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Hidradenitis Suppurativa as a Systemic Inflammatory Disease: Comorbidities and the Need for a Multidisciplinary Approach

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Abstract

Hidradenitis suppurativa (HS) is a chronic, recurrent inflammatory disease that has traditionally been considered a localized skin disease. However, more and more epidemiological, immunological, and clinical evidence supports its identification as a systemic inflammatory disease. This article review summarizes the current evidence regarding the multisystemic character of HS, with an emphasis on the burden of comorbidities, pathophysiological links to systemic inflammation, and consequences for clinical management. The available literature indicates that HS is associated with a significantly increased number of cardiometabolic diseases, cardiovascular events, inflammatory bowel disease, arthritis, mental disorders, polycystic ovary syndrome, non-alcoholic fatty liver disease, and selected malignancies. These correlations seem to result from common inflammatory pathways, including TNF-α, IL-17, IL-1, and IL-23 signaling, as well as chronic systemic inflammation and metabolic disorders.

Recognizing HS as a systemic disease has important practical implications, involving the need for screening for comorbidities, risk assessment, and multidisciplinary care. Overall, recent evidence indicates that HS should be considered not only as a dermatological condition but also as a complex systemic inflammatory disorder requiring thorough clinical evaluation and an integrated long-term medical approach.

Keywords: **hidradenitis suppurativa; acne inversa; psychiatric comorbidity; systemic inflammation; comorbidities; cardiometabolic risk; inflammatory bowel disease; inflammatory arthritis; polycystic ovary syndrome; non-alcoholic fatty liver disease; multidisciplinary management**

1. INTRODUCTION

Hidradenitis suppurativa (HS), also known as acne inversa, is a chronic, recurring inflammatory disease of the hair follicles. Its characteristic features include painful inflammatory nodules, abscesses, and fistulas localised mainly in the regions prone to chafing, such as the armpits, groin, and anogenital area [1].The disease leads to progressive fibrosis of the tissues, chronic pain, and significant worsening of the quality of patients' lives, sometimes compared with other severe chronic diseases [3].

For many years, HS was considered a skin disease with a local manifestation. However, over the last ten years, there has been a significant shift in how this disease is viewed — more and more epidemiological, immunological, and clinical data show that HS should be considered a systemic disease associated with chronic inflammation [1],[2]. A case-control study of clinically verified HS cases has shown a significantly higher burden of comorbidities than in the control population. It is one of the first strong arguments for the systemic nature of the disease [2]. Current data indicate that HS belongs to the group of immune-mediated inflammatory diseases (IMID). It shares common pathogenic mechanisms with psoriasis, inflammatory bowel diseases, and spondyloarthropathies. Dysregulation of the immune response is critical, as it involves inflammatory pathways that depend on TNF-α, IL-17, and IL-1, among others. It leads to prolonged chronic inflammation, also outside the skin [9]. There is more and more evidence to suggest that the systemic inflammatory component may be responsible for the increased number of metabolic, cardiovascular, and autoimmune diseases observed in patients with HS [1].

The clinical consequences of this change in thinking are fundamental. Patients with HS have an increased risk of numerous comorbidities, including metabolic syndrome, cardiovascular disease, inflammatory bowel disease, inflammatory joint disease, and psychiatric disorders, highlighting. It shows the need for a clear diagnostic and therapeutic approach [29].Current North American guidelines highlight that the assessment of patients with HS should include not only the classification of skin lesion severity but also systematic screening for comorbidities and assessment of systemic risk factors [3]. Despite a growing number of publications, significant gaps remain in knowledge of the actual systemic extent of HS. Data are often heterogeneous, and recommendations for screening for comorbidities are not standardized. As a result, many patients remain undiagnosed for comorbidities, which may affect prognosis and mortality.

The aim of this paper is to present current evidence demonstrating that hidradenitis suppurativa is a systemic inflammatory disease and to provide a summary of its comorbidities and clinical implications.

2. METHODS

This paper is a narrative review of the literature, grounded in the principles of Evidence-Based Medicine (EBM). Our aim was to evaluate the available evidence on the systemic aspect of hidradenitis suppurativa (HS) and its associated comorbidities. The literature review was based on PubMed/MEDLINE and Google Scholar databases, with publications available until January 2026. The search was based on combinations of keywords: “hidradenitis suppurativa,” “systemic inflammation,” “comorbidity,” “metabolic syndrome,” “cardiovascular disease,” “inflammatory bowel disease,” “arthritis,” “depression,” “NAFLD,” and their variants. In addition, the bibliographies of key review articles and current clinical guidelines were assessed to identify additional publications. The analysis primarily included publications with the highest level of scientific credibility, including systematic reviews and meta-analyses, randomized clinical trials, large cohort and population studies, and current clinical guidelines on HS [4]. Single case reports and small case series were excluded. In cases of multiple studies on the same topic, the most recent meta-analyses or the largest-sample studies were preferred.

