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Relationship Between Hemoglobin-Albumin-Lymphocyte-Platelet Score and Obstructive Sleep Apnea Severity

Hemoglobin-Albumin-Lenfosit-Trombosit Skoru ile Obstrüktif Uyku Apnesi Şiddeti Arasındaki İlişki

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Abstract

Objective: Obstructive sleep apnea (OSA) syndrome is a common disease characterized by chronic intermittent hypoxia and systemic inflammation. We aimed to evaluate the relationship between OSA severity and the hemoglobin-albumin-lymphocyte-platelet (HALP) score, systemic immune-inflammation index (SII), and systemic inflammation response index (SIRI).

Materials and Methods: Patients who underwent polysomnography at University of Health Sciences Türkiye, Kayseri City Hospital between 2022 and 2025 and met the criteria were included. OSA severity was determined according to the apnea-hypopnea index (AHI). HALP score, SII, and SIRI were calculated from blood parameters and compared across severity groups, and their correlations with AHI were assessed. Separate multivariable ordinal logistic regression models adjusted for age, sex, and body mass index (BMI) were used to evaluate the associations of these indices with OSA severity.

Results: Among 257 participants, HALP differed across groups ($p = 0.018$) and was higher in severe OSA than in simple snoring (adjusted $p = 0.014$). SII and SIRI did not differ significantly across groups ($p = 0.346$ and $p = 0.120$, respectively). After adjustment, HALP, SII, and SIRI were not independently associated with OSA severity ($p = 0.242$, $p = 0.206$, and $p = 0.053$, respectively). AHI correlated weakly with HALP (rho = 0.220; $p < 0.001$) and SIRI (rho = 0.140; $p = 0.025$).

Conclusion: HALP was higher in severe OSA than in simple snoring and showed a weak positive correlation with AHI; however, it was not independently associated with OSA severity after adjustment for age, sex, and BMI. These findings suggest that HALP may reflect OSA-related inflammatory and nutritional changes but has limited value as an independent marker of disease severity.

Keywords: Inflammatory indices, polysomnography, obstructive sleep apnea

Öz

Amaç: Obstrüktif uyku apnesi (OSA) sendromu, kronik aralıklı hipoksi ve sistemik inflamasyon ile ilişkili yaygın bir hastalıktır. Bu çalışmada, hemoglobin-albümin-lenfosit-trombosit (HALP) skoru ile sistemik immün-enflamasyon indeksi (Sİİ) ve sistemik inflamasyon yanıt indeksi (SİRİ) gibi inflamatuvar belirteçlerin OSA şiddeti ile ilişkisinin değerlendirilmesi amaçlandı.

Gereç ve Yöntem: 2022-2025 yılları arasında Sağlık Bilimleri Üniversitesi, Kayseri Şehir Hastanesi'nde polisomnografi uygulanan ve kriterleri karşılayan hastalar çalışmaya dahil edildi. OSA şiddeti, apne-hipopne indeksine (AHİ) göre belirlendi. HALP skoru, Sİİ ve SİRİ kan parametrelerinden hesaplanarak şiddet grupları arasında karşılaştırıldı ve AHİ ile korelasyonları değerlendirildi. Bu indekslerin OSA şiddeti ile ilişkilerini değerlendirmek için yaş, cinsiyet ve vücut kitle indeksine (VKİ) göre düzeltilmiş ayrı çok değişkenli ordinal lojistik regresyon modelleri kullanıldı.

Bulgular: Toplam 257 katılımcı arasında HALP, gruplar arasında farklılık gösterdi ($p = 0,018$) ve ağır OSA grubunda basit horlama grubuna göre daha yüksekti (düzeltilmiş $p = 0,014$). Sİİ ve SİRİ, gruplar arasında anlamlı farklılık göstermedi (sırasıyla $p = 0,346$ ve $p = 0,120$). Düzeltme sonrasında HALP, Sİİ ve SİRİ, OSA şiddeti ile bağımsız olarak ilişkili değildi (sırasıyla $p = 0,242$, $p = 0,206$ ve $p = 0,053$). AHİ, HALP (rho = 0,220; $p < 0,001$) ve SİRİ (rho = 0,140; $p = 0,025$) ile zayıf düzeyde korelasyon gösterdi.

