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Immunopathogenesis of Hidradenitis Suppurativa: Updated Review

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ABSTRACT

Introduction and Objective. Hidradenitis suppurativa (HS) is a chronic, recurrent inflammatory skin disease characterized by follicular occlusion, painful nodules, abscesses, sinus tract formation, and scarring, primarily affecting intertriginous areas. Although previously considered a disorder of the apocrine glands, HS is now recognized as a complex immunoinflammatory condition involving dysregulation of innate and adaptive immune pathways. This review aims to summarize current knowledge on the immunopathogenesis of HS, with particular emphasis on recent molecular and cellular insights relevant to targeted therapy.

Brief Overview of Current Knowledge. The initiating event in HS is follicular hyperkeratosis, leading to follicular rupture and secondary inflammation. Disease progression is driven by persistent immune activation, dominated by Th1/Th17 polarization. Key mechanisms include increased expression of proinflammatory cytokines such as IL-17, IL-1β, TNF-α, and IFN-γ, activation of the complement system, especially C5a, and dysregulation of antimicrobial peptides. Aberrant interactions between keratinocytes, fibroblasts, plasma cells, innate lymphoid cells, and T-cell subsets contribute to chronic inflammation and tissue destruction.

Summary. Hidradenitis suppurativa is a multifactorial immunoinflammatory disease in which follicular occlusion initiates a complex immune cascade. Identification of biomarkers and therapeutic targets, including IL-17-driven pathways, complement components, and CD2–CD58 signaling, supports the development of personalized, mechanism-based treatment strategies. Improved understanding of HS immunopathogenesis may enhance disease stratification and clinical outcomes.
Keywords: hidradenitis suppurativa, immunopathogenesis, immunology, complement activation, T-cell responses

Introduction and Objective

Hidradenitis suppurativa (HS), also known as acne inversa or Verneuil's disease, is a chronic inflammatory skin disorder characterized by follicular occlusion. The disease manifests with deep nodules, abscesses, draining tunnels, and progressive scarring, most commonly affecting intertriginous areas such as the axillae, inframammary folds, groin, genital region, buttocks, and perianal skin [1, 2]. These lesions frequently cause malodor and purulent discharge and are associated with significant discomfort and psychosocial burden, substantially impairing patients' quality of life and everyday functioning. HS affects approximately 1% of the population and occurs more commonly in women. Disease severity is often exacerbated by comorbidities, delayed diagnosis, and modifiable risk factors such as cigarette smoking and obesity [3, 4, 5, 6].

Dermatologists play a key role not only in diagnosing HS but also in identifying associated comorbidities and coordinating interdisciplinary management, as recommended by current international guidelines [7]. The global prevalence of HS varies widely, ranging from 0.00033% to 4.1%, which may reflect differences in genetic background, socioeconomic conditions, and methodological variations between epidemiological studies [8,9]. Although the epidemiology of HS is relatively well described in North America and Europe, the disease remains underexplored in many non-Western populations. Data from Africa, for example, may help clarify whether the higher prevalence of HS among African Americans results from genetic, environmental, or socioeconomic factors. In Europe, HS meets the definition of a rare disease according to the European Union, affecting fewer than 5 individuals per 10,000. In Poland, national estimates indicate an exceptionally low recorded prevalence of approximately 0.001%, though this is likely to represent a significant underestimation [10, 11, 12, 13, 14, 15].

Recent research has shifted the understanding of HS from an infectious process to a complex, multifactorial inflammatory disorder. Although its pathogenesis is not yet fully elucidated, several mechanisms, such as follicular occlusion, immune dysregulation, bacterial factors, and persistent inflammation, are believed to contribute to disease development [16, 17, 18]. Further clarification of these pathways is essential, as it may provide a basis for more targeted therapeutic approaches.

Aim

This review aims to provide an updated and comprehensive overview of the immunopathogenesis of hidradenitis suppurativa, with particular emphasis on recent discoveries that enhance the current

understanding of the underlying inflammatory pathways. By summarizing emerging molecular and cellular mechanisms, this article aims to highlight potential areas for therapeutic innovation and support the development of more targeted and effective treatment strategies.

Methods

This manuscript was prepared as a narrative review based on a comprehensive analysis of the current literature on hidradenitis suppurativa. A systematic search of major biomedical databases, including PubMed, Frontiers, Scopus, and Google Scholar, was conducted using combinations of the following keywords: hidradenitis suppurativa, acne inversa, immunopathogenesis, inflammation, IL-17, TNF-α, and follicular occlusion. Both original research articles and review papers were included, with a particular emphasis on studies published within the last decade to ensure the incorporation of the most up-to-date insights into the pathogenesis and immunological mechanisms of HS. Additional references were identified by screening the bibliographies of key articles.

