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Clinical and Anthropometric Correlates of Polysomnography-Defined Severity in Obstructive Sleep Apnea Syndrome

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ABSTRACT

Objective: Obstructive sleep apnea syndrome (OSAS) is a common sleep-related breathing disorder frequently associated with cardiometabolic morbidity. In this retrospective study of 339 adults who underwent polysomnography (PSG) between January 2020 and December 2024, we investigated the relationship between routinely obtained clinical and anthropometric measures—body mass index (BMI), neck circumference (NC), waist circumference (WC), and the Epworth Sleepiness Scale (ESS)—and PSG-defined OSAS severity.

Materials and Methods: Adults (≥18 years) who underwent overnight PSG and had complete clinical and anthropometric data were included. OSAS severity was defined using the apnea–hypopnea index (AHI): no OSA (<5 events/h), mild (5–14.9), moderate (15–29.9), and severe (≥30). Associations between AHI and clinical or anthropometric measures were assessed using Spearman's correlation analysis.

Results: A total of 339 patients were included (mean age 46.1±12.0 years; 73.2% male; mean BMI 33.2±6.2 kg/m^2). The median ESS score was 4 (interquartile range [IQR]: 2–9), and the median AHI was 10.1 (IQR: 6.3–32.2). AHI showed statistically significant but weak correlations with age (r=0.207), weight (r=0.136), NC (r=0.273), and WC (r=0.184), whereas the association with ESS was stronger (r=0.649; all p<0.05). No significant correlations were observed with height or BMI. Patients with moderate-to-severe OSAS had higher NC, WC, ESS scores, symptom burden, and cardiometabolic comorbidities compared to those with normal-to-mild OSAS.

Conclusion: NC, WC, and ESS were associated with PSG-defined OSAS severity. However, correlations with age, weight, NC, and WC were weak in magnitude despite statistical significance, while BMI showed limited association. These routinely obtained measures may aid clinical assessment in sleep laboratory populations.

Keywords: Anthropometry, apnea–hypopnea index, Epworth Sleepiness Scale, neck circumference, Obstructive sleep apnea syndrome, polysomnography, waist circumference.

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INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a prevalent sleep-related respiratory disorder characterized by recurrent episodes of airflow limitation or cessation due to upper airway obstruction during sleep. The condition is widely recognized as a contributor to several major health problems, including cardiovascular disease, metabolic syndrome, and type 2 diabetes. Polysomnography (PSG), the gold standard for diagnosing OSAS and determining its severity, enables the measurement of brain activity, muscle tone, respiratory patterns, blood oxygen levels, and other physiological parameters during sleep.[1] Clinical and anthropometric characteristics are among the key factors influencing susceptibility to OSAS. Excess body weight, along with increased neck and waist circumference and elevated body mass index (BMI), has consistently been associated with a higher risk of upper airway obstruction during sleep. BMI is directly related to OSAS severity; as BMI increases, so does the propensity for upper airway collapse. Excess adipose tissue in the neck area contributes to airway obstruction, thereby increasing the likelihood of apnea events.[2]

When PSG findings are analyzed, individuals with elevated BMI, neck circumference (NC), and waist circumference (WC) generally exhibit higher apnea–hypopnea index (AHI) values. A positive correlation has been demonstrated between increases in BMI and NC and AHI, as these factors contribute to narrowing of the upper airway and increase the frequency of apnea–hypopnea episodes.[3] Data from the United States suggest that moderate-to-severe obstructive sleep apnea (defined as AHI ≥15 events per hour) affects approximately 13% of men and 6% of women aged 30–70 years.[4] The seriousness of comorbidities associated with OSAS—which is common yet often underdiagnosed—underscores the need for reliable screening programs, particularly for asymptomatic individuals.[5]

However, the strength of the associations between anthropometric variables and PSG-defined severity varies across populations, suggesting that further characterization of these relationships may provide additional clinical insight, particularly in referred sleep laboratory cohorts.

