-inflammatory cytokines typical of HS [120]. In clinical setting, androgenic gender-affirming therapy has shown to exacerbate HS in transgender men

[121]. Conversely, antiandrogenic drugs like finasteride and spironolactone have demonstrated a certain degree of effectiveness in the treatment of HS in female patients, even compared to conventional antibiotic treatments [122–124].

The female hormones, estrogen and progesterone, play a significant role throughout the lifespan of women with HS, from puberty to old age [111]. Numerous studies [1, 16, 118, 125–128] have highlighted a connection between hormonal fluctuations—such as those occurring during the peripubertal and perimenstrual periods, as well as pregnancy—and HS flares, although the exact underlying mechanism remains unclear.

The onset of HS during puberty and the occurrence of perimenstrual flares further underscore the impact of sex hormones on the disease [1]. Premenstrual HS flares are observed in approximately 43–63% of patients [16, 126, 127]. On the other hand, contrasting data have been published regarding pregnancy, as it involves increased prolactin levels as well as both anti-inflammatory and proinflammatory states that may influence the progression of HS [125, 128].

Importantly, although most women with HS have normal fertility, they have a lower likelihood of having a live birth compared to women without HS (52.0% vs. 70.74%, respectively) [129]. They are also more likely to undergo elective termination during pregnancy. Additionally, a case report described a miscarriage in a 40-year-old woman with severe HS and vulvar infections, highlighting a serious but rare risk associated with advanced HS [35].

Finally, menopause has traditionally been believed to be associated with a reduction in the severity of HS [128], as estrogen levels decline and stabilize at lower levels. However, conflicting evidence suggests that HS symptoms may remain stable or even worsen during menopause [93, 128]. Furthermore, menopause has been linked to an increase in numbers of lesions, which can further impact patients' QoL [128].

From a mechanistic standpoint, the role of sex hormones in the pathogenesis of HS is not yet fully understood, although some insights have been gained. Prolactin (PRL) functions not only as a hormone but also as a cytokine, modulating the immune system, primarily by inhibiting the negative selection of autoreactive B lymphocytes [130, 131]. Similarly, estrogen and progesterone can influence the immune system. Estrogens, for example, stimulate the maturation of dendritic cells (DCs) and the differentiation of T cells [15]. They also promote the proliferation, survival of lymphoid T cells, and the expression of pro-inflammatory cytokines, particularly IFN-γ, which may help explain the elevated IFN-γ levels observed in female patients with HS [12]. In contrast, progesterone limits DC activation, reduces CD4 + T cell stimulation, and increases the production of anti-inflammatory mediators, particularly IL-10 [132, 133]. Future studies investigating the impact of sex hormones on HS pathogenesis could provide valuable insights into the complex mechanisms underlying this debilitating skin disorder.

Given the unpredictable nature of HS and hormonal fluctuations throughout women's life stages, patients should be encouraged to closely monitor their disease activity with the assistance of specialists, such as endocrinologists and dermatologists. This approach can help identify flare-ups early, allowing for timely treatment adjustments to better manage hormone-dependent symptoms and minimize complications.

Psychological aspects

Patients with HS may face both physical and psychological challenges, as the pain and discomfort can significantly impact daily activities, sleep quality, and productivity at work or school [134, 135].

Moreover, the stigma associated with cutaneous lesions in visible areas, along with their suppurative nature, can hinder social interactions and self-esteem, leading to feelings of isolation [136]. Women may also experience sexual difficulties due to embarrassment and fear of rejection [137]. In the study by Fisher & Ziv [138] on women's self-perception and life experiences, the most troubling issues were the somatic symptoms, particularly pain, alongside the emotional burden of shame, and the loss of femininity and intimacy.

Chronic stress in these patients can activate the hypothalamus-pituitary axis, leading to increased cortisol production [115]. This, in turn, exacerbates systemic and skin inflammation and creates a feedback loop between HS symptoms and psychological/psychiatric issues [114]. Conversely, the inflammatory cytokine milieu associated with the HS condition can contribute to the worsening of mental disorders [114, 139].

