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Physical Activity and Hidradenitis Suppurativa: A Narrative Review

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Abstract

Background. Hidradenitis suppurativa (HS) is a systemic inflammatory dermatosis characterized by follicular occlusion and a cytokine profile dominated by TNF-α and IL-17. Obesity is a primary modifiable risk factor, where each 1-unit increase in BMI correlates with an 8 to 12 percent rise in HS risk, and adipose tissue functions as a pro-inflammatory endocrine organ exacerbating the disease course.

Aim. The objective of this analysis is to determine whether and how physical activity modifies the clinical progression, inflammatory status, and metabolic profile of patients diagnosed with hidradenitis suppurativa.

Material and methods. This study synthesized clinical data regarding the pathophysiology of HS, the endocrine function of adipose tissue, and the systemic effects of various physical activity intensities. Analysis focused on the correlation between BMI and Hurley staging, the impact of exercise on pro-inflammatory cytokines (IL-6, TNF-α, IL-1β), and the role of mechanical factors in lesion formation.

Results. Physical activity exerts a bidirectional effect on HS. Systemically, regular moderate exercise (50–70% HRmax) induces an anti-inflammatory state by reducing C-reactive protein and increasing IL-10, while weight reduction of at least 10 percent is associated with significant clinical improvement. Conversely, physical exertion may cause local exacerbations; sweat-induced maceration and mechanical friction in intertriginous zones can trigger new lesions via the Koebner phenomenon. While active patients exhibit lower Hurley

stages and improved quality of life scores, approximately 60 percent of the HS population remains sedentary due to chronic pain and mechanical limitations caused by sinus tracts.
Conclusions. Physical activity modifies the course of HS primarily as an indirect systemic stabilizer that reduces the inflammatory burden and improves metabolic sensitivity. However, to avoid local mechanical provocation of the pilosebaceous unit, exercise regimens must be individualized, prioritizing low-friction modalities to balance systemic benefits against the risk of localized disease flares.

Key words: Hidradenitis suppurativa, acne inversa, sport, physical activity modifying factor

1. Introduction

Hidradenitis suppurativa (HS), also known as acne inversa, is a chronic systemic inflammatory skin disease. The predominant clinical manifestations include painful inflammatory nodules. Abscesses, sinus tracts, and scarring are also observed in advanced stages of the disease. Lesions occur primarily in the axillary, inguinal, and anogenital regions due to the presence of apocrine glands [1].

Acne inversa is a systemic disorder in which the pathogenesis is dominated by dysregulation of the immune response, chronic inflammation, and alterations of both the cutaneous and gut microbiome [2,3].

The primary mechanism underlying HS development is follicular hyperkeratosis and occlusion, leading to follicular rupture and a secondary inflammatory response involving proinflammatory cytokines such as TNF-α, IL-1β, and IL-17. During the disease course, chronic destructive inflammatory lesions develop, resulting in the formation of sinus tracts and scarring [1].

HS affects approximately 1% of the population. It occurs three times more frequently in women, with peak incidence in the second to fourth decades of life. The Dermatology Life

Quality Index (DLQI) in HS is markedly elevated, indicating a substantial negative impact on patients' quality of life. Patients report chronic pain, reduced mobility, difficulties in daily functioning, and significant psychological burden, including an increased prevalence of depression and anxiety [4].

The most important risk factors for HS include obesity, tobacco smoking, genetic predisposition, and hormonal disturbances. Obesity plays a particularly significant role, as it not only increases the risk of disease onset but also exacerbates its clinical course.

The mechanisms linking obesity with HS primarily include chronic low-grade inflammation, increased production of adipokines (e.g., leptin), mechanical friction and maceration within skin folds, and alterations in both the gut and skin microbiome.

Observational studies have demonstrated that a higher body mass index (BMI) positively correlates with HS severity as assessed by the Hurley staging system [5].

Lifestyle factors play a significant role in both the development and progression of HS. Low levels of physical activity and unhealthy dietary habits are associated with an increased risk of disease onset and a more severe clinical course.

2. Pathophysiology of Acne Inversa

The etiopathogenesis of HS is multifactorial. First, it involves abnormalities of the pilosebaceous unit. The key initiating event is hyperkeratosis of the follicular infundibulum, leading to occlusion and retention of keratin. Over time, progressive follicular dilatation occurs, followed by rupture. The released contents - keratin, bacteria, and degradation products—enter the dermis, where they induce a robust immune response. It is worth noting that genetic predisposition plays a significant role in this mechanism, as a positive family history is observed in approximately 30% of patients [6].