Sonuç: HALP, ağır OSA grubunda basit horlama grubuna göre daha yüksekti ve AHİ ile zayıf pozitif korelasyon gösterdi; ancak yaş, cinsiyet ve VKİ'ye göre düzeltme sonrasında OSA şiddeti ile bağımsız olarak ilişkili değildi. Bu bulgular, HALP'nin OSA ile ilişkili enflamatuvar ve nütrisyonel değişiklikleri yansıtabileceğini, ancak hastalık şiddetinin bağımsız bir belirteci olarak sınırlı değere sahip olduğunu düşündürmektedir.

Anahtar Kelimeler: Enflamatuvar indeksler, polisomnografi, obstrüktif uyku apnesi

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Introduction

Obstructive sleep apnea syndrome (OSAS) is a significant public health problem with increasing prevalence, characterized by recurrent complete (apnea) or partial (hypopnea) obstructions of the upper airway during sleep. Chronic intermittent hypoxia is the primary pathophysiological mechanism of the disease and is the main factor triggering systemic inflammation, increasing oxidative stress, and leading to endothelial dysfunction.[1,2] This process predisposes patients with OSAS to the development of cardiovascular and cerebrovascular diseases such as hypertension, coronary artery disease, and stroke.[3,4]

Although polysomnography (PSG) and the apnea-hypopnea index (AHI) are currently the gold standard methods for determining the severity of OSA, the AHI may be insufficient to directly reflect the systemic inflammatory burden or atherothrombotic risk in patients with OSAS.[5] Therefore, there is a need for readily available, inexpensive, and reliable biomarkers that can predict the severity and prognosis of the disease. Although simple hematological parameters such as the neutrophil/lymphocyte ratio (NLR) have been frequently studied in recent years, single ratios like NLR may be insufficient to reflect complex inflammatory processes. As a result, composite indices including multiple cell lines and nutritional parameters have come to the fore.[6] Among these indices, the systemic immune-inflammation index (SII) and the systemic inflammation response index (SIRI) show the balance between inflammatory and immune responses, with SII additionally reflecting thrombosis through its platelet component. The hemoglobin-albumin-lymphocyte-platelet (HALP) score is a more comprehensive marker that incorporates the patient's nutritional status and blood viscosity in addition to inflammation. These indices may serve as cost-effective biomarkers in identifying chronic inflammation, although conflicting results have been obtained in this regard.[7-9] In addition, inflammation is thought to disrupt sleep patterns, as secreted inflammatory mediators can cross the blood-brain barrier and affect sleep regulation. These markers can therefore also be used as predictors of OSA, which is closely associated with chronic inflammation.[10]

Considering the secondary polycythemia (increased hemoglobin) and oxidative stress-related albumin decrease that can occur in OSAS, the HALP score may be closely linked to the pathophysiology of OSAS. The HALP score has mainly been used in research involving patient populations such as those with malignancy or receiving intensive care. Although SII and SIRI have been studied in patients with OSAS, the HALP score has been evaluated in very few studies.[11] Obesity, which is highly prevalent in patients with OSAS, is itself a pro-inflammatory condition. Therefore, this study aimed to include patients without morbid obesity, systemic disease, or active inflammatory/infectious disease in order to more objectively determine the inflammatory response created by OSA-related hypoxemia alone. The study also aimed to determine the levels of these composite indices in this population group and to examine their relationship with OSA severity.

Materials and Methods

Study Design and Patient Selection

The medical records of patients who presented to the Sleep Disorders Laboratory of University of Health Sciences Türkiye, Kayseri City Hospital between March 2022 and December 2025 and underwent overnight PSG were retrospectively evaluated. Ethical approval for the study was obtained from the University of Health Sciences Türkiye, Kayseri City Hospital Non-Interventional Clinical Research Ethics Committee (approval no: 753, date: 14.01.2026). Due to the retrospective nature of the study and the use of anonymized data, the requirement for informed consent was waived by the ethics committee.