3. EPIDEMIOLOGY AND RISK FACTORS

The estimated global prevalence of HS is approximately 0.3–1% of the general population, but this figure varies depending on the research methodology and geographical region [5]. Meta-analyses show that the disease is more common in women than in men, with symptoms typically appearing in young adulthood, most often between the ages of 20 and 40 [6]. A significant epidemiological problem remains with the delay in diagnosis, which, according to observational studies, averages 7–10 years from the onset of the first symptoms. This delay appears to be associated with disease progression and an increased risk of developing comorbidities [1].

One of the most important arguments in favor of the systemic character of HS is the strong association between the disease and systemic risk factors, such as obesity or nicotine use. Obesity is one of the best-documented risk factors for HS. Meta-analyses have shown a significantly higher risk of developing the disease in people with elevated BMI, as well as a higher prevalence of metabolic syndrome, insulin resistance, and type 2 diabetes [7]. Adipose tissue can act as an endocrine organ and produce pro-inflammatory cytokines, which may worsen the chronic inflammation seen in HS. Another strongly related, modifiable risk factor for HS is smoking. Epidemiological studies show a higher

percentage of smokers among HS patients compared to the general population [5]. Nicotine can promote hyperkeratinization of hair follicles and modify the immune response, which can play a role in the persistence of inflammation.

Some patients' family history of the disease suggests a genetic component, for example, mutations affecting the function of the γ-secretase complex. Furthermore, the higher rates of HS in women and exacerbations associated with the hormonal cycle indicate the possible role of hormone-related factors in the pathogenesis of the disease [1]. Moreover, there are significant similarities to other immune-mediated inflammatory diseases, such as psoriasis and inflammatory bowel disease. Common risk factors — obesity, smoking, and chronic inflammation — suggest a shared pathophysiological mechanism, recognized as “metabolic inflammation”[1].

These associations may lay the foundation for the concept that HS is not an isolated skin disease but part of a wider spectrum of inflammatory diseases linked by common metabolic and immune mechanisms.

4. PATHOPHYSIOLOGY LINKING SKIN DISEASE TO SYSTEMIC INFLAMATION

The key event initiating HS development is hair follicle occlusion caused by abnormal keratinization of the epithelium. This leads to its enlargement and rupture, followed by the release of keratin, bacteria, and cellular elements into the surrounding tissues, which triggers a strong inflammatory response [8]. This results in an inflammatory response that is both innate and adaptive and involves neutrophils, macrophages, and T lymphocytes. This leads to the formation of abscesses, fistulas, and chronic tissue damage characteristic of advanced stages of the disease. Immunological studies have shown increased expression of a broad spectrum of proinflammatory cytokines in HS lesions and in the peripheral circulation of patients. A particularly important role is attributed to the axis: TNF-α, IL-17, IL-1β, and IL-23 [23]. Activation of these pathways causes chronic inflammation to continue and further inflammatory cells to be recruited [9].The immune profile of HS shows significant similarities to other immune-mediated inflammatory diseases (IMIDs), further supporting the concept of the disease's systemic nature [1]. The importance of those mechanisms is confirmed by the effectiveness of biological therapies targeting TNF-α and IL-17, which reduce disease activity and inflammatory symptoms.

In addition to local skin changes, patients with HS exhibit elevated inflammatory markers, such as CRP and proinflammatory cytokines, suggesting chronic systemic inflammation [10]. This mechanism might be the connection between HS and metabolic and cardiovascular diseases. Adipose tissue also plays an important role, as obese individuals produce adipokines and inflammatory mediators that intensify immune system activation. This creates a vicious cycle of inflammation, in which skin inflammation and metabolic conditions mutually exacerbate each other.

It is best to note that HS is not an infectious disease. The presence of bacteria in lesions is rather secondary and is associated with skin barrier dysfunction and chronic inflammation. Microbiological studies indicate dysbiosis of the skin microbiome, which may maintain the activation of the immune response [8]. Chronic stimulation of the immune system leads to persistent inflammation outside the skin and is a potential mechanism for the development of comorbidities observed in patients with HS.

A summary of immunological, clinical, and therapeutic data indicates that HS should be viewed as a systemic inflammatory disease in which the skin is only the most visible manifestation of the disease process. Common inflammatory pathways with other IMIDs, along with the presence of systemic inflammation markers, provide a biological basis for the observed increased risk of comorbidities.