Importantly, recognizing the hormonal impact and understanding the psychological challenges women face at different life stages underscore the need for integrated clinical management of female HS patients. Rising neuroendocrine modulator PRL levels, potentially sustained by psycho-emotional stress [140], during pregnancy and the postpartum period can worsen HS flare-ups [1, 141, 142]. Additionally, pregnancy itself presents psychological challenges, including stress and body changes such as weight gain, which can exacerbate HS by increasing friction in intertriginous areas [143, 144]. Additionally, breastfeeding women with HS may face extra challenges, particularly if lesions are present on the breasts.

During the postpartum period and breastfeeding, the focus should be on safely managing HS according to the mother's preferences, supporting recovery from

Table 2 Dietary patterns and inteventions in HS

Type of Study	Intervention	Sample	Duration	Main result	Reference
Dietary pattern	Mediterranean diet	82 subjects (41 HS vs. 41 Control group)	Cross-sectional observational study	High adherence to the Mediterranen diet was associated with low HS severity	Barrea et al., 2018 [171]
	Very low-calorie ketogenic diet (VLCKD)	12 women with HS and overweight or obesity	28 days	VLCKD improvements HS severity and associated metabolic markers	Verde et al., 2024 [175]
	Low dairy diet	47 subjects	3 months	83% of participants reported disease improvement	Danby, 2015 [176]
Foods	Wheat and brewer's yeast	12 subjects	12 months	Resolution of lesions, improvement in QoL	Cannistrà et al., 2013 [177]
Supplements	Vitamin D	14 subjects	6 months	↓ number of nodules at 6 months	Guillet et al., 2015 [178]
	Zinc	66 subjects	3 months	↓ number of inflammatory nodules, flares and erythema	Hessam et al., 2016 [179]

Abbreviations: HS, hidradenitis suppurativa; QoL, quality of life; VLCKD, very low-calorie ketogenic diet

childbirth, and providing psychoeducational and psychosocial support [111]. Key management goals and clinical considerations during the postpartum and breastfeeding period for women with HS include being aware of pregnancy-related outcomes. Women with HS are at higher risk for adverse maternal, pregnancy-related, and neonatal outcomes, such as gestational hypertension and diabetes, caesarean section, spontaneous abortion and preterm birth. Of note, HS results as an independent risk factor for these complications [145].

Clinicians should be familiar with the safety profile of HS treatments, including psychotropic medications (see the next paragraph), during lactation to minimize disruption of breastfeeding [95]. Altogether, it would be important not only to focus on the physical manifestations of HS while neglecting the psychological aspects, as this could create a vicious cycle where stress triggers flares, and HS flares, in turn, exacerbate stress.

Psychiatric comorbidities, psychological interventions, and psychotropic drugs

Patients with HS may be at an increased risk for psychiatric comorbidities, in addition to the physical comorbidities affecting organ systems. Shavit et al. [146] studied 3,207 patients with HS and 6,412 matched controls, revealing a higher prevalence of mental health disorders in HS cohort. These included depression disorders, anxiety disorders, bipolar I and II disorders, post-traumatic stress disorder and psychoses. Similarly, a Finnish study [147] using data from the Finnish Health Care register found that HS is associated with a significant prevalence of psychiatric disorders, with 24.1% of patients diagnosed with at least one mental disorder. Major depression was found in 15.3%, anxiety disorders in 6.9%, and psychotic disorders in 4.7%. Women with HS had a higher overall prevalence of mental disorders (25.5%) compared to men (22.0%). However, men exhibited stronger associations with anxiety disorders (OR 1.70) and schizophrenia (OR 1.88). This highlights the gender differences in the psychiatric burden of HS, with women experiencing more depression and anxiety, while men faced higher risks for specific disorders. Furthermore, a Danish cross-sectional analysis by Thorlacius et al. [148] found that patients with HS have a significantly higher risk of suicide. This finding is further supported by a study from Tiri et al. [149], which specifically examines the elevated suicide risk among women with HS. Tiri's study reports a 2.4-fold increase in suicide risk for women with HS compared to the general population, with 63.6% of suicides occurring in women. While depression is a key factor, it mediates only 11.7% of suicides in women with HS, compared to 44.9% in men. Collectively, these findings underscore the urgent need for mental health support and awareness in HS patients, emphasizing the profound impact of this chronic condition on mental well-being.