Complete blood count and routine biochemical tests were performed on blood samples collected the morning after the PSG night, after at least 8 hours of fasting. Patients with incomplete or missing laboratory data were excluded from the study. On admission for the PSG night, a detailed medical history, including systemic diseases and current medications, was recorded manually in each patient's file. These files were reviewed, and patients actively receiving antibiotic therapy were excluded because of possible active infection. Additionally, patients under 18 years of age, those with sleep efficiency below 50% on PSG, those with active infections, known malignancies, chronic inflammatory/rheumatological diseases, diabetes mellitus, or hypertension, those taking regular medications, morbidly obese patients [body mass index (BMI) >40 kg/m^2], and those with severe renal/hepatic impairment were excluded from the study. A total of 257 patients meeting these selection criteria were included in the study.

Polysomnography (Sleep Study)

PSG recordings were obtained using a Philips Respironics® system. The system consisted of six-channel electroencephalography (F3/M2, C3/M2, O1/M2, F4/M1, C4/M1, O2/M1), two electrooculography channels, chin and bilateral tibialis anterior muscle electromyography, an oronasal thermistor, a nasal cannula, a snoring sensor, thoracic and abdominal respiratory effort belts, electrocardiography, and pulse oximetry. PSG was conducted in the laboratory between 23:00 (lights off) and 07:00 (lights on). All recordings were scored by an experienced and certified sleep specialist (D.K.G.) in accordance with the American Academy of Sleep Medicine guidelines.[12] Apnea was defined as a reduction in airflow of 90% or more lasting at least 10 seconds. Hypopnea was defined as a 30% or greater reduction in airflow lasting at least 10 seconds and resulting in at least 3% desaturation or arousal. The AHI was calculated by dividing the total number of apneas and hypopneas by total sleep time in hours. The oxygen desaturation index (ODI) was defined as the number of episodes of a ≥3% decrease in oxygen saturation per hour of sleep. Based on the AHI, patients were divided into four groups: Group 1: simple snoring (control; AHI <5), Group 2: mild OSA (5≤ AHI <15), Group 3: moderate OSA (15≤ AHI <30), Group 4: severe OSA (AHI ≥30).

Calculation of Inflammatory Indices

Hemoglobin and serum albumin concentrations and leukocyte, neutrophil, lymphocyte, monocyte, and platelet counts were recorded. The indices were calculated using the following formulas:

- SII: platelet count × neutrophil count/lymphocyte count,
- SIRI: neutrophil count × monocyte count/lymphocyte count,
- HALP score: hemoglobin (g/L) × albumin (g/L) × lymphocyte count (/L)/platelet count (/L).

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Normality was assessed using histograms, Q-Q plots, and the Shapiro-Wilk test, and homogeneity of variances was assessed using Levene's test. Because the continuous variables were not normally distributed, they are presented as median (Q1-Q3) and were compared using the Kruskal-Wallis test. When the overall test was significant, pairwise comparisons were performed using the Dunn-Bonferroni post-hoc test. Categorical variables are presented as n (%) and were compared using Pearson's chi-square test, followed, when appropriate, by Bonferroni-adjusted pairwise comparisons. Separate multivariable ordinal logistic regression models were constructed for HALP, SII, and SIRI, with ordered OSA severity (simple snoring, mild, moderate, and severe OSA) as the dependent variable. Each model was adjusted for age, sex, and BMI. HALP was scaled per 10-point increase, SII per 100-unit increase, and SIRI per 1-unit increase. Results are presented as odds ratios (ORs) with 95% confidence intervals (CIs). The proportional-odds assumption was assessed using the test of parallel lines. Correlations between AHI and the inflammatory indices were evaluated using Spearman's rank correlation analysis. All tests were two-sided, and $p < 0.05$ was considered statistically significant.

Results

A total of 257 patients were included in the study and divided into four groups: simple snoring (n = 90), mild OSA (n = 53), moderate OSA (n = 51), and severe OSA (n = 63). The demographic and polysomnographic characteristics of the patients are presented in Table 1.

Age differed significantly among the groups ($p = 0.005$). Dunn-Bonferroni post-hoc analysis showed that patients in the simple snoring group were younger than those in the moderate and severe OSA groups (adjusted $p = 0.016$ and $p = 0.020$, respectively). No other pairwise differences were statistically significant.

BMI differed significantly among the groups ($p < 0.001$). Dunn-Bonferroni post-hoc analyses showed that BMI was significantly higher in the mild, moderate, and severe OSA groups than in the simple snoring group (adjusted $p = 0.030$, $p < 0.001$, and $p < 0.001$, respectively). BMI did not differ significantly among the OSA severity groups.