Clinical Presentation of Disease in Hidradenitis Suppurativa

Hidradenitis suppurativa (HS), also known as acne inversa or Verneuil's disease, is a chronic inflammatory skin disorder characterized by recurrent, painful nodules resulting from follicular occlusion. The disease typically begins with a single, deep-seated inflammatory nodule measuring 1-2 cm, most commonly located in areas with apocrine glands, such as the axillae, inframammary folds, groin, perineum, and buttocks. Less frequently, lesions develop on the lower abdomen, suprapubic region, nape, postauricular areas, eyelids, or scalp [1, 2, 19, 20].

The initial nodule may persist for several weeks and often evolves into an abscess when the overlying skin ruptures, erosions or ulcers can develop. Many patients report one or two new nodules or abscesses per month. As the disease progresses, interconnected sinus tracts form and drain to the skin surface, becoming increasingly branched and challenging to treat. Pain is the dominant clinical symptom and is commonly accompanied by malodorous purulent discharge arising from abscesses and tunnels. The clinical course is typically relapsing-remitting, with quiescent periods interrupted by acute flares characterized by severe pain and the appearance of new inflammatory lesions. Early manifestations may include itching, paresthesia, pain, and fatigue, which often precede visible inflammation [19, 20].

The complications of HS can be particularly debilitating. Chronic inflammation may cause scarring that restricts limb mobility, while persistent disease can lead to urethral or anal strictures and the

formation of fistulas. Disfigurement, which may result from recurrent abscesses, scarring, and tissue destruction, can have profound psychosocial consequences for patients [21].

To classify disease severity, dermatologists commonly use the Hurley staging system [22], which categorizes HS into three stages:

• Stage I: single or multiple abscesses without sinus tracts or scarring;

• Stage II: recurrent abscesses with sinus tracts and scarring, with single or multiple widely separated lesions;

• Stage III: widespread involvement with multiple interconnected sinus tracts and abscesses across an entire affected area.

The natural history of HS remains incompletely understood; however, available evidence suggests that the disease is progressive in most untreated individuals. Importantly, modifiable lifestyle factors significantly influence disease severity. Smoking cessation and weight reduction have been associated with substantial improvements in clinical outcomes, and some individuals may even experience partial remission following these interventions [6, 23].

Etiology of Hidradenitis Suppurativa

Hidradenitis suppurativa is a multifactorial disorder in which genetic, environmental, metabolic and microbiological factors interact to trigger a chronic inflammatory response. Several genetic variants have been implicated in disease predisposition, and patients with HS frequently report a family history of the disorder [23, 24, 25]. Modifiable lifestyle factors, including cigarette smoking and obesity, significantly contribute to systemic and cutaneous inflammation, while adipokines, hormonal contraceptives (HC), and glucose dysregulation or diabetes have also been shown to influence disease activity. Growing evidence further highlights the role of the microbiome in HS, with alterations in bacterial composition frequently observed in individuals affected by the condition [6, 23, 24, 25]. Notably, smoking cessation and weight reduction have been associated with decreased disease severity and, in some cases, partial remission [23].

Bacterial colonization contributes to cutaneous inflammation in a subset of individuals. Studies have indicated a potential role for Corynebacterium spp. [27] and Streptococcus milleri [28] in the etiopathogenesis of hidradenitis suppurativa, although their precise contribution remains under investigation.

Immunopathogenesis – New Development

Historically, hidradenitis suppurativa was believed to originate from primary inflammation of the apocrine glands. However, accumulating evidence demonstrates that the initial pathogenic event is follicular hyperkeratosis within the pilosebaceous–apocrine unit [1,2,29]. Hyperkeratosis leads to follicular plugging and subsequent rupture, releasing keratin, bacteria, and cellular debris into the surrounding dermis. This activates innate immune pathways and induces secondary inflammation of apocrine structures [29].

The inflammatory response in HS involves dysregulation of both the innate and adaptive immune systems. Multiple cell types including T cells, B cells [30], monocytes, neutrophils, fibroblasts, and epithelial cells [31] contribute to the chronic inflammatory state. HS is strongly associated with the Th17 axis, largely due to the role of IL-17 in promoting psoriasiform epithelial changes and neutrophilic infiltration. Early studies showed that IL-17 stimulates keratinocytes, fibroblasts, and synoviocytes to release cytokines and chemokines, amplifying inflammation [31, 32].

