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Sleep Quality, Obstructive Sleep Apnea Risk, and Metabolic–Inflammatory Profiles in Hidradenitis Suppurativa: A Prospective Observational Study

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Abstract

Background: Hidradenitis suppurativa (HS) is a chronic systemic inflammatory disorder increasingly associated with metabolic and cardiovascular comorbidities. Obstructive sleep apnea (OSA) shares overlapping inflammatory and metabolic pathways; however, whether increased OSA risk in HS reflects obesity alone or a broader inflammatory–metabolic phenotype remains unclear. We prospectively evaluated sleep quality, OSA risk, and their association with metabolic and inflammatory parameters in patients with HS.

Methods: In this prospective controlled study, 38 patients with clinically confirmed HS and 41 age-matched controls were enrolled. Sleep quality and OSA risk were assessed using the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and STOP-BANG questionnaire. Fasting metabolic parameters and inflammatory markers, including neutrophil-to-lymphocyte ratio (NLR), were analyzed. Multivariable logistic regression models were constructed to evaluate independent associations.

Results: HS patients demonstrated significantly worse sleep-related outcomes, including higher PSQI ($p=0.028$) and ESS scores ($p=0.016$). Excessive daytime sleepiness (ESS ≥ 10) was more frequent in HS (39.5% vs. 9.8%, $p=0.002$). STOP-BANG scores were significantly higher ($p=0.013$), and high OSA risk was observed in 26.3% of HS patients compared with 2.4% of controls ($p=0.009$). Importantly, body mass index did not differ between groups ($p=0.729$). HS patients exhibited a pro-inflammatory and atherogenic metabolic profile characterized by lower HDL ($p<0.001$), higher triglycerides ($p=0.002$), elevated neutrophil counts ($p<0.001$), and increased NLR ($p=0.011$). In multivariable analysis, lower HDL (OR 0.818, $p<0.001$) and higher NLR (OR 1.381, $p=0.036$) remained independently associated with HS.

Conclusions: HS is associated with impaired sleep parameters and markedly increased OSA risk independent of obesity. This increased vulnerability appears to be driven by a systemic inflammatory and atherogenic metabolic phenotype. Routine OSA screening should be considered in HS patients to mitigate potential cardiopulmonary risk.

Keywords: Hidradenitis suppurativa, Obstructive sleep apnea, Systemic inflammation

Introduction

Hidradenitis suppurativa (HS) is a chronic, relapsing inflammatory skin disorder characterized by painful nodules, abscesses, sinus tract formation, and scarring in intertriginous areas. Increasing evidence suggests that HS should be regarded not merely as a cutaneous condition but as a systemic inflammatory disease associated with substantial metabolic and cardiovascular comorbidity burden [1, 2]. Dysregulated innate and adaptive immune responses, including elevated levels of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-17, contribute to persistent systemic inflammation in HS[2]. Consistent with this inflammatory state, patients with HS frequently exhibit obesity, insulin resistance, dyslipidemia, and features of metabolic syndrome [3, 4].

Obstructive sleep apnea (OSA) is a prevalent sleep-related breathing disorder characterized by recurrent upper airway collapse, intermittent hypoxia, and sleep fragmentation. OSA is independently associated with systemic inflammation, oxidative stress, endothelial dysfunction, and cardiometabolic disease[5, 6]. Intermittent hypoxia induces activation of inflammatory pathways and sympathetic overactivity, mechanisms that overlap with those implicated in chronic inflammatory disorders [6]. Beyond obesity a shared risk factor systemic inflammation and metabolic dysregulation are increasingly recognized as central contributors to OSA pathophysiology [5, 7].

Emerging epidemiological and clinical data indicate that sleep disturbances are common among patients with HS[8]. Chronic pain, nocturnal pruritus, psychological distress, and inflammatory activation may all contribute to impaired sleep quality in this population. However, whether the increased risk of OSA in HS is solely attributable to elevated body mass index or reflects a broader inflammatory–metabolic phenotype remains uncertain. Importantly, HS has been associated with adverse lipid profiles and increased cardiovascular risk markers[3, 4], factors that are also mechanistically linked to OSA development and severity[5, 6].