Many individuals at high risk for OSAS face barriers to accessing sleep clinics due to immobility, transportation limitations, or residence in remote areas. Excess body weight is a prominent clinical feature often used to identify at-risk individuals. However, beyond BMI or witnessed apneas, there is a need for practical screening tools that account for diverse body types and can rapidly identify those at risk. Although screening tools exist, understanding how simple bedside measurements relate to objective PSG-defined severity within clinical populations remains clinically relevant.[6]

KEY MESSAGES

- Neck circumference and waist circumference showed statistically significant but weak correlations with polysomnography-defined OSAS severity.
- The Epworth Sleepiness Scale demonstrated a stronger association with the apnea–hypopnea index compared with anthropometric measures, reflecting its relevance as a complementary clinical parameter.
- Patients with moderate-to-severe OSAS had a higher symptom burden and a greater prevalence of cardiometabolic comorbidities, highlighting the need for comprehensive clinical evaluation in sleep clinic populations.

Given that OSAS can lead to significant health complications and that PSG-derived AHI values are critical for determining disease severity, exploring the associations between routinely obtained clinical and anthropometric parameters and PSG outcomes may assist clinicians in contextualizing disease burden. Therefore, careful assessment of anthropometric measurements—such as BMI, NC, and WC—should be an integral component of OSAS evaluation.

This study sought to evaluate how various clinical and anthropometric characteristics relate to polysomnographic outcomes in patients with a confirmed diagnosis of OSAS. Rather than proposing a predictive model, the study focuses on describing the extent to which commonly collected clinical measures correlate with PSG-defined severity. By providing population-specific data, this work may support more individualized diagnostic considerations within sleep laboratory settings. We hypothesized that significant correlations exist between clinical variables, such as age, sex, and subjective sleep quality, and anthropometric measures, such as BMI, NC, and WC, in relation to PSG parameters—particularly the AHI.

MATERIALS AND METHODS

Study Protocol and Design

This study was designed as a retrospective descriptive investigation. Patients aged ≥18 years who presented to the pulmonology outpatient clinic of our hospital between January 2020 and December 2024 with complaints of sleep disturbances, were indicated for PSG, and subsequently underwent overnight PSG in the sleep laboratory were included.

Ethics Approval/Informed Consent

This research was conducted in accordance with national and institutional guidelines governing human research and adhered

to the ethical principles of the Declaration of Helsinki. Approval for the study was granted by Recep Tayyip Erdogan University Non-Interventional Clinical Research Ethics Committee (Approval Number: E-40465587-050.01.04-1367, 2025/50, Date: 12.02.2025). The requirement for informed consent was waived due to the retrospective design and the use of anonymized patient data.

Patients and Data Collection

All clinical and anthropometric assessments were performed during the routine outpatient sleep clinic evaluation prior to polysomnography. Polysomnography was conducted on the same night or within one week of the outpatient assessment.

Neck circumference was measured with the participant standing upright and the head positioned in the Frankfort horizontal plane, with the measuring tape placed at the level of the cricothyroid cartilage. Waist circumference was measured at the midpoint between the lower rib margin and the iliac crest during quiet expiration. Body weight and height were obtained using calibrated clinical devices, and body mass index was calculated as weight (kg) divided by height squared (m^2).

Epworth Sleepiness Scale (ESS) scores were retrieved retrospectively from routine sleep clinic evaluation records. The ESS consists of eight questions, each rated on a scale from 0 to 3, yielding a total score ranging from 0 to 24, with higher scores indicating greater self-reported daytime sleepiness.

All respiratory events recorded during overnight polysomnography were evaluated in accordance with the 2012 guidelines of the American Academy of Sleep Medicine (AASM). Apnea was defined as a reduction in airflow of at least 90% lasting for a minimum of 10 seconds, whereas hypopnea was defined as a decrease in airflow of at least 30% lasting at least 10 seconds and associated with either a ≥3% oxygen desaturation or an electroencephalographic arousal.

The apnea–hypopnea index was defined as the average number of apneic and hypopneic respiratory events per hour of recorded sleep. Based on AHI values, OSAS severity was categorized into four groups:

- ***No OSA:*** AHI <5 events/h
- ***Mild OSA:*** AHI 5–14.9 events/h
- ***Moderate OSA:*** AHI 15–29.9 events/h
- ***Severe OSA:*** AHI ≥30 events/h

Inclusion Criteria: Participants were eligible for inclusion if they were aged 18 years or older; had been referred for overnight polysomnography due to suspected sleep-disordered breathing; had complete PSG recordings available; and had comprehensive clinical and anthropometric data documented, including age, sex, height, body weight, neck circumference, waist circumference, and Epworth Sleepiness Scale scores.