Another important aspect is chronic inflammation. Following follicular rupture, both innate and adaptive immune responses are activated. The inflammatory process assumes the form of a feed-forward loop, in which tissue damage further amplifies immune activation [7]. Characteristic features of inflammation in HS include infiltration by neutrophils, macrophages, and T lymphocytes, activation of dendritic cells, predominance of the Th17 response,

increased expression of matrix metalloproteinases and TGF-β, all of which promote fibrosis and scar formation [7].

Within the immunological basis of inflammation, TNF-α plays a central role. It activates endothelial cells, facilitates leukocyte migration, and amplifies the inflammatory response. Elevated levels of TNF-α in HS tissues provide the rationale for the efficacy of biologic therapies (e.g., adalimumab) [8]. An additional key component is the IL-17/Th17 pathway. IL-17 expression in HS skin is increased by as much as 30–149-fold compared to healthy control skin. IL-17 induces the production of chemokines and antimicrobial peptides, promotes neutrophil recruitment, and intensifies the inflammatory response. This environment favors keratinocyte proliferation and further follicular occlusion [8].

The skin and gut microbiome also play a significant, although not yet fully elucidated, role in HS pathogenesis. Lesional skin demonstrates reduced microbial diversity, the presence of opportunistic bacteria (e.g., *Staphylococcus*, *Corynebacterium*, *Prevotella*), and biofilm formation within sinus tracts. Interactions between the microbiome and the immune system contribute to chronic immune stimulation [9].

3. Obesity as a Risk Factor and Determinant of HS Severity

Obesity represents one of the best-documented, modifiable risk factors for both the development and progression of hidradenitis suppurativa (HS). Numerous cohort studies and population-based analyses have demonstrated a significant positive correlation between body mass index (BMI) and the prevalence of HS. The prevalence of HS in the general population is estimated at approximately 0.7–1.2%, whereas in individuals with obesity (BMI ≥30 kg/m²) it increases up to 2–3-fold [10,11]. In cross-sectional studies, the mean BMI of patients with HS ranged from 28 to 33 kg/m², significantly exceeding values observed in control populations [11]. Multivariate analyses have shown that each 1-unit increase in BMI is associated with an approximately 8–12% increase in HS risk [12]. Moreover, obesity correlates not only with disease occurrence but also with earlier onset and a more severe clinical course.

Adipose tissue functions as an active endocrine organ, producing numerous adipokines and proinflammatory cytokines. In patients with obesity, a state of chronic low-grade inflammation develops, which may initiate and amplify the immunological processes

characteristic of HS. Key findings include elevated levels of TNF-α, IL-6, and leptin, as well as reduced levels of adiponectin, which exerts anti-inflammatory effects [8,13].

Obesity is closely associated with the development of insulin resistance, which plays a significant role in the pathogenesis of HS. Hyperinsulinemia promotes increased keratinocyte proliferation, enhanced follicular keratinization, and increased androgen production (indirectly via effects on sex hormone-binding globulin, SHBG). These mechanisms facilitate follicular occlusion, a critical initiating step in HS. Clinical studies have demonstrated that insulin resistance (e.g., assessed using the HOMA-IR index) occurs significantly more frequently in patients with HS than in the general population, independent of BMI [14]. Additionally, HS frequently coexists with metabolic syndrome, with a prevalence of up to 40–50% in this patient group [11].

Obesity also amplifies mechanical factors that play an important role in the localization of HS lesions. Excess body weight leads to increased friction in skin folds (axillary, inguinal, inframammary regions), occlusion of follicular ostia, and microtrauma that predisposes to follicular rupture. These processes initiate an inflammatory cascade and may explain the characteristic distribution of HS lesions. Observational studies have shown that weight reduction is associated with a significant decrease in the number of inflammatory lesions and the frequency of disease flares [15].

Patients with obesity more frequently present with Hurley stage II–III disease (a three-stage classification system for HS severity) and exhibit a substantially higher number of inflammatory lesions. Clinical analyses have demonstrated that individuals with BMI ≥30 kg/m² have up to a twofold increased risk of severe disease (Hurley stage III) compared to those with normal body weight [12]. Furthermore, obesity is associated with a poorer response to treatment, including biologic therapies, which may be attributable to chronic immune system activation. Obesity is also a significant factor influencing HS recurrence. Studies have demonstrated a higher rate of relapse of inflammatory lesions among obese patients, who also exhibit an increased risk of persistent sinus tracts and chronic lesions. Importantly, a reduction in body weight of ≥10% may lead to significant clinical improvement and, in some cases, even disease remission [15,16].