Sex distribution also differed significantly among the four groups ($p = 0.001$). Bonferroni-adjusted pairwise comparisons demonstrated that the only significant difference was between severe OSA and simple snoring ($p < 0.001$). Severe OSA was more prevalent among male than female participants (28.7% vs. 11.3%, $p = 0.009$).

When sleep architecture was evaluated, the percentages of N1, N3, and REM sleep differed significantly among the groups ($p = 0.002$, $p = 0.021$, and $p = 0.005$, respectively), whereas

Table 1. Demographic, sleep architecture, and respiratory characteristics according to OSA severity.

Variable	Simple snoring (n = 90)	Mild OSA (n = 53)	Moderate OSA (n = 51)	Severe OSA (n = 63)	p-value
Age, years	34.0 (26.8–38.3)[a]	37.0 (32.0–39.0)[ab]	37.0 (33.0–40.0)[b]	37.0 (33.0–40.0)[b]	0.005
Male sex, n (%)	56 (62.2)[a]	42 (79.2)[ab]	41 (80.4)[ab]	56 (88.9)[b]	0.001
BMI, kg/m^2	26.3 (24.3–27.7)[a]	27.0 (26.1–28.7)[b]	28.7 (26.6–29.8)[b]	28.7 (27.2–29.8)[b]	<0.001
Total sleep time, min	376.0 (320.3–409.0)	400.5 (353.3–427.0)	357.0 (312.0–423.5)	387.5 (323.0–415.0)	0.076
Sleep efficiency, %	88.1 (72.1–93.0)	88.4 (80.2–93.6)	86.7 (73.2–93.5)	86.8 (77.2–91.7)	0.667
Sleep onset latency, min	18.0 (10.4–32.4)	17.5 (9.8–31.5)	19.5 (10.0–35.0)	15.0 (8.0–32.5)	0.779
REM latency, min	165.5 (97.8–246.8)	137.5 (85.9–247.8)	146.8 (87.4–211.8)	159.0 (118.9–228.6)	0.446
Minimum O_2 saturation, %	88.0 (85.0–90.0)[a]	85.0 (82.0–87.0)[b]	81.0 (78.0–85.0)[c]	78.0 (71.0–82.0)[d]	<0.001
Mean O_2 saturation, %	94.0 (92.5–94.5)[b]	93.0 (92.0–94.0)[b]	92.0 (90.0–93.0)[a]	91.0 (89.5–93.0)[a]	<0.001
ODI, events/h	3.0 (1.0–4.5)[a]	10.2 (7.7–12.4)[b]	21.3 (18.3–27.3)[c]	48.2 (37.7–70.0)[d]	<0.001
Arousal index, events/h	10.0 (5.2–15.5)[a]	12.2 (8.1–16.2)[ab]	13.6 (9.9–22.2)[b]	14.0 (7.6–23.7)[b]	0.005
N1 sleep, %	4.3 (2.7–5.8)[a]	4.8 (3.6–6.9)[ab]	5.5 (4.0–7.0)[b]	5.9 (3.6–8.1)[b]	0.002
N2 sleep, %	51.0 (44.0–58.9)	49.8 (45.4–56.9)	51.0 (41.7–57.8)	55.4 (42.4–66.2)	0.510
N3 sleep, %	15.4 (11.2–21.1)[ab]	16.8 (12.4–20.4)[a]	14.7 (11.2–18.3)[ab]	13.3 (6.0–18.0)[b]	0.021
REM sleep, %	9.9 (4.4–14.7)[ab]	13.0 (8.8–15.5)[b]	10.8 (6.6–15.8)[b]	8.4 (3.9–11.9)[a]	0.005

Continuous variables are presented as median (Q1-Q3) and were compared using the Kruskal-Wallis test, followed by Dunn-Bonferroni post-hoc comparisons when the overall test was significant. Categorical variables are presented as n (%) and were compared using Pearson's chi-square test, followed by Bonferroni-adjusted pairwise comparisons. Different superscript letters indicate a statistically significant difference; values sharing at least one letter are not significantly different.