Exclusion Criteria: Patients were excluded if they had a prior history of upper airway surgery for obstructive sleep apnea, documented neuromuscular disease, craniofacial abnormalities, central sleep apnea, chronic respiratory failure, pregnancy, or incomplete clinical, anthropometric, or polysomnographic records. Only individuals who met all eligibility criteria were included in the final analysis.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics software (version 21; SPSS Inc., Chicago, IL, USA). The distribution of continuous variables was assessed using the Kolmogorov–Smirnov normality test. Normally distributed data were expressed as mean±standard deviation, whereas non-normally distributed variables were reported as median (interquartile range). Categorical variables were summarized as counts and percentages.

For group comparisons, the Student's t-test was applied to normally distributed variables, while the Mann–Whitney U test was used for non-normally distributed variables. Categorical data were analyzed using Pearson's chi-square test or Fisher's exact test, as appropriate. Fisher's exact test was applied when expected cell frequencies were less than five.

Because the apnea–hypopnea index did not follow a normal distribution and the Epworth Sleepiness Scale is an ordinal variable, associations between AHI and clinical or anthropometric parameters were examined using Spearman's rank correlation analysis. Statistical significance was defined as $p<0.05$.

A post hoc power analysis evaluating differences in neck circumference between the moderate-to-severe and normal-to-mild OSA groups demonstrated sufficient statistical power (Cohen's d=0.50; power=0.99).

Given the descriptive and retrospective nature of the study and the strong collinearity among anthropometric variables, multivariable regression modeling was not performed. Accordingly, the analysis was restricted to correlation-based evaluation of associations between clinical parameters and AHI.

The primary endpoints of the study were the apnea–hypopnea index and the Epworth Sleepiness Scale. Secondary outcomes included anthropometric parameters (BMI, neck circumference, and waist circumference), symptom profiles, and cardiometabolic comorbidities.

Confidence intervals for Spearman correlation coefficients were not routinely generated by the statistical software used in this retrospective analysis. Future prospective studies should incorporate confidence interval estimation to improve precision and clinical interpretability.

RESULTS

A total of 339 patients were included in the analysis. The mean age was 46.10±12.00 years, and 73.20% were male. Mean anthropometric values were as follows: height 171±9 cm, weight 97±19 kg, BMI 33.20±6.20 kg/m^2, NC 41±4 cm, and WC 110±17 cm. The median ESS score was 4 (interquartile range [IQR]: 2–9), and the median AHI was 10.10 (IQR: 6.30–32.20). Snoring (52.20%), witnessed apneas (41.30%), morning fatigue (33.90%), nocturia (30.70%), and morning dry mouth (33.60%) were the most frequently reported symptoms. Based on AHI values, 10.1% of patients had no obstructive sleep apnea (OSA), 51.30% had mild OSA, 6.50% had moderate OSA, and 32.20% had severe OSA. Hypertension (36.90%) and type 2 diabetes (17.40%) were the most common comorbidities (Table 1).

Spearman's correlation analysis demonstrated significant positive associations between AHI and age (r=0.207, p<0.001), weight (r=0.136, p=0.012), NC (r=0.273, p<0.001), WC (r=0.184, p=0.001), and ESS (r=0.649, p<0.001).

The correlations with age, weight, NC, and WC were weak, whereas the association between ESS and AHI was strong.

No statistically significant correlations were observed between AHI and height (r=0.094, p=0.082) or BMI (r=0.086, p=0.114) (Table 2).

A scatter plot illustrating the association between ESS and AHI is presented in Figure 1.

When grouped by disease severity, patients with moderate-to-severe OSA were older (49±10 vs. 45±13 years, p=0.003; Cohen's d=0.35, 95% confidence interval [CI]: 0.12–0.58) and predominantly male (p<0.001). NC and WC were also significantly higher in this group (NC: 42±4 vs. 40±4 cm; p<0.001; Cohen's d=0.50, 95% CI: 0.27–0.73; WC: 113±17 vs. 108±17 cm, p<0.001; Cohen's d=0.29, 95% CI: 0.07–0.51). ESS scores were substantially higher in moderate-to-severe OSA (median 9 [IQR: 7–12]) compared with normal-to-mild cases (median 2 [IQR: 0–4], p<0.001).