A pivotal analysis, “Association of Hidradenitis Suppurativa With T Helper 1/T Helper 17 Phenotypes: A Semantic Map Analysis” by Thomi et al., used semantic mapping to characterize immune signatures in 24 untreated HS lesions compared with healthy controls. The study demonstrated clustering of Th1/Th17 cytokines, including IL-17, IFN-γ, IL-12, IL-23, IL-32, IL-1β, and TNF, along a common inflammatory axis. IL-1α and granulysin were upregulated in HS skin, while IL-13 showed an inverse correlation, supporting the concept that HS is predominantly a Th1/Th17-driven disease rather than a Th2-mediated condition [33]. Antimicrobial peptides such as LL-37, psoriasin, and human β-defensins were elevated, reflecting an additional layer of innate immune activation.

Another significant contribution comes from the study “Complement activation in hidradenitis suppurativa: a new pathway of pathogenesis?” by Kanni et al., which provided clear evidence of systemic complement activation in HS. The authors demonstrated that circulating C5a and C5b-9 levels were significantly elevated in HS and showed non-normal distribution patterns, whereas TNF-α levels exhibited approximate normality. Importantly, C5a concentrations were highest in patients with Hurley stage I disease, while C5b-9 levels and total complement activity progressively declined with increasing clinical severity [34]. This inverse relationship suggests that complement activation is most prominent in the early stages of HS and may reflect early inflammatory activity preceding the development of chronic, tissue-destructive lesions.

Further insights were provided by the study “CD2 expressing innate lymphoid and T cells are critical effectors of immunopathogenesis in hidradenitis suppurativa” by Kashyap et al. The authors showed that CD2 central to T-cell and NK-cell activation is upregulated in HS skin. Blocking the CD2:CD58 interaction in HS skin explants revealed distinct populations of NKT and NK cells occupying defined tissue niches, interacting with keratinocytes and fibroblasts through a complex CD2-centered immune network [35]. These findings highlight CD2 as a key regulator of immune dysregulation in HS and a potential therapeutic target.

Summary

Hidradenitis suppurativa (HS) is a chronic, relapsing inflammatory skin disease that represents a complex immunoinflammatory disorder rather than a condition limited to follicular occlusion alone. Increasing evidence indicates that dysregulated innate and adaptive immune responses play a central role in disease development and progression. Key immunopathological features include Th1/Th17 polarization, complement system activation, imbalance of antimicrobial peptides, plasma cell-derived cytokine production, and abnormal interactions among keratinocytes, fibroblasts, innate lymphoid cells, and T-cell subsets. These mechanisms collectively contribute to persistent inflammation, tissue destruction, and the characteristic chronic and recurrent clinical course of HS. Recent translational studies have identified several immune mediators, such as C5a, C5b-9, IL-17, IL-1 family cytokines, and CD2-related signaling pathways, as potential biomarkers of disease activity, severity, and progression. Some of these factors may support disease stratification, therapeutic response monitoring, and early detection of inflammatory activity. Advances in understanding HS immunopathogenesis have also facilitated the development of targeted and personalized treatment strategies, including therapies directed against IL-17, complement components, CD2-CD58 interactions, and plasma cell-associated pathways, complementing existing biologic options.

Despite significant progress, important gaps remain in fully elucidating disease mechanisms, identifying reliable biomarkers, and optimizing long-term management strategies. Continued integration of molecular, immunological, and clinical research is essential to refine therapeutic approaches and improve outcomes for patients with hidradenitis suppurativa.

Disclosure:

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Author's contribution

Conceptualization: Wiktoria Marszał; Methodology: Wiktoria Marszał; Software: Nina Saracen, Sandra Czyż; Validation: Wiktoria Marszał; Formal analysis: Nina Saracen, Wiktoria Marszał; Investigation: Sandra Czyż; Resources: Sandra Czyż, Wiktoria Marszał; Data curation: Nina Saracen; Writing – original draft: Wiktoria Marszał; Writing – review & editing: Nina Saracen, Sandra Czyż; Visualization: Sandra Czyż; Supervision: Nina Saracen; Project administration: Wiktoria Marszał

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Our work did not involve direct human subject research or obtaining their consent for participation in the study.

Data Availability Statement

As a review paper, our work does not present new data or analyses. Therefore, there are no specific databases or data availability to report. The information and findings presented in this review are based on previously published studies, which can be accessed through their resective sources as cited in the reference section.

Conflicts of Interest Statement

The authors declare that there are no significant conflicts of interest associated with this research work.

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