BMI: Body mass index, ODI: Oxygen desaturation index, OSA: Obstructive sleep apnea, REM: Rapid eye movement.

the percentage of N2 sleep did not (p = 0.510). In the Dunn-Bonferroni post-hoc analyses, the N1 sleep percentage was higher in the moderate and severe OSA groups than in the simple snoring group (adjusted p = 0.025 and p = 0.002, respectively). The N3 sleep percentage was lower in the severe OSA group than in the mild OSA group (adjusted p = 0.032). Similarly, the REM sleep percentage was lower in the severe OSA group than in the mild and moderate OSA groups (adjusted p = 0.005 and p = 0.041, respectively). Minimum O_2 saturation, mean O_2 saturation, and ODI differed significantly among the groups (all p-values < 0.001). Post-hoc pairwise comparisons showed that ODI was higher in the severe OSA group than in the other groups. Minimum O_2 saturation was lowest in the severe OSA group, and mean O_2 saturation was lower in the moderate and severe OSA groups than in the simple snoring and mild OSA groups. The arousal index also differed significantly among the groups (p = 0.005); post-hoc pairwise comparisons showed that it was higher in the moderate and severe OSA groups than in the simple snoring group (adjusted p-values, respectively, p = 0.011 and p = 0.024).

The distributions of the inflammatory indices are presented in Table 2. HALP scores differed significantly among the groups (p = 0.018). Dunn-Bonferroni post-hoc analysis showed that the HALP score was significantly higher in the severe OSA group than in the simple snoring group [74.0 (61.3–100.4) vs. 63.5 (48.6–85.6); adjusted p = 0.014]. No other pairwise differences in HALP scores were statistically significant. SII and SIRI did not differ significantly among the groups (p = 0.346 and p = 0.120, respectively); therefore, no post-hoc comparisons were performed for these indices.

Separate multivariable ordinal logistic regression models were constructed to evaluate the associations of HALP, SII, and SIRI with ordered OSA severity after adjustment for age, sex, and BMI (Table 3). None of the inflammatory indices was independently associated with OSA severity: HALP, OR 1.038 per 10-point increase (95% CI: 0.975–1.105; p = 0.242); SII, OR 1.042 per 100-unit increase (95% CI: 0.977–1.110; p = 0.206); and SIRI, OR 1.286 per 1-unit increase (95% CI: 0.997–1.660; p = 0.053). Age, male sex, and BMI were independently associated with greater OSA severity in all three models (all p < 0.001). All three models were statistically significant (model χ^2 = 79.982–83.183; all p < 0.001), with Nagelkerke R^2 values ranging from 0.286 to 0.296. The parallel-lines tests were not significant, although their validity should be interpreted cautiously because SPSS reported convergence-related warnings for the general models.

Spearman correlation analysis showed weak positive correlations between AHI and HALP (rho = 0.220; p < 0.001) and between AHI and SIRI (rho = 0.140; p = 0.025). AHI was not significantly correlated with SII (rho = −0.053; p = 0.396) (Table 4).

Discussion

This study investigated the relationship between OSA severity and composite inflammatory indices after excluding morbid obesity and major comorbidities that may affect systemic inflammation. HALP differed significantly across the four groups and was higher in severe OSA than in simple snoring, whereas SII and SIRI did not differ significantly. HALP and SIRI showed weak positive correlations with AHI. However, none of the three indices was independently associated with ordered OSA severity after adjustment for age, sex, and BMI. These findings suggest that HALP may reflect OSA-related inflammatory and nutritional changes, but its ability to provide information on disease severity beyond established demographic and anthropometric factors is limited.

In the unadjusted group comparison, HALP was significantly higher in severe OSA than in simple snoring, and it showed a weak positive correlation with AHI. The HALP score is a composite index containing both inflammatory and nutritional components, calculated using HALP values.[8] In OSA, chronic intermittent hypoxia resulting from recurrent upper airway obstructions leads to sympathetic activation, oxidative stress, and systemic inflammation.[1] Furthermore, hypoxia-induced erythropoietin production can increase hemoglobin levels.[13] The influence of OSA-related pathophysiological processes on the components of the HALP score may explain the higher HALP values observed in patients with severe OSA. In the literature, the HALP score has mainly been investigated in conditions such as malignancies and critical illnesses, and the number of studies evaluating its relationship with OSA is quite limited.[11] Our findings suggest that the HALP score may reflect the inflammatory burden associated with OSA. Nevertheless, the weak correlation and the loss of statistical significance after multivariable adjustment indicate that the unadjusted HALP difference should not be interpreted as evidence of a strong or independent dose-response relationship with OSA severity.