Symptom frequency increased markedly with disease severity: snoring (96% vs. 25%), witnessed apneas (95% vs. 7%), morning fatigue (79% vs. 4%), nocturia (75% vs. 3%), and dry mouth (77% vs. 6%) (all p<0.001).

Table 1. Demographic and clinical characteristics of the study population (n=339)

Parameter	Value
Age (years)	46.1±12.0
Sex, n (%)	
Female	91 (26.8)
Male	248 (73.2)
Height (cm)	171±9
Weight (kg)	97±19
BMI (kg/m^2)	33.2±6.2
NC (cm)	41±4
WC (cm)	110±17
ESS (points)	4 (IQR: 2–9)
AHI (events/h)	10.1 (IQR: 6.3–32.2)
OSA-related symptoms, n (%)	
Snoring	177 (52.2)
Witnessed apnea	140 (41.3)
Morning fatigue	115 (33.9)
Nocturia	104 (30.7)
Dry mouth	114 (33.6)
OSA severity classification, n (%)	
No OSA (AHI <5)	34 (10.0)
Mild OSA (AHI: 5–15)	174 (51.3)
Moderate OSA (AHI: 16–30)	22 (6.5)
Severe OSA (AHI ≥30)	109 (32.2)
Comorbidities, n (%)	
Hypertension	125 (36.9)
Type 2 diabetes mellitus	59 (17.4)
Coronary artery disease	24 (7.1)
Chronic obstructive pulmonary disease	8 (2.4)
Asthma	18 (5.3)
Allergy	79 (23.3)
Arrhythmia	22 (6.5)

Table 1 (Cont). Demographic and clinical characteristics of the study population (n=339)

Parameter	Value
Hyperlipidemia	42 (12.4)
Gastritis	45 (13.3)
Restless legs syndrome	24 (7.1)

Data are presented as mean±standard deviation, median (interquartile range), or number (percentage), as appropriate. Primary outcomes were apnea–hypopnea index (AHI) and the Epworth Sleepiness Scale (ESS). Secondary outcomes included anthropometric parameters, symptom profiles, and comorbidities.

Table 2. Correlation between apnea–hypopnea index (AHI) and clinical/anthropometric variables (Spearman's rank correlation analysis)

Parameter	Correlation Coefficient (r)	p
Age (years)	0.207	<0.001*
Height (cm)	0.094	0.082
Weight (kg)	0.136	0.012*
BMI (kg/m^2)	0.086	0.114
NC (cm)	0.273	<0.001*
WC (cm)	0.184	0.001*
ESS (points)	0.649	<0.001*

Apnea–hypopnea index (AHI) was defined as the primary outcome variable. All other variables were treated as secondary explanatory variables. *p<0.05 indicates statistical significance.

Similarly, cardiometabolic comorbidities were more prevalent in moderate-to-severe OSA, including hypertension (53% vs. 27%), type 2 diabetes (25% vs. 13%), coronary artery disease (11% vs. 5%), arrhythmia (15% vs. 1%), hyperlipidemia (20% vs. 8%), and restless legs syndrome (14% vs. 3%) (all p<0.05). These findings indicate a progressive increase in both symptom burden and comorbidity frequency with advancing OSA severity (Table 3).

DISCUSSION

The findings of this study highlight four key points:

a) Significant associations between clinical and anthropometric parameters—namely NC, WC, and the ESS—and the AHI.

b) A strong positive correlation between ESS and AHI, suggesting that subjective daytime sleepiness may serve as a useful marker of disease severity.