Inflammatory indices such as the neutrophil-to-lymphocyte ratio (NLR) have been proposed as accessible biomarkers of systemic inflammatory burden in HS and correlate with disease activity [9]. Similarly, reduced high-density lipoprotein (HDL) levels and atherogenic lipid alterations are frequently reported in HS and may reflect chronic inflammatory dysregulation[3, 4]. Given that both systemic inflammation and dyslipidemia are implicated in OSA pathogenesis[5, 7], HS patients may represent a subgroup characterized by overlapping inflammatory and sleep-related risk profiles.

To date, no prospective controlled study has comprehensively evaluated sleep quality, risk of obstructive sleep apnea, and their associations with metabolic and inflammatory markers in patients with hidradenitis suppurativa. We therefore conducted a prospective observational study to assess sleep quality and OSA risk in HS patients compared with matched controls and to investigate whether these alterations are associated with metabolic and systemic inflammatory parameters.

Materials and Methods

Study Design and Ethical Approval

This prospective observational controlled study was conducted to evaluate sleep quality, OSA risk, and their association with metabolic and systemic inflammatory parameters in patients with HS.

The study was prospectively designed prior to participant enrollment, and all data were collected in a predefined and standardized manner. Patients with a confirmed diagnosis of HS and age and sex comparable control participants without HS were consecutively recruited during the study period.

Ethical approval was obtained from the Istanbul Medipol University Non Interventional Clinical Research Ethics Committee (Approval Date: 15 December 2026; Decision No: E-10840098-202.3.02-8425). The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to inclusion.

Study Population

Adult patients (≥18 years) with a clinical diagnosis of hidradenitis suppurativa were prospectively recruited from the outpatient clinic. The diagnosis of HS was established by a dermatologist based on established clinical criteria.

The control group consisted of age and sex matched individuals without a history of HS or other chronic inflammatory dermatologic diseases. Controls were recruited from the same healthcare setting to minimize selection bias.

Inclusion criteria (HS group):

- Age ≥18 years
- Clinically confirmed diagnosis of HS
- Willingness to participate and provide informed consent

Exclusion criteria (both groups):

- Known diagnosis of obstructive sleep apnea under treatment
- Chronic pulmonary diseases
- Active infection within the previous four weeks
- Chronic inflammatory or autoimmune disorders other than HS
- Malignancy
- Pregnancy

Clinical and Demographic Assessment

Demographic data including age, sex, smoking status, alcohol consumption, and body mass index (BMI) were recorded. Comorbid conditions such as hypertension and diabetes mellitus were documented.

Disease severity in HS patients was assessed using:

- Hurley staging system
- International Hidradenitis Suppurativa Severity Score System (IHS4)

Dermatology Life Quality Index (DLQI) scores were recorded to evaluate disease-related quality of life.

Sleep Assessment

Sleep quality and OSA risk were evaluated using validated questionnaires:

- **Pittsburgh Sleep Quality Index (PSQI)** to assess subjective sleep quality. A global score >5 was considered poor sleep quality.
- **Epworth Sleepiness Scale (ESS)** to evaluate daytime sleepiness. A score ≥10 indicated excessive daytime sleepiness.
- **STOP-BANG questionnaire** to assess OSA risk. Participants were categorized as low (0–2), intermediate (3–4), or high risk (5–8) for OSA.

All questionnaires were administered at the time of enrollment.

Laboratory and Inflammatory Parameters

Fasting blood samples were obtained from all participants in the morning following an overnight fast.

The following laboratory parameters were recorded:

- Lipid profile (LDL, HDL, total cholesterol, triglycerides)
- Fasting plasma glucose
- Hemoglobin A1c (HbA1c)
- Complete blood count including neutrophil, lymphocyte, and platelet counts

NLR was calculated as:

NLR = Neutrophil count / Lymphocyte count

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for MacOS, version 30.0 (IBM Corp., Armonk, NY, USA).

Categorical variables were expressed as number and percentage [n (%)]. Continuous variables were presented as mean ± standard deviation (SD) or median (interquartile range, IQR), depending on distribution.

Normality was assessed using visual (histogram, Q–Q plots) and analytical methods. Between-group comparisons were performed using:

- Independent samples Student's t-test or Welch test for normally distributed variables
- Mann–Whitney U test for non-normally distributed variables
- Pearson's chi-square or Fisher's exact test for categorical variables

To evaluate factors independently associated with HS presence, logistic regression analyses were performed. Variables with $p<0.10$ in univariate analysis and clinically relevant variables were included in multivariable logistic regression models.