However, unlike the HALP score, there was no significant difference in SII or SIRI among the four groups in our study. SIRI showed a very weak positive correlation with AHI and a borderline, but statistically nonsignificant, association with ordered OSA severity in the adjusted model. This apparent discrepancy between

Table 2. Inflammatory markers according to OSA severity.

Variable	Simple snoring (n = 90)	Mild OSA (n = 53)	Moderate OSA (n = 51)	Severe OSA (n = 63)	p-value
HALP score	63.5 (48.6–85.6)[a]	69.1 (59.0–90.2)[ab]	72.6 (58.9–93.7)[ab]	74.0 (61.3–100.4)[b]	0.018
SII	457.7 (350.6–601.5)	401.3 (287.1–555.9)	400.6 (313.5–568.2)	453.7 (294.6–611.0)	0.346
SIRI	0.95 (0.71–1.34)	0.92 (0.68–1.42)	0.98 (0.73–1.67)	1.11 (0.76–1.82)	0.120

Values are presented as median (Q1-Q3) and were compared using the Kruskal-Wallis test. Dunn-Bonferroni post hoc comparisons were performed when the overall test was statistically significant. Different superscript letters indicate a statistically significant difference; values sharing at least one letter are not significantly different.

HALP: Hemoglobin-albumin-lymphocyte-platelet score, OSA: Obstructive sleep apnea, SII: Systemic immune-inflammation index, SIRI: Systemic inflammation response index.

Table 3. Multivariable ordinal logistic regression models for OSA severity.

Model	Predictor	Adjusted OR	95% CI	p-value	Nagelkerke R^2	Model χ^2	Parallel-lines p
HALP	HALP, per 10 points	1.038	0.975–1.105	0.242	0.286	79.982	0.263†
	Age, per year	1.068	1.029–1.107	<0.001			
	Male sex	3.104	1.680–5.732	<0.001			
	BMI, per 1 kg/m²	1.356	1.232–1.492	<0.001			
SII	SII, per 100 units	1.042	0.977–1.110	0.206	0.287	80.338	0.515†
	Age, per year	1.071	1.033–1.112	<0.001			
	Male sex	3.667	2.032–6.619	<0.001			
	BMI, per 1 kg/m²	1.366	1.241–1.502	<0.001			
SIRI	SIRI, per 1 unit	1.286	0.997–1.660	0.053	0.296	83.183	0.494†
	Age, per year	1.073	1.034–1.113	<0.001			
	Male sex	3.493	1.946–6.271	<0.001			
	BMI, per 1 kg/m²	1.368	1.242–1.505	<0.001			

The dependent variable was ordered OSA severity: simple snoring, mild OSA, moderate OSA, and severe OSA. Each inflammatory index was entered in a separate model adjusted for age, sex, and BMI. The model χ^2 had 4 degrees of freedom and $p < 0.001$ in all models. †SPSS reported that the validity of the parallel-lines likelihood-ratio test was uncertain because the general model reached the maximum number of step-halving iterations; the reported p-values should therefore be interpreted cautiously.

OSA: Obstructive sleep apnea, OR: Odds ratio, CI: Confidence interval, BMI: Body mass index, HALP: Hemoglobin-albumin-lymphocyte-platelet score, SII: Systemic immune-inflammation index, SIRI: Systemic inflammation response index.

Table 4. Spearman correlations between AHI and inflammatory indices (n = 257).