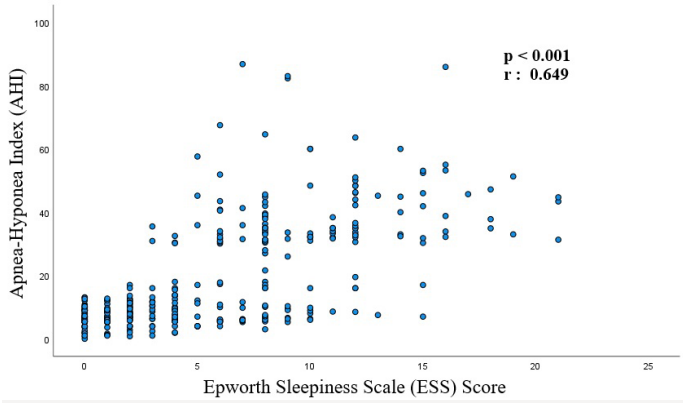

Figure 1. Scatter plot illustrating the monotonic association between the Epworth Sleepiness Scale (ESS) score and the apnea–hypopnea index (AHI). The plot provides a descriptive visualization of the relationship based on Spearman's correlation analysis and does not represent a predictive regression model.

c) A notable increase in age, central obesity indicators, and symptom burden among individuals with moderate-to-severe OSAS.

d) A significant rise in the prevalence of cardiometabolic comorbidities with increasing OSA severity.

This study provides a comprehensive evaluation of the relationships between clinical and anthropometric parameters and PSG findings in patients diagnosed with OSAS. Consistent with previous research, the results demonstrate meaningful correlations between AHI and specific anthropometric indicators, particularly NC and WC. Rather than implying novelty, these findings reaffirm the clinical relevance of routinely obtained anthropometric markers in OSAS assessment, especially in settings where access to PSG may be limited.

The predominance of male patients in our cohort (73.2%) aligns with existing epidemiological data indicating a higher prevalence of OSAS among men. The mean BMI of 33.2 kg/m^2 coupled with mean WC and NC measurements of 110 cm and 41 cm, respectively, reflects the strong association between OSAS and central obesity.[7] The distribution of OSAS severity in our cohort—with 51.3% classified as mild and 32.2% as severe— likely reflects referral bias and the selective nature of patients undergoing PSG, underscoring the heterogeneity of OSAS in clinical practice. Symptomatically, the most frequently reported complaints were snoring (52.2%), morning fatigue (33.9%), and nocturia (30.7%), reflecting both upper airway obstruction and systemic manifestations of the disorder. Hypertension (36.9%) and type 2 diabetes mellitus (17.4%) were the most prevalent comorbidities, consistent with literature linking OSAS to increased cardiometabolic

Table 3. Comparison of patients with normal-to-mild and moderate-to-severe obstructive sleep apnea

Variable	Normal-to-Mild OSA (AHI ≤15) (n=208)	Moderate-to-Severe OSA (AHI ≥16) (n=131)	p
Age (years)	45±13	49±10	0.003*
Sex (F/M)	71/137	20/111	<0.001*
Height (cm)	170±9	172±8	0.090
Weight (kg)	96±19	100±19	0.076
BMI (kg/m^2)	32.9±6.1	33.7±6.5	0.256
NC (cm)	40±4	42±4	<0.001*
WC (cm)	108±17	113±17	0.010*
ESS (points)	2 (0–4)	9 (7–12)	<0.001*
Snoring (%)	51 (25)	126 (96)	<0.001*
Witnessed apnea (%)	15 (7)	125 (95)	<0.001*
Morning fatigue (%)	8 (4)	107 (79)	<0.001*
Nocturia (%)	6 (3)	98 (75)	<0.001*
Dry mouth (%)	13 (6)	101 (77)	<0.001*
Hypertension (%)	56 (27)	69 (53)	<0.001*
Type 2 diabetes mellitus (%)	26 (13)	33 (25)	0.003*
Coronary artery disease (%)	10 (5)	14 (11)	0.040*
Chronic obstructive pulmonary disease (%)	4 (2)	4 (3)	0.504†
Asthma (%)	12 (6)	6 (5)	0.634
Arrhythmia (%)	3 (1)	19 (15)	<0.001*
Allergy (%)	54 (26)	25 (19)	0.145
Hyperlipidemia (%)	16 (8)	26 (20)	<0.001*
Restless legs syndrome (%)	6 (3)	18 (14)	<0.001*

Data are presented as mean±standard deviation, median (interquartile range), or number (percentage), as appropriate. Primary outcomes were apnea–hypopnea index (AHI) and the Epworth Sleepiness Scale (ESS). Secondary outcomes included anthropometric parameters, symptom profiles, and comorbidities. Categorical variables were compared using Pearson's chi-square test or Fisher's exact test, as appropriate. Fisher's exact test was applied when expected cell counts were <5. *Statistically significant difference between groups (p<0.05). † Fisher's exact test.

risk through mechanisms such as systemic inflammation, oxidative stress, and insulin resistance.[8,9] These observations further support the concept of OSAS as a multisystem disorder rather than an isolated respiratory condition.