Two separate multivariable models were constructed:

- Model 1 (Sleep model): age, sex, BMI, smoking status, PSQI
- Model 2 (Metabolic–inflammatory model): age, sex, BMI, HDL, NLR

Results were reported as odds ratios (OR) with 95% confidence intervals (CI).

Multicollinearity was assessed using the variance inflation factor (VIF). Model calibration was evaluated using the Hosmer–Lemeshow goodness-of-fit test.

A two-sided p value <0.05 was considered statistically significant.

Results

Baseline Demographic and Clinical Characteristics

A total of 79 participants were included in the analysis, comprising 38 patients with hidradenitis suppurativa (HS) and 41 controls. Baseline demographic and clinical characteristics are summarized in **Table 1**.

Sex distribution differed significantly between groups. Male sex was markedly more prevalent in the HS group compared with controls (63.2% vs. 26.8%, p=0.001; Table 1). Age did not differ significantly between groups (39±10 vs. 41±14 years, p=0.518). Similarly, smoking status, alcohol consumption, body mass index (BMI), hypertension, and diabetes mellitus were comparable between groups (all $p>0.05$; Table 1).

With respect to sleep parameters, HS patients demonstrated significantly worse sleep-related outcomes (Table 1). Mean Pittsburgh Sleep Quality Index (PSQI) scores were higher in the HS group (4.2±4.7 vs. 2.2±3.0, p=0.028). Although the proportion of participants with poor sleep quality (PSQI >5) was numerically greater among HS patients (44.7% vs. 24.4%), this difference approached but did not reach statistical significance (p=0.057).

Daytime sleepiness was significantly more pronounced in HS. Epworth Sleepiness Scale (ESS) scores were higher in the HS group (5.1±5.9 vs. 2.3±3.6, p=0.016), and excessive daytime sleepiness (ESS ≥10) was significantly more frequent among HS patients (39.5% vs. 9.8%, p=0.002; Table 1).

Similarly, STOP-BANG scores were significantly elevated in HS (2.3±2.6 vs. 1.0±1.6, p=0.013). High OSA risk (STOP-BANG score 5–8) was observed in 26.3% of HS patients compared with 2.4% of controls (p=0.009; Table 1). These findings indicate a substantially increased OSA risk profile in HS patients.

Metabolic and Inflammatory Parameters

Comparisons of laboratory and inflammatory parameters between groups are presented in **Table 2**.

HS patients exhibited a distinctly adverse lipid profile. LDL levels were significantly higher in the HS group (p<0.001), while HDL levels were markedly lower (39.5±6.9 vs. 58.3±15.4 mg/dL, p<0.001). Triglyceride levels were also significantly elevated in HS patients (p=0.002). Total cholesterol levels differed significantly between groups (p=0.006; Table 2).

Regarding glycemic parameters, fasting glucose levels were statistically different between groups (p=0.024), whereas HbA1c did not show a significant difference (p=0.103; Table 2).

Blood pressure measurements demonstrated significantly higher systolic and diastolic values in HS patients (both p<0.001; Table 2), reinforcing the presence of a cardiometabolic burden.

Inflammatory markers were consistently elevated in HS. Neutrophil counts (p<0.001), platelet counts (p<0.001), and neutrophil-to-lymphocyte ratio (NLR) values (4.3±3.9 vs. 2.5±2.0, p=0.011) were significantly higher in HS patients. In contrast, lymphocyte counts and platelet-to-lymphocyte ratio (PLR) did not differ significantly between groups (p=0.776 and p=0.336, respectively; Table 2).

These results collectively support the presence of a systemic inflammatory and atherogenic metabolic phenotype in HS.

Logistic Regression Analyses

Univariate Analysis

Results of the univariate logistic regression analysis are shown in **Table 3**.

Male sex was strongly associated with HS presence (OR 4.675, 95% CI 1.799–12.148; p=0.002). Higher PSQI scores were modestly associated with increased odds of HS (OR 1.146, 95% CI 1.013–1.296; p=0.030).

Among metabolic and inflammatory variables, HDL levels demonstrated a strong inverse association with HS (OR 0.839 per 1 mg/dL increase; p<0.001), whereas higher NLR values were positively associated with HS (OR 1.234; p=0.016). Age, BMI, and smoking status were not significantly associated with HS (Table 3).