There are few studies on psychological interventions and psychotropic drugs in HS, despite the high prevalence of psychiatric disorders and significant impact on QoL [150].

Psychological therapies, such as cognitive behavioural therapy (CBT) and compassion-focused therapy, may be beneficial as they have been for other skin conditions [151]. CBT helps reduce pain and psychological distress by emphasizing the relationships between chronic pain, thoughts, emotions, and behaviors, while compassion-focused therapy can enhance self-acceptance and reduce feelings of shame. Additionally, group therapy sessions for HS patients, led by a healthcare professional, may reduce feelings of shame and stigmatization while increasing social acceptance. Mindfulness-based stress reduction (MBSR) can also be beneficial in this context [150].

Accurately assessing these connections is crucial, as HS is frequently associated with mental disorders, but these comorbidities are often underdiagnosed. Psychiatric conditions are present not just additional medical issues; they are integral to the overall management of the illness. Furthermore, preexisting psychiatric conditions may affect management, influencing factors such as adherence, empowerment, illness awareness, acceptance, and changes in dysfunctional habits. However, it is important to also consider the possible impact of psychotropic drugs on both the course of HS and hormonal variation. For example, antipsychotic drugs are linked to hyperprolactinemia [152], which can, in turn, can exacerbate HS symptoms [1]. In particular, the use of lithium, a common medication for bipolar disorder, has been reported to induce or exacerbate HS [153]. This may help explain the observed association between HS and bipolar disorder [154].

Other therapies for treating both depression and pain include gabapentin and pregabalin, which can also be used in combination with nonsteroidal anti-inflammatory drugs (NSAIDs) to enhance pain relief. Pregabalin is often preferred by patients due to its lower incidence of drowsiness compared to gabapentin. Duloxetine, as a serotonin and norepinephrine reuptake inhibitor (SNRI), is particularly noted for its dual role in managing both pain and depression, making it especially relevant for HS patients who may experience both conditions. Additionally, combining duloxetine with gabapentin or pregabalin can provide synergistic effects in pain management. In cases of depression, duloxetine should be tried first, followed by venlafaxine as a SNRI, unless the patient's insurance does not cover duloxetine. Of note, venlafaxine is associated with a poorer side effect profile compared to duloxetine. Common side effects may include nausea, dizziness, and somnolence [155].

It is important to note that, despite being one of the most painful conditions treated by dermatologists, HS lacks an accepted pain treatment algorithm [156]. Notably, patients with HS may be at greater risk for alcohol misuse, followed by cannabis and opioid use [157]. Opioids are frequently prescribed for pain management, but their use during breastfeeding should be avoided due to the risk of respiratory and central nervous system depression in the infant [158].

Altogether, the presence of psychiatric disorders should be considered in clinical decision-making, therapeutic management, and patient monitoring to develop optimal strategies for prevention and combined treatments. While guidelines address the psychological implications and psychiatric comorbidities, they should also account for the impact of concomitant pharmacological therapies on disease progression and hormonal variations. Referrals to psychiatrists and psychologists are essential components of a multidisciplinary approach recommended for managing HS patients who experience psychiatric disorders.

Nutritional aspects

Women with HS often experience a high BMI, which is associated with dietary and lifestyle factors [159], and is likely influenced by hormonal changes. However, research on HS has revealed that female patients often avoid certain foods (e.g., dairy, refined sugars and alcohol) in hopes of alleviating their symptoms, as reported in a survey study [160]. In fact, the consumption of Western diet characterized by high-fat dairy products and refined simple carbohydrates, has been linked to HS [161]. Dairy products contribute three main factors: casein, whey, and natural androgens, along with their precursors and growth factors [161, 162]. These components can promote follicular duct blockage, leading to leakage and rupture of genetically susceptible pilosebaceous units, which are central to HS symptoms [163]. Furthermore, this diet predisposes patients to obesity, diabetes, and metabolic syndrome, thus adding the endocrine factor to this multifactorial disease [164].