Although the observed associations are largely consistent with previous literature, the present findings provide clinically relevant insight derived from a real-world tertiary sleep clinic population characterized by high rates of obesity, cardiometabolic comorbidity, and symptomatic sleep-disordered breathing. Our cohort represents a referral-based clinical population characterized with a high prevalence of central obesity, cardiometabolic comorbidity, and a broad spectrum of OSAS severity, reflecting contemporary clinical practice in a regional healthcare setting. In contrast to population-based screening cohorts, our study captures patients with symptomatic sleep-disordered breathing who were referred for diagnostic polysomnography, thereby

offering practical information applicable to daily clinical decision-making. Furthermore, the regional referral patterns and healthcare access characteristics of our center contribute to a distinct patient profile, with a predominance of male patients, advanced obesity, and high cardiometabolic risk. These features underscore the clinical relevance of our findings for sleep clinics serving similar populations and healthcare systems.

Apnea–hypopnea index showed statistically significant positive associations with age, body weight, neck circumference, waist circumference, and ESS scores. However, correlations with age, weight, NC, and WC were weak in magnitude, indicating small effect sizes despite statistical significance. Among these variables, ESS exhibited the strongest association with AHI ($r=0.649$, $p<0.001$), suggesting a moderate-to-strong monotonic relationship between subjective daytime sleepiness and respiratory disturbance severity.[10] However, given the ordinal nature of ESS and the skewed distribution of AHI, this association should be interpreted as reflecting a monotonic relationship rather than serving as a predictive metric. The associations between AHI and both NC and WC—indicators of central adiposity—further support the role of fat distribution, particularly in the upper body, in the pathophysiology of OSAS.[7] Conversely, the absence of significant associations between AHI and either height or BMI underscores the limited predictive value of BMI alone. This observation is consistent with evidence that BMI does not adequately capture regional fat deposition, particularly in the upper body, which may explain discrepancies across studies. In line with these findings, Yetkin et al.[11] recently demonstrated that alternative anthropometric indices reflecting body fat distribution, such as the triponderal mass index, are significantly associated with obstructive sleep apnea severity and may provide complementary information beyond conventional BMI measurements.

The robust positive correlation between ESS and AHI ($r=0.649$, $p<0.001$) indicates that subjective daytime sleepiness generally parallels objective respiratory disturbance. Although this finding supports the clinical relevance of ESS, it should not be interpreted as a predictive model, as the present study did not include multivariable or prognostic analyses. Nevertheless, the strength of this association aligns with previous evidence suggesting that ESS may serve as a practical clinical indicator of OSAS severity.[10] Such a relationship may be useful for prioritizing patients for diagnostic evaluation; however, validation in larger and more heterogeneous populations is required, and the influence of coexisting comorbidities should be evaluated in adjusted models.

In this study, significant differences in both demographic and anthropometric characteristics were observed across OSAS severity levels. The older age observed in the moderate-to-severe OSAS group reflects the cumulative risk associated with aging and the progressive nature of the disorder. The predominance of male patients in this group—along with higher AHI values—is likely attributable to narrower upper airway anatomy and differences in fat distribution patterns between sexes.[12] The notably higher neck and waist circumference measurements among individuals with more severe OSAS further reinforce the role of central obesity as a major structural risk factor predisposing to upper airway collapse. Regarding symptomatology, the near-universal prevalence of snoring, witnessed apneas, morning fatigue, nocturia, and dry mouth in the moderate-to-severe OSAS group demonstrates a close relationship between disease severity and symptom burden. The significantly elevated ESS scores in this group further confirm the clinical alignment between subjective sleepiness and objective respiratory disturbance.