Multivariable Analysis

Multivariable logistic regression results are summarized in **Table 4**.

In Model 1 (sleep model), including age, sex, BMI, smoking status, and PSQI, only male sex remained independently associated with HS (OR 7.455, 95% CI 1.933–28.753; p=0.004). PSQI did not retain independent statistical significance (p=0.111; Table 4).

In Model 2 (metabolic–inflammatory model), including age, sex, BMI, HDL, and NLR, both HDL and NLR remained independently associated with HS presence. Higher HDL levels were independently protective (OR 0.818; p<0.001), whereas higher NLR values were independently associated with increased HS odds (OR 1.381; p=0.036). In this model, sex and BMI were no longer statistically significant (Table 4).

Summary of Findings

As demonstrated in Tables 1–4, HS is associated with significantly impaired sleep parameters, increased OSA risk, an adverse lipid profile characterized by low HDL and elevated LDL and triglycerides, and a heightened inflammatory state reflected by increased neutrophil counts and NLR.

Importantly, BMI did not differ between groups, suggesting that the observed increase in OSA risk among HS patients may not be attributable solely to adiposity but rather to an underlying inflammatory–metabolic phenotype.

Table 1. Demographic and Clinical Characteristics of Participants With and Without Hidradenitis Suppurativa

Variables (N=79)	**Total**	**No HS (n=41)**	**HS (n=38)**	**p-value**
Sex				0.001
Male	35 (44.3%)	11 (26.8%)	24 (63.2%)	
Female	44 (55.7%)	30 (73.2%)	14 (36.8%)	
Age (years)	40 ± 12	41 ± 14	39 ± 10	0.518
Smoking status				0.098
Non-smoker	49 (62.0%)	29 (70.7%)	20 (52.6%)	
Current smoker	30 (38.0%)	12 (29.3%)	18 (47.4%)	

Alcohol consumption	12 (15.2%)	6 (14.6%)	6 (15.8%)	0.886
Pittsburgh Sleep Quality Index (PSQI)	3.2 ± 4.0	2.2 ± 3.0	4.2 ± 4.7	0.028
≤5 (good sleep quality)	52 (65.8%)	31 (75.6%)	21 (55.3%)	0.057
>5 (poor sleep quality)	27 (34.2%)	10 (24.4%)	17 (44.7%)	
Epworth Sleepiness Scale (ESS)	3.6 ± 5.0	2.3 ± 3.6	5.1 ± 5.9	0.016
<10 (normal)	60 (75.9%)	37 (90.2%)	23 (60.5%)	0.002
≥10 (excessive daytime sleepiness)	19 (24.1%)	4 (9.8%)	15 (39.5%)	
STOP-BANG questionnaire	1.6 ± 2.2	1.0 ± 1.6	2.3 ± 2.6	0.013
0–2 (low risk)	53 (67.1%)	31 (75.6%)	22 (57.9%)	0.009
3–4 (intermediate risk)	15 (19.0%)	9 (22.0%)	6 (15.8%)	
5–8 (high risk)	11 (13.9%)	1 (2.4%)	10 (26.3%)	
Hurley stage				NA
Stage I	20 (52.6%)	–	20 (52.6%)	
Stage II	4 (10.5%)	–	4 (10.5%)	
Stage III	14 (36.8%)	–	14 (36.8%)	
Dermatology Life Quality Index (DLQI)	2.5 (1–4)	–	2.5 (1–4)	NA
Body Mass Index (BMI, kg/m²)	25.8 ± 4.4	25.6 ± 5.7	26.0 ± 2.6	0.729
Hypertension (HT)	18 (22.8%)	9 (22.0%)	9 (23.7%)	0.854
Diabetes Mellitus (DM)	8 (10.1%)	6 (14.6%)	2 (5.3%)	0.266
IHS4 score	5.3 ± 4.4	–	5.3 ± 4.4	NA

Treatment				<0.001
Biologic therapy	16 (42.1%)	–	16 (42.1%)	
Antibiotics	13 (16.5%)	0 (0%)	13 (34.2%)	
Isotretinoin	9 (11.4%)	0 (0%)	9 (23.7%)	
Antidepressants	21 (26.6%)	7 (17.1%)	14 (36.8%)	0.047