Casein increases insulin-like growth factor 1 (IGF-1) levels, whereas whey is associated with hyperinsulinemia. Both mechanisms activate androgen receptors, potentially exacerbating HS [162]. Notably, a low-glycemic diet, including the elimination of sugary drinks, has shown potential benefits for HS patients [161]. However, restricting only dairy without additional dietary modifications, has not consistently prevented disease worsening compared to controls not following a dairy-free diet [165]. Wheat and brewer's yeast have been also identified as triggers for inflammation in HS patients [166]. These ingredients, commonly found in processed foods like bread, pizza dough, pastries, cakes, beer, wine, and cheese, contain *Saccharomyces cerevisiae*, a yeast that produces fermentation alcohol and carbon dioxide [167, 168]. Studies suggest that restricting wheat and brewer's yeast can lead to symptom control and significant QoL improvements over a year [161, 167, 169].

Several dietary strategies are currently being explored to enhance the clinical condition of HS. However, evidence regarding the role of specific dietary and lifestyle factors as complements to traditional HS treatments across genders remains scarce [167]. The Paleo and elimination diets typically exclude gluten, dairy, grains, legumes, and nightshades, aiming to enhance gut microbiome diversity [161, 170]. An even stricter auto-immune protocol diet, which follows the Paleo diet approach, also includes limitations on eggs, seeds, nuts, and artificial sweeteners [161]. While elimination diets may offer symptom relief, they are often highly restrictive and challenging for patients to maintain long-term. These diets

Table 3 The role of the multidisciplinary team in the management of HS in women

Specialist	Role
Dermatologist	- Diagnosis of HS and clinical severity scoring - Suggest the appropriate treatment for pregnant or breastfeeding patients - Refer the patient for surgical treatment when indicated - Refer the patient to an endocrinologist for additional investigations - Refer to general practitioner for smoke cessation and obesity management - Refer to physiotherapist for adequate training program
Surgeon	- Choice of the surgical technique: local incision and drainage, single-nodule excision or wide surgical excision - Post-surgical wound management
Endocrinologist	- BMI measurement - Hormonal disturbance assessment - Glucose glucose tolerance test, evaluation of other blood tests (hemoglobin A 1c, fasting glucose, basal insulin) - Assessment of dyslipidemia (lipid panel), fatty liver (liver function test), hypertention (blood pressure measurement) - Management of metabolic impairment in patients with PCOS
Psycologist	- Evaluation of mental health symptoms - Emotional self-regulation - Goal setting for lifestyle modification - Education and support to improve therapy adherence - Psycho-neuro-endocrino-immunology evaluation - Psychosexual counselling
Nutritionist	- Weight trend assessment (follow-up from baseline) - Provide education regarding healthy eating habits

NB Collaboration between the dermatologist and gynecologist/obstetrician is crucial for managing HS during pregnancy. Furthermore, involving a psychiatrist is important when psychiatric comorbidities are present. Abbreviations: BMI, body mass index; HS, hidradenitis suppurativa; PCOS, polycystic ovary syndrome

seek to identify dietary triggers by systematically reintroducing foods after an elimination phase while monitoring HS symptom flares. This tailored approach may empower patients to better manage their condition. However, larger, randomized controlled trials are needed to further evaluate the dietary impact on HS disease, and treatment outcomes. Among dietary patterns, the Mediterranean diet has been shown to alleviate the severity of HS [171]. This diet is rich in olive oil, which provides omega-3 fatty acids known for their anti-inflammatory, antioxidant, and anti-fibrotic properties, primarily through the inhibition of TLRs, particularly TLR2 and TLR4 [172, 173].