5. SYSTEMIC COMORBIDITY SPECTRUM

An increasing number of epidemiological studies suggest that hidradenitis suppurativa (HS) is a systemic disease rather than a purely local skin condition. Population analyses show that patients with HS have a significantly higher risk of many comorbidities involving the metabolic, cardiovascular, digestive, rheumatological, endocrine, and psychiatric systems [1],[27]. A case-control study involving over 1,000 patients showed that people with HS have a substantially higher burden of comorbidities than the general population, further confirming the systemic nature of this disease [2].

The best-documented group of comorbidities in HS is cardiometabolic diseases. A meta-analysis of several dozen observational studies showed an approximately twofold higher risk of metabolic syndrome in patients with HS compared to the control population [7],[25]. The most common disorders include: obesity, type 2 diabetes, dyslipidemia, and hypertension. Population studies have shown that patients with HS have an approximately 1.5–2 times higher risk of type 2 diabetes and dyslipidemia compared to people without the disease [6],[21]. Obesity is especially associated with the pathogenesis of these conditions. Adipose tissue acts as an endocrine organ, producing inflammatory mediators such as TNF-α, IL-6, and adipokines that increase chronic inflammation and promote insulin resistance [1]. The increased risk of metabolic diseases also translates into a higher risk of cardiovascular complications. A large cohort study of the Danish population showed that patients with HS have a substantially increased risk of serious cardiovascular events, such as myocardial infaction or ischemic stroke, and also a higher risk of all-cause mortality compared to the general population [11]. The mechanism behind these correlations is explained by chronic inflammation leading to endothelial dysfunction and accelerated progression of atherosclerosis.

Inflammatory bowel disease (IBD) is also an important group of comorbidities. A meta-analysis of observational studies found that HS is associated with approximately 2.1-fold increased risk of Crohn's disease and approximately 1.5-fold increased risk of ulcerative colitis [12]. Some cohort studies have even shown a more than fivefold higher risk of IBD in patients with HS compared to the control population [12],[22]. HS and IBD belong to a group of immune-mediated inflammatory diseases that activate similar immune pathways, including the TNF-α/IL-17/IL-23 axis [1].

Another important group of comorbidities is inflammatory diseases of the musculoskeletal system. A meta-analysis of observational studies has shown that inflammatory joint diseases occur in 3–12% of patients with HS, a significantly higher rate than in the general population [13]. The most commonly reported are axial spondyloarthropathies, peripheral arthritis, and enthesitis (enthesopathies). As with other comorbidities, HS and spondyloarthropathies share common immune mechanisms involving activation of the IL-17/IL-23 pathway, which explains the effectiveness of similar biological therapies in both diseases [1].

A very important, but often overlooked aspect of HS is mental disorders. A meta-analysis of more than 40,000 patients showed that the prevalence of depression in this population is approximately 16.9% higher than in the general population [14],[24]. In other analyses, the prevalence of depression was estimated at up to approximately 21%, and that of anxiety disorders at approximately 12% [15],[26]. Among the factors that affect the mental state of patients are: chronic pain, unpleasant odor of skin lesions, purulent discharge, scarring and skin deformities, and social stigmatization. An increased risk of suicidal thoughts has also been reported in patients with HS [16]. In addition to psychosocial factors, biological mechanisms related to the effects of inflammatory cytokines on the nervous system may also serve a role [1].

The connection between HS and hormonal disorders is best documented in the case of polycystic ovary syndrome (PCOS). A meta-analysis of observational studies showed an approximately twofold higher risk of PCOS in women with HS compared to the general population [17]. PCOS is characterized by hyperandrogenism, insulin resistance, and metabolic disorders that can worsen the course of HS.

A growing number of studies also suggest an association between HS and liver disease, particularly non-alcoholic fatty liver disease (NAFLD). A meta-analysis of observational studies showed an approximately 1.7–1.8-fold higher risk of NAFLD in patients with HS compared to the control population [18]. This relationship is explained by common mechanisms involving chronic inflammation, insulin resistance, and lipid metabolism disorders.

The chronic nature of the disease and long-term inflammation may also increase the risk of certain cancers. The best-documented is the growth of squamous cell carcinoma in chronic HS lesions, especially in the anogenital area [20]. Population studies have also found an increased risk of lymphoma in patients with HS compared to the general population [20].

6. COMORBIDITY SCREENING AND RISK STRATIFICATION

Because of the wide range of comorbidities in patients with hidradenitis suppurativa (HS), greater emphasis is being placed on systematically assessing the overall health of these patients. The modern approach to HS goes beyond just treating skin changes and includes identifying and watching for other diseases that might affect disease progression and the overall prognosis. Early recognition of these conditions is key to implementing the right treatment and can improve patients' overall health results [21]. Studies indicate that some comorbidities remain undiagnosed for many years, particularly in the case of metabolic disorders and depression. For this reason, it is recommended to actively look for symptoms of these diseases during routine dermatological care.