Abbrevirations: HS, Hidradenitis Suppurativa; PSQI, Pittsburgh Sleep Quality Index; ESS, Epworth Sleepiness Scale; BMI, Body Mass Index; HT, Hypertension; DM, Diabetes Mellitus; IHS4, International Hidradenitis Suppurativa Severity Score System; DLQI, Dermatology Life Quality Index; OSA, Obstructive Sleep Apnea
Notes: Categorical variables are presented as number and percentage [n (%)].
Continuous variables are presented as mean ± standard deviation (SD) for normally distributed data and as median (interquartile range, IQR) for non-normally distributed data.

Table 2. Comparison of Laboratory and Inflammatory Parameters According to the Presence of Hidradenitis Suppurativa

Variables (N=79)	**Total**	**No HS (n=41)**	**HS (n=38)**	**p-value**
Laboratory Parameters				
LDL (mg/dL)	122 (93–146)	102 (85–125)	127 (109–166)	<0.001
HDL (mg/dL)	49.2 ± 15.3	58.3 ± 15.4	39.5 ± 6.9	<0.001
Triglycerides (mg/dL)	154.7 ± 100.7	120.4 ± 70.1	191.7 ± 115.7	0.002
Total cholesterol (mg/dL)	170 (132–211)	184 (159–214)	137.5 (102–209)	0.006
Fasting glucose (mg/dL)	90 (87–97)	91 (88–103)	88 (87–94)	0.024
HbA1c (%)	5.7 (5.2–6.3)	5.5 (5.2–6.0)	6.1 (5.4–6.6)	0.103
Systolic blood pressure (mmHg)	119 ± 9.6	114.9 ± 7.8	123.4 ± 9.4	<0.001
Diastolic blood pressure (mmHg)	80 (70–80)	75 (70–80)	80 (80–90)	<0.001
Neutrophil count (×10³/μL)	5.6 ± 2.3	4.7 ± 1.5	6.5 ± 2.6	<0.001
Lymphocyte count (×10³/μL)	2.3 ± 0.9	2.3 ± 0.7	2.3 ± 1.1	0.776

Platelet count (×10³/μL)	266.8 ± 72.6	237.6 ± 52.4	298.3 ± 78.6	<0.001
NLR	3.4 ± 3.2	2.5 ± 2.0	4.3 ± 3.9	0.011
PLR	106.4 (84.4–153.0)	109.8 (87.4–132.5)	102.7 (84.4–269.0)	0.336

Abbreviations: HS, Hidradenitis Suppurativa; LDL, Low-Density Lipoprotein; HDL, High-Density Lipoprotein; HbA1c, Hemoglobin A1c; NLR, Neutrophil-to-Lymphocyte Ratio; PLR, Platelet-to-Lymphocyte Ratio.

Note: Normally distributed continuous variables are presented as mean ± standard deviation (SD). Non-normally distributed continuous variables are presented as median (interquartile range, IQR).

Table 3. Univariate Logistic Regression Analysis of Factors Associated with the Presence of Hidradenitis Suppurativa

Variable	**OR (95% CI)**	**p-value**
Age	**0.988 (0.952–1.025)**	**0.518**
Sex		
Female	**Reference**	**–**
Male	**4.675 (1.799–12.148)**	**0.002**
BMI (kg/m²)	**1.019 (0.919–1.129)**	**0.725**
Smoking status		
Non-smoker	**Reference**	**–**
Current smoker	**2.175 (0.861–5.492)**	**0.100**
PSQI	**1.146 (1.013–1.296)**	**0.030**
HDL (mg/dL)	**0.839 (0.775–0.908)**	**<0.001**
NLR	**1.234 (1.040–1.465)**	**0.016**

Abbreviations OR, Odds Ratio; CI, Confidence Interval; BMI, Body Mass Index; PSQI, Pittsburgh Sleep Quality Index; HDL, High-Density Lipoprotein; NLR, Neutrophil-to-Lymphocyte Ratio; HS, Hidradenitis Suppurativa.

Note: Odds ratios (OR) and 95% confidence intervals (CI) were calculated using univariate logistic regression analysis. Continuous variables were entered into the model as continuous predictors.