Inflammatory index	Spearman rho	p-value
HALP	0.220	<0.001
SII	−0.053	0.396
SIRI	0.140	0.025

AHI: Apnea-hypopnea index, HALP: Hemoglobin-albumin-lymphocyte-platelet score, SII: Systemic immune-inflammation index, SIRI: Systemic inflammation response index.

the correlation and group comparison results may reflect the distinct information provided by categorical and continuous analyses: a weak monotonic relationship with AHI may not produce clearly separated distributions across the four severity groups. More importantly, the adjusted result did not meet the prespecified threshold for statistical significance. Therefore, the SIRI findings should be considered exploratory and should not be interpreted as evidence of an independent association. Although some studies in recent years have reported that SII and SIRI may be related to the severity of OSA, different studies have obtained different results.[11,14] These discrepancies may be due to differences in patient population selection criteria or accompanying comorbidities, which may also account for our findings. The exclusion of conditions that can affect systemic inflammation, such as diabetes, hypertension, active infection, chronic inflammatory diseases, and morbid obesity, may have reduced the inflammatory differences among groups. This approach may also have allowed for a more objective assessment of the inflammatory effect caused by OSA alone, while reducing the sensitivity of indices such as SII and SIRI.

Our findings partially differ from those of a recent study by Kayali Dinç et al.[11] investigating the relationship between HALP and OSA. That study found no significant association between OSA severity and several hematological inflammation indices, including HALP. In our study, HALP was higher in severe OSA than in simple snoring in the unadjusted comparison. This difference may be explained by the characteristics of the study populations. Our study aimed to evaluate the inflammatory effects of OSA in a more homogeneous population by excluding conditions that could affect systemic inflammation. However, HALP was not independently associated with ordered OSA severity after adjustment for age, sex, and BMI. Taken together, these findings indicate that HALP may capture changes accompanying OSA but does not appear to add independent information on severity beyond established clinical factors.

Age, male sex, and higher BMI were independently associated with greater OSA severity in all three adjusted ordinal models. The association with male sex is consistent with the existing literature and may be related to differences in upper airway anatomy, fat distribution, and hormonal factors.[15] The association between BMI and OSA severity was also expected, because fat accumulation around the upper airway increases the tendency toward pharyngeal collapse.[16] Although patients with morbid obesity were excluded, BMI still varied within the study population. The consistent associations of age, sex, and BMI across all models suggest that these established demographic and anthropometric factors explain substantially more variation in OSA severity than the inflammatory indices examined in this study.

One of the clinically relevant findings of the present study is that the HALP score, calculated from routinely available and inexpensive laboratory parameters, was higher in severe OSA than in simple snoring in the unadjusted analysis. While PSG remains the gold standard method for diagnosing OSA, biomarkers like HALP may help identify high-risk patients. However, given that HALP is not independently associated with OSA severity, it seems more appropriate to consider this index as a tool to support clinical evaluation rather than as a diagnostic

tool. Biomarkers calculated from routine laboratory parameters may also contribute to identifying high-risk individuals in centers lacking PSG capabilities. Another important aspect of this study is the exclusion of conditions that can affect systemic inflammation, which allowed for the evaluation of the inflammatory effects of OSA in a more homogeneous population.

Study Limitations

The main limitation of our study is its retrospective and single-center design. In addition, the inflammatory indices were assessed at only a single time point, and their dynamic changes were not prospectively monitored. Furthermore, advanced inflammatory markers such as interleukin-6 or tumor necrosis factor-α were not included in the study. Another limitation is the exclusion of diabetes, hypertension, and other comorbid diseases from the study population. Nevertheless, this approach allowed for a more objective assessment of the inflammatory effects attributable to OSA itself by minimizing the potential confounding influence of comorbid diseases.

Conclusion

In conclusion, HALP was higher in severe OSA than in simple snoring and showed a weak positive correlation with AHI; however, it was not independently associated with OSA severity after adjustment for age, sex, and BMI. SII and SIRI likewise showed no independent associations with OSA severity. These results suggest that HALP may reflect OSA-related inflammatory and nutritional changes, but its value as an independent marker of disease severity is limited. Larger prospective studies are required to determine whether HALP provides incremental clinical information beyond established risk factors and polysomnographic measures.

Ethics

Ethics Committee Approval: Ethical approval for the study was obtained from the University of Health Sciences Türkiye, Kayseri City Hospital Non-Interventional Clinical Research Ethics Committee (approval no: 753, date: 14.01.2026).

Informed Consent: Due to the retrospective nature of the study and the use of anonymized data, the requirement for informed consent was waived by the ethics committee.

Footnotes

Authorship Contributions

Surgical and Medical Practices: D.K.G., Concept: D.K.G., M.G., Design: D.K.G., Data Collection or Processing: D.K.G., Analysis or Interpretation: M.G., Literature Search: M.G., Writing: D.K.G.