4. Physical Activity, Obesity, and Inflammation

Regular physical activity leads to a significant reduction in body mass and fat tissue content, particularly visceral fat, which is the main source of inflammatory mediators. Interventional studies have demonstrated that exercise programs lasting ≥8 weeks result in a significant decrease in body fat percentage accompanied by an increase in lean body mass [17]. Meta-analyses indicate that a body weight reduction of 5–10% is associated with a significant decrease in cardiometabolic risk and a reduction in inflammatory markers, including C-reactive protein (CRP) and IL-6 [18].

Physical activity increases insulin sensitivity and improves the lipid profile (↓ LDL, TG; ↑ HDL). Studies have shown that exercise induces significant changes in glucose and lipid metabolism even immediately after a single training session, with these effects being more pronounced in individuals with obesity [19].

Regular physical activity reduces CRP levels, decreases proinflammatory cytokines (IL-6, TNF-α, and IL-1β), and increases anti-inflammatory cytokines such as IL-10. It has been demonstrated that programs of moderate physical activity result in significant reductions in IL-1β, IL-6, and TNF-α [17,20]. Similar findings have been reported in studies involving combined (aerobic-resistance) training, where reductions in TNF-α and IL-6 were observed [21].

Another important aspect is the endocrine function of skeletal muscles, which secrete myokines, including IL-6. Although adipose tissue also produces IL-6, the fraction derived from skeletal muscle exerts anti-inflammatory effects. It inhibits TNF-α production, stimulates IL-10 secretion, and reduces oxidative stress [22]. Furthermore, physical activity induces remodeling of the adipose tissue microenvironment by decreasing macrophage infiltration and promoting a phenotypic shift from proinflammatory (M1) to anti-inflammatory (M2) macrophages [20,22].

Physical activity can be classified according to intensity. Moderate-intensity activity, corresponding to 50–70% of maximal heart rate (HRmax), demonstrates a consistent effect in reducing inflammatory markers and improving body composition, forming the foundation of obesity management recommendations [20]. High-intensity physical activity (70–90% HRmax), despite conferring greater metabolic benefits, may transiently induce an increase in inflammatory markers. Meta-analyses indicate that high-intensity interval training (HIIT) is the most effective in reducing IL-6 and TNF-α; however, it is associated with more pronounced acute inflammatory responses, particularly in individuals with obesity [19,20].

5. Physical Activity and the Course of Acne Inversa (Hidradenitis Suppurativa)

The beneficial effects of physical activity include, among others, body weight reduction. Obesity constitutes one of the most important modifiable risk factors for HS [24]. Physical activity, as a key component of obesity management, may indirectly influence the course of HS by reducing visceral adipose tissue, decreasing the production of proinflammatory adipokines, and lowering levels of TNF-α and IL-6.

Another important aspect is the improvement of metabolic status. Patients with HS frequently present with metabolic disturbances, including insulin resistance and dyslipidemia [25]. Studies have demonstrated that HS coexists with an unfavorable metabolic profile and chronic activation of inflammatory pathways. Physical activity improves metabolic parameters by increasing insulin sensitivity, improving lipid metabolism, and reducing oxidative stress. A 2023 systematic review emphasized that lifestyle modifications, including physical activity and weight reduction, may lead to both subjective and objective improvement in the clinical course of HS [23].

However, physical activity may also have adverse effects in patients with dermatological conditions, including acne inversa. Increased sweating associated with exercise leads to epidermal maceration and promotes bacterial colonization, which may exacerbate inflammation of hair follicles. Areas predisposed to HS (axillary, inguinal, and anogenital regions) are particularly susceptible to this mechanism [23].

Mechanical friction of the skin, especially in intertriginous areas, constitutes a significant factor initiating inflammatory lesions. Repeated mechanical microtrauma leads to disruption

of the epidermal barrier and hyperkeratinization, ultimately resulting in activation of the inflammatory response. Friction is particularly intensified during aerobic physical activity (e.g., running), especially in individuals with excess body weight.

Microtrauma resulting from friction, as well as from hair removal or intense physical exertion, may initiate the formation of new lesions via the Koebner phenomenon. Damage to the pilosebaceous unit leads to its occlusion and a secondary inflammatory response [25].

Therefore, individualization of recommendations and appropriate selection of physical activity (e.g., swimming, low-friction training modalities) are of key importance, as they may minimize the risk of exacerbations while preserving metabolic benefits.