A. Recommended range of screening tests

Based on available epidemiological data recommendations, the assessment of patients with HS should include a basic set of screening tests for the most common comorbidities.

The most important elements of such an assessment include:

- measurement of BMI and waist circumference,
- measurement of blood pressure,
- determination of blood glucose or HbA1c,
- assessment of the lipid profile,
- assessment of the patient's mental state (e.g., short screening questionnaires).

This strategy permits the early detection of risk factors for cardiovascular and metabolic diseases - the most common comorbidities in HS [21]. Besides laboratory tests, clinical history is also important. When other worrying symptoms are found, additional diagnostics should be considered to evaluate for comorbidities. Patients with severe forms of HS and coexisting obesity or metabolic syndrome may require more intensive monitoring and treatment.

An integrated strategy that includes comorbidity assessment and personalized therapy may improve the prognosis and quality of life of patients with HS.

7. MULTIDISCIPLINARY CARE MODEL

As there is more and more evidence showing the systemic nature of hidradenitis suppurativa (HS), it's important to take a multidisciplinary approach to caring for patients with this disease. HS shouldn't be treated solely as a dermatological condition, because there are many comorbidities that need specialists from different medical fields to be involved.

A dermatologist is usually the first to diagnose HS and plays a key role in coordinating further diagnosis and treatment. While assessing skin lesions in patients with HS, it is important to identify symptoms suggestive of comorbidities and refer patients to the appropriate specialists [1].

The following specialists most commonly involved include:

- internist or family doctor – monitoring metabolic risk factors
- gastroenterologist – diagnosis and treatment of inflammatory bowel disease
- rheumatologist – assessment of inflammatory symptoms of joint diseases
- psychiatrist or psychologist – treatment of mood disorders and psychological support
- surgeon – treatment of advanced skin lesions requiring surgical intervention

This approach enables a thorough patient assessment and improves treatment outcomes.

An important part of caring for patients with HS is also educating them about the disease and its potential complications. Patients should be informed about the importance of: weight loss, quitting smoking, and early reporting of new systemic symptoms. Awareness of the disease can improve patient cooperation with the doctor and increase the effectiveness of treatment.

8. KNOWLEDGE GAPS AND FUTURE DIRECTIONS

Despite the growth in research on hidradenitis suppurativa (HS), many aspects of its systemic nature remain poorly understood. Current epidemiological and immunological data show a strong association between HS and numerous comorbidities, but the mechanisms responsible for these associations call for further investigation. Most studies analyzing comorbidities in HS are observational. While they allow for the identification of epidemiological relationships, they do not always make it possible to determine the relationship between HS and other systemic diseases [1]. An additional limitation is the heterogeneity of the study populations and differences in research methodology. In many analyses, it is also difficult to fully account to confounding factors such as obesity, smoking, and lifestyle, which in themselves increase the risk of metabolic and cardiovascular diseases.

One of the most important areas for future research is conducting long-term prospective studies to better understand the relationship between HS and comorbidities. These studies could help determine whether the chronic inflammation observed in HS directly contributes to the development of metabolic, cardiovascular, or inflammatory bowel diseases. There is also a need for studies assessing the impact of HS treatment on the risk of comorbidities. In particular, it is interesting to know whether effective control of inflammation in HS may result in a reduction in the risk of systemic complications.

Another important area of research is the identification of biomarkers that could help assess disease activity and the risk of developing comorbidities. Inflammatory biomarkers, such as proinflammatory cytokines and C-reactive protein, may in the future be used to assess the systemic nature of HS.

Advances in research in this area may lead to a more individualized approach to the treatment of patients with HS.

9. CONCLUSIONS

Hidradenitis suppurativa (HS) is a chronic inflammatory disease that, for many years, was primarily considered a dermatological condition. However, a growing number of epidemiological and immunological studies indicate that HS should be treated as a systemic disease. The high prevalence of comorbidities, including metabolic disorders, cardiovascular disease, inflammatory bowel disease, rheumatic diseases, and mental disorders, suggests common pathophysiological mechanisms linked to chronic inflammation.

Understanding the systemic nature of HS is clinically important. Care for patients with HS should not be limited to treating skin lesions, but should also include assessing and monitoring potential comorbidities. Early detection of risk factors and joint effort among specialists from different medical fields can improve the prognosis and quality of life of patients. Further translational research including clinical, immunological, and genetic data may help to better understand the pathogenesis of HS. Also, a better understanding of common inflammatory pathways may enable the development of new therapeutic methods and improved treatment outcomes for patients with HS in the future.

To summarize, the accumulating evidence supports viewing HS as a systemic inflammatory disease rather than a purely cutaneous disorder. This approach may lead to more integrated patient care and a better understanding of this complex disease.

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