Table 4. Multivariable Logistic Regression Analysis of Factors Associated with the Presence of Hidradenitis Suppurativa

Variable	Model 1		Model 2	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age (years)	0.948 (0.898–1.001)	0.056	1.012 (0.943–1.086)	0.751
Sex				
Female	Reference	–	Reference	–
Male	7.455 (1.933–28.753)	0.004	2.457 (0.524–11.521)	0.254
BMI (kg/m²)	0.944 (0.814–1.094)	0.444	0.870 (0.695–1.088)	0.223
Smoking status				
Non-smoker	Reference	–	–	–
Current smoker	0.533 (0.151–1.880)	0.328	–	–
PSQI	1.161 (0.966–1.396)	0.111	–	–
HDL (mg/dL)	–	–	0.818 (0.733–0.913)	<0.001
NLR	–	–	1.381 (1.022–1.868)	0.036

Model Definitions: Model 1 (Sleep model): Sex + Age + BMI + Smoking status + Pittsburgh Sleep Quality Index (PSQI)
Model 2 (Metabolic–inflammatory model): Sex + Age + BMI + High-Density Lipoprotein (HDL) + Neutrophil-to-Lymphocyte Ratio (NLR)

Abbreviations: OR, Odds Ratio; CI, Confidence Interval; BMI, Body Mass Index; PSQI, Pittsburgh Sleep Quality Index; HDL, High-Density Lipoprotein; NLR, Neutrophil-to-Lymphocyte Ratio; HS, Hidradenitis Suppurativa.

Note: Odds ratios (OR) and 95% confidence intervals (CI) were calculated using multivariable logistic regression analysis. Continuous variables were entered into the models as continuous predictors. Model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test. Multicollinearity was evaluated using the variance inflation factor (VIF).

Discussion

In this prospective controlled study, we demonstrate that patients with HS exhibit significantly impaired sleep quality, increased daytime sleepiness, and a markedly elevated risk of OSA compared with matched controls. Importantly, these alterations occur in the absence of significant differences in body mass index, suggesting that mechanisms beyond adiposity contribute to increased OSA vulnerability in HS.

Although data on HS and sleep disorders remain limited, recent evidence supports this relationship. A systematic review including over 343,000 subjects reported that HS patients have a higher likelihood of sleep disorders including OSA, and that sleep impairment may contribute to poorer quality of life and additional comorbidities in this population[10].

From a pulmonary perspective, our findings are clinically meaningful. STOP-BANG scores were significantly higher in the HS group, and the prevalence of high OSA risk was more than tenfold greater than in controls. Excessive daytime sleepiness was also substantially more frequent among HS patients. Consistent with these results, a recent case control study found that HS patients had poorer PSQI and STOP-BANG scores compared to non HS dermatologic controls[11].

Inflammation as a Shared Pathophysiological Axis

OSA is now understood not only as an upper airway mechanical disorder but also as a systemic disorder characterized by intermittent hypoxia, oxidative stress, endothelial dysfunction, and chronic low-grade inflammation[12]. Similarly, HS is increasingly recognized as a systemic inflammatory disease driven by dysregulated innate immunity and elevated pro inflammatory cytokines.

In our cohort, HS patients exhibited significantly higher neutrophil counts, platelet counts, and neutrophil to lymphocyte ratio (NLR) values, reflecting a pro inflammatory immune phenotype. NLR remained independently associated with HS in multivariable analysis, reinforcing its role as a marker of systemic inflammatory burden.

Intermittent hypoxia in OSA promotes activation of nuclear factor-κB pathways, sympathetic overactivity, and cytokine release mechanisms that overlap with those implicated in HS. Thus, systemic inflammation may represent a bidirectional amplifier linking HS and OSA risk. Rather than obesity alone, a chronic inflammatory milieu may predispose HS patients to sleep disordered breathing through altered ventilatory control, endothelial dysfunction, and upper airway neuromuscular instability. This concept is supported by the notion that elevated inflammation is a shared pathophysiological axis across systemic disorders and OSA risk[12].

Metabolic Dysregulation and OSA Risk

Beyond inflammation, HS patients in our study demonstrated a distinctly atherogenic lipid profile characterized by significantly lower HDL and higher LDL and triglyceride levels. HDL remained independently and inversely associated with HS in multivariable analysis.