6. Comparison of Physically Active and Inactive Patients with HS

An analysis of differences between hidradenitis suppurativa patients with active versus sedentary lifestyles represents a clinically significant, yet insufficiently investigated domain. Available data suggest that physical activity may influence both the clinical course of the disease and metabolic parameters as well as quality of life; however, findings remain inconclusive and are burdened by numerous limitations [26].

Disease severity in HS, assessed for example using the Hurley staging system or the International Hidradenitis Suppurativa Severity Score System (IHS4), is positively correlated with obesity and metabolic factors [26]. Data suggest that physically inactive patients more frequently present with higher Hurley stages (II–III). In contrast, physical activity, particularly when combined with dietary interventions, is associated with lower disease severity in cross-sectional studies [27].

HS is among the dermatoses with the greatest negative impact on quality of life. Mean Dermatology Life Quality Index (DLQI) scores range from 8.4 to 16.9, corresponding to a large or very large impairment in quality of life [28]. In a study involving more than 400 patients, the mean DLQI was 13.18 ± 7.99, and as many as 60% of patients reported a very large or extremely large impairment in quality of life [29]. Physically active patients more frequently report better quality of life and exhibit lower rates of depression and social isolation.

Physical activity may indirectly reduce disease severity through its effects on obesity and inflammation. A cross-sectional study demonstrated that higher levels of physical activity and adherence to a healthy lifestyle correlate with lower disease activity [27].

7. Limitations of Physical Activity in Patients with HS

Pain is one of the predominant symptoms of HS and one of the principal factors limiting physical activity. A retrospective study from 2023 demonstrated that pain intensity is significantly correlated with deterioration in quality of life and impairment of daily functioning [30]. Lesions are most commonly located in the axillary, inguinal, anogenital, and inframammary regions. These areas are particularly susceptible to friction during movement, pressure, and increased sweating. Consequently, even basic forms of physical activity, such as walking or running, may exacerbate pain. In patients with advanced HS (Hurley stage II–III), pain may be chronic and may also have a neuropathic component, further limiting mobility [30].

Inflammatory lesions, sinus tracts, and scarring lead to significant mechanical limitations. The most important include restricted range of motion, pain upon skin stretching, and the risk of lesion rupture, resulting in purulent discharge during physical exertion. In severe cases, HS may be considered a condition impairing the ability to perform physical work [31].

HS significantly affects daily functioning and lifestyle choices. Studies indicate that patients with HS more frequently adopt a sedentary lifestyle [26]. The disease is associated with an increased risk of obesity and metabolic syndrome [26]. Additionally, the need for frequent dressings and recurrent disease flares may lead to irregular engagement in physical activity.

8. Conclusion

Physical activity may modify the course of hidradenitis suppurativa; however, its effects are complex, bidirectional, and dependent on multiple clinical and environmental factors. From a systemic perspective, regular physical exercise demonstrates potentially beneficial effects. It leads to improvement in metabolic parameters, increased insulin sensitivity, and reduction of chronic inflammation through decreased levels of proinflammatory cytokines (TNF-α, IL-6, IL-1β) and increased levels of anti-inflammatory mediators. These mechanisms may limit

inflammatory activity in HS, reduce the number of lesions, and contribute to a milder disease course.

At the same time, physical activity may exert adverse effects at the local level. Increased sweating, friction, and mechanical microtrauma promote damage to the pilosebaceous unit and initiation of the inflammatory response. Consequently, this may lead to the development of new lesions, exacerbation of existing foci, and more frequent disease flares, particularly in the context of high-intensity or improperly selected exercise.

Thus, physical activity does not exert a unidirectional effect on HS - it may both ameliorate and exacerbate the disease course. Its impact depends on the type, intensity, and conditions of exercise, as well as individual patient characteristics, including lesion distribution and disease severity.

Available evidence suggests that physical activity primarily acts as an indirect modifier of HS through its effects on metabolic and inflammatory pathways, while simultaneously carrying the potential for local irritative effects. Appropriately selected, individualized physical activity—minimizing friction and skin moisture—may constitute an important component of adjunctive therapy; however, its role as an independent therapeutic factor remains limited and requires further investigation.

AI.

AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. **AI** were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

Disclossure

Author Contributions

Conceptualisation: PB, ZM, ZB, WB,

Methodology: WJ, JR

Check: ZM, ZB,

Formal analysis: JR, WB

Investigation: WJ, ZB

Resources:PB, ZM, WJ

Data curation: WB, WJ, JR

Writing –rough preparation: PB, WJ, ZM

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In preparing this work, the authors used ChatGPT for the purpose of enhancing language clarity, improve readability and basic data analysis. After using this tool/service, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication

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