Other interventions that have shown to reduce HS severity include ketogenic therapy, and low-dairy diet, as well as dietary supplements such as zinc and vitamin D (Table 2). Notably, zinc is an inhibitor of 5α-reductase isoenzymes, thereby delivering both anti-inflammatory and anti-androgenic effects [161] whereas vitamin D plays a crucial role in immune regulation and bolstering defence against infections through the stimulation of AMPs in sebocytes [174]. A number of other supplements have been linked to HS, such as curcumin, copper, vitamin B12, ginger, and resveratrol. However, research on these supplements is still limited [164].

In addition to diet and a healthy lifestyle, maintaining optimal body weight is important for disease management. An overweight body is more prone to increased friction from skin folds and a humid skin milieu, which favours the thriving and growth of microbes and promotes low-grade systemic inflammation, which are the factors leading to the worsening of the clinical picture of HS. Clinicians must be empathetic when recommending weight-loss strategies, considering the psychosocial burden of HS and the stress and frustration that can arise also from the inability to afford healthy food options [164]. A holistic approach to managing HS allows for addressing the broader aspects of the disease. Table 3 outlines key considerations for managing female patients with HS within a collaborative framework.

Conclusions

HS is a non-communicable inflammatory skin disease that is more prevalent in women across European countries. It disrupts the immune system and can lead to complications affecting multiple organ systems, with increasing evidence linking it to metabolic, endocrine, gastrointestinal, and psychiatric comorbidities, especially in women. Female patients with HS also face unique challenges due to hormonal fluctuations during life stages such as menstruation, pregnancy, breastfeeding, and menopause. Therefore, effective management requires a multidisciplinary approach, including dermatology, endocrinology, psychology, and nutrition services. Strong relationships with gynecologists are essential for managing HS in women of childbearing age, while psychiatrists should be involved when psychiatric comorbidities are present. Close collaboration among specialists is crucial to prevent complications. Regular consultations with a dermatologist are key for managing flare-ups and adjusting treatment plans. For patients with obesity, weight reduction can help reduce friction and alleviate flare-ups, while psychotherapy provides essential emotional

support. As research continues, developing clinical protocols that consider gender differences in disease management is vital, and clinicians must stay updated to ensure comprehensive care that addresses every aspect of this complex condition.

Abbreviations

ACTH	Adrenocorticotropic hormone
AMPs	Antimicrobial peptides
AR	Androgen receptor
BMI	Body mass index
CI	Confidence interval
CRH	Corticotropin-releasing hormone
DC	Dendritic cell
FDA	Food and drug administration
FSH	Follicle-stimulating hormone
hCG	Human chorionic gonadotropin
HS	Hidradenitis suppurativa
IBD	Inflammatory bowel disease
IGF-1	Insulin-like growth factor 1
IFN-γ	Interferon gamma
IgG	Immunoglobulin G
IL	Interleukin
LH	Luteinizing hormone
MØ	Macrophage
NSAIDs	Nonsteroidal anti-inflammatory drugs
OR	Odds ratio
PCOS	Polycystic ovary syndrome
PRL	Prolactin
QoL	Quality of life
SHBG	Sex hormone-binding globulin
TLRs	Toll-like receptors
TMAO	Trimethylamine N-oxide
TRH	Thyroid-releasing hormone
TSH	Thyroid-stimulating hormone
tT3	Total triiodothyronine
TH	T helper
TNF-α	Tumour necrosis factor alpha
UC	Ulcerative colitis
VLCKD	Very low-calorie ketogenic diet

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Author contributions
Emanuele Scala drafted the manuscript and revised the final version. The research was conducted by an international team of specialists led by Anna Dattolo, a psychologist with expertise in psychodermatology, along with dermatologist Monica Torres and clinical nutritionist Evelyn Frias-Toral. Monica Torres also designed the figures. The literature review and manuscript writing involved contributions from Anna Dattolo, Monica Torres, Evelyn Frias-Toral, Alessia Paganelli, Mariana Zhang, Stefania Madonna, Laura Mercurio, Gabriela Cucalón, Federico Garbarino, Cristina Albanesi, and Emanuele Scala. All authors have read and approved the final version.

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