Low HDL is more than a marker of dyslipidemia; it reflects impaired anti-inflammatory and endothelial-protective functions. Reduced HDL levels may contribute to vascular dysfunction and increased upper airway collapsibility via impaired nitric oxide bioavailability and heightened sympathetic tone key mechanisms implicated in OSA pathophysiology.

Importantly, BMI did not differ between groups and was not independently associated with HS in regression models. This finding suggests that increased OSA risk in HS patients may be mediated by metabolic inflammatory mechanisms rather than generalized obesity. For pulmonologists, this is particularly relevant: HS patients with normal or mildly elevated BMI may still harbor a significant risk of OSA.

Sleep Quality Versus OSA Risk

Although PSQI scores were higher in HS patients, sleep quality did not remain independently associated with HS in multivariable analysis. This suggests that impaired sleep quality may be a downstream consequence of inflammatory or metabolic dysregulation rather than an independent driver of HS itself.

In contrast, the elevated STOP-BANG scores highlight a structural and physiological vulnerability to OSA. The STOP-BANG questionnaire has been validated as a highly sensitive screening tool for OSA risk across diverse populations, and scores ≥3 are consistently associated with increased likelihood of moderate to severe OSA[13]. Taken together, these findings support the concept that HS is associated not only with subjective sleep impairment but also with a biologically plausible risk of sleep disordered breathing.

Clinical Implications for Pulmonary Practice

From a respiratory medicine standpoint, our results raise important clinical considerations:

1. HS patients may represent a high-risk group for undiagnosed OSA.
2. OSA screening using validated tools such as STOP-BANG should be considered in HS patients, especially those with elevated NLR or low HDL levels.
3. Early identification of OSA in this population may mitigate cardiovascular risk and systemic inflammatory burden.

These recommendations align with recent dermatology studies reporting higher STOP-BANG and PSQI scores among HS patients compared to other chronic inflammatory dermatologic populations (e.g., psoriasis), highlighting the importance of routine assessment for sleep disorders in HS[14].

Given the bidirectional interaction between inflammation and intermittent hypoxia, untreated OSA may further amplify systemic inflammation in HS, potentially worsening disease severity and cardiometabolic risk. This underscores the need for interdisciplinary collaboration between dermatology and pulmonology to optimize comprehensive care.

Strengths and Limitations

The strengths of our study include its prospective design, controlled comparison group, and comprehensive evaluation of sleep, metabolic, and inflammatory parameters within a unified framework.

However, several limitations should be acknowledged. OSA risk was assessed using validated questionnaires rather than polysomnography. While STOP-BANG is widely used for risk stratification, objective sleep studies would provide more definitive diagnostic confirmation. Additionally, the sample size was modest, which may limit statistical power for certain subgroup analyses.

Future studies incorporating overnight polysomnography and longitudinal follow-up are warranted to determine whether treatment of OSA favorably modifies inflammatory burden or clinical outcomes in HS.

Conclusion

In conclusion, hidradenitis suppurativa is associated with impaired sleep quality and a markedly increased risk of obstructive sleep apnea independent of body mass index. This increased vulnerability appears to be driven by a systemic inflammatory and atherogenic metabolic phenotype characterized by elevated NLR and reduced HDL levels.

These findings suggest that HS should be considered not only a dermatologic condition but also a systemic inflammatory disorder with pulmonary and sleep-related implications. Recognition of OSA risk in HS patients may enable earlier intervention and improved cardiopulmonary outcomes.

Declarations

Ethics Approval and Consent to Participate

Ethical approval for this prospective observational study was obtained from the Istanbul Medipol University Non-Interventional Clinical Research Ethics Committee (Approval Date: 15 December 2026; Decision No: E-10840098-202.3.02-8425).

The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

Consent for Publication

Not applicable.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

Funding

No specific funding was received for this study.

Authors' Contributions

GT and MD conceptualized and designed the study. GT, MD and ŞB were involved in patient recruitment, clinical evaluation, and data collection. ÖA, as a dermatologist, contributed to the dermatological assessment, confirmation of HS diagnoses, and evaluation of disease severity. GT performed the statistical analysis. GT drafted the initial manuscript. All authors contributed to the interpretation of the data, critically revised the manuscript for important intellectual content, and approved the final version.

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Clinical Trial Registration

Not applicable.

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