With respect to accompanying medical conditions, cardiometabolic disorders—such as hypertension, type 2 diabetes, coronary artery disease, arrhythmias, and lipid abnormalities—were observed far more frequently among individuals with moderate-to-severe OSAS compared with those with milder disease. This pattern suggests that increasing AHI levels are associated with multisystem involvement, potentially mediated by mechanisms such as systemic inflammation, endothelial dysfunction, and autonomic imbalance.[13] Although these findings are consistent with previous reports, causal inferences cannot be drawn due to the cross-sectional design. Furthermore, considering the heterogeneous nature of OSAS phenotypes, risk stratification based on clinical and anthropometric indicators should be complemented by phenotype-specific therapeutic strategies. Recent literature has highlighted that, particularly in positional OSAS, individualized treatment approaches may enhance therapeutic effectiveness.[14]

Taken together, these findings further support the concept that OSAS is a complex, multisystemic disorder extending beyond isolated nocturnal respiratory disturbances. Rather than extending existing knowledge, the present study consolidates current evidence within a real-world clinical context. The integration of anthropometric profiling, symptom assessment, and comorbidity screening into routine clinical evaluation remains essential. Early identification of nonmodifiable risk factors, such as central obesity, advanced age, and male sex, is critical for developing targeted diagnostic strategies and tailored treatment plans. Given the progressive nature of OSAS and its strong links to cardiometabolic morbidity, optimal patient care requires a multidisciplinary approach and individualized follow-up.

This study has several limitations. First, its retrospective design and reliance on data from a single center may limit

the applicability of the findings to broader or more diverse populations. As a result, the observed associations may primarily reflect the characteristics of patients referred to a tertiary sleep clinic rather than those of community-based or asymptomatic populations. Second, because the study population consisted of patients referred for suspected sleep disorders, selection bias is inevitable; therefore, the results may not be fully applicable to asymptomatic individuals or community-based cohorts. This referral-based sampling may have led to an overrepresentation of individuals with more advanced disease and greater symptom burden. Third, although anthropometric measurements such as neck and waist circumferences were extracted from standard medical records, the possibility of interobserver variability in these assessments cannot be entirely excluded. Such variability may have introduced random error, potentially attenuating the strength of the observed associations. Fourth, due to the cross-sectional nature of the study, causal relationships between the investigated parameters and OSAS severity cannot be inferred. Accordingly, the findings should be interpreted as associative rather than indicative of causal pathways. Finally, the study did not include assessments of craniofacial structure, upper airway imaging, or relevant biochemical markers, all of which could have contributed to a more comprehensive understanding of the pathophysiological mechanisms linking anthropometric features to OSAS. The absence of these variables limits our ability to explore mechanistic pathways underlying the observed clinical associations. Future prospective, multicenter studies addressing these limitations are needed to enhance the validity and generalizability of our findings.

CONCLUSION

This study demonstrates that clinical and anthropometric measures—particularly neck circumference, waist circumference, and the Epworth Sleepiness Scale—are associated with polysomnography-defined OSAS severity. These findings support the relevance of simple, routinely collected clinical parameters in the assessment of patients undergoing evaluation for suspected OSAS. However, as the analyses were correlational, these measures should not be interpreted as independent predictors of disease severity without further multivariable or prognostic modeling.

Obstructive sleep apnea syndrome remains a multifaceted condition with systemic consequences that extend beyond disordered breathing during sleep. Even in patients with mild symptoms or low subjective sleepiness, incorporating anthropometric evaluation and comorbidity screening may help clinicians better characterize overall disease burden. Early identification of key risk factors—such as central obesity, older age, and male sex—may assist in guiding diagnostic prioritization and clinical decision-making.

Given the progressive and cardiometabolic nature of OSAS, patient care should rely on comprehensive assessment and multidisciplinary management. Future prospective studies incorporating adjusted analyses and predictive modeling are needed to clarify the independent contribution of these clinical parameters and to determine their potential role in diagnostic pathways.

Ethics Committee Approval: Ethics committee approval was obtained from Recep Tayyip Erdogan University Non-Interventional Clinical Research Ethics Committee (Approval Number: E-40465587-050.01.04-1367, 2025/50, Date: 12.02.2025).

Informed Consent: Written informed consent was obtained from the patient or the next of kin for publication.

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