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References

1. Sforza E, Roche F. Chronic intermittent hypoxia and obstructive sleep apnea: an experimental and clinical approach. Hypoxia (Auckl). 2016;4:99-108. doi: 10.2147/HP.S103091.
2. Janaszak-Jasiecka A, Siekierzycka A, Płoska A, Dobrucki IT, Kalinowski L. Endothelial dysfunction driven by hypoxia-the ınfluence of oxygen deficiency on NO bioavailability. Biomolecules. 2021;11(7):982. doi: 10.3390/biom11070982.
3. Jehan S, Farag M, Zizi F, et al. Obstructive sleep apnea and stroke. Sleep Med Disord. 2018;2(5):120-125.
4. Yeghiazarians Y, Jneid H, Tietjens JR, et al. Obstructive sleep apnea and cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2021;144(3):e56-e67. Erratum in: Circulation. 2022;145(12):e775. doi: 10.1161/CIR.0000000000000988.
5. Malhotra A, Ayappa I, Ayas N, et al. Metrics of sleep apnea severity: beyond the apnea-hypopnea index. Sleep. 2021;44(7):zsab030. doi: 10.1093/sleep/zsab030.
6. Huang H, Wang W, Ma Q, Cao K. Systemic inflammation-based hematological indices and 90-day functional outcomes after intravenous thrombolysis in acute ischemic stroke: a systematic review. Front Neurol. 2025;16:1699939. doi: 10.3389/fneur.2025.1699939.
7. Wei X, Zhang Z, Wei J, Luo C. Association of systemic immune inflammation index and system inflammation response index with clinical risk of acute myocardial infarction. Front Cardiovasc Med. 2023;10:1248655. doi: 10.3389/fcvm.2023.1248655.
8. Farag CM, Antar R, Akosman S, Ng M, Whalen MJ. What is hemoglobin, albumin, lymphocyte, platelet (HALP) score? A comprehensive literature review of HALP's prognostic ability in different cancer types. Oncotarget. 2023;14:153-172. doi: 10.18632/oncotarget.28367.
9. He G, Deng M, Tong Y, et al. Systemic immune-inflammation index and systemic inflammation response index: novel hematological predictors of obstructive sleep apnea. Front Neurol. 2026;17:1716598. doi: 10.3389/fneur.2026.1716598.
10. Aihara K, Oga T, Chihara Y, et al. Analysis of systemic and airway inflammation in obstructive sleep apnea. Sleep Breath. 2013;17(2):597-604. doi: 10.1007/s11325-012-0726-y.
11. Kayali Dinç AS, Gülhan Yaşar N, Yeniçeri A, Ağgünlü İ, Yurtsever Kum N, Çayönü M. Role of hematological parameters and inflammatory indices in determining the severity of obstructive sleep apnea syndrome: reality or myth? Eur Arch Otorhinolaryngol. 2025;282(8):4343-4348. doi: 10.1007/s00405-025-09528-0.
12. Troester MM, Quan SF, Berry RB, et al. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications (version 3). American Academy of Sleep Medicine; 2023.
13. Zhang XB, Zeng YM, Zeng HQ, Zhang HP, Wang HL. Erythropoietin levels in patients with sleep apnea: a meta-analysis. Eur Arch Otorhinolaryngol. 2017;274(6):2505-2512. doi: 10.1007/s00405-017-4483-1.
14. Shahul S, Chaya SK, Khader Mathamveed S, et al. A Retrospective study on the association of the systemic ınflammatory response ındex in predicting the severity of obstructive sleep apnea. Diagnostics (Basel). 2025;15(16):2091. doi: 10.3390/diagnostics15162091.
15. Young T, Skatrud J, Peppard PE. Risk factors for obstructive sleep apnea in adults. JAMA. 2004;291(16):2013-2016. doi: 10.1001/jama.291.16.2013.
16. Fattal D, Hester S, Wendt L. Body weight and obstructive sleep apnea: a mathematical relationship between body mass index and apnea-hypopnea index in veterans. J Clin Sleep Med. 2022;18(12):2723-2729. doi: 10.5664/jcsm.10190 .