Original article

Association between Obesity Severity and Nocturnal Hypoxemia in Obstructive Sleep Apnea

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Abstract

Aim: To evaluate the association between obesity severity and nocturnal hypoxemia in patients with obstructive sleep apnea (OSA), and to compare the ability of body mass index (BMI) and the apnea–hypopnea index (AHI) to identify nocturnal hypoxemia.

Methods: This retrospective cross-sectional study included 70 adult patients diagnosed with OSA who underwent full-night attended polysomnography. Anthropometric data, including BMI and obesity class, and polysomnographic parameters were collected. Nocturnal hypoxemia was defined as peripheral oxygen saturation (SpO_2) < 90%. Associations between nutritional status, nocturnal oxygenation, snoring parameters, and AHI were analysed. Receiver operating characteristic (ROC) analysis was used to assess the discriminatory ability of BMI and AHI for nocturnal hypoxemia.

Results: The median age was 58 years (IQR 47-64), and 76% of patients were male. Obesity was present in 74.3% of patients, including 42.3% with class I, 32.7% with class II, and 25.0% with class III obesity. Mean nocturnal oxygen saturation and oxygen saturation nadir declined progressively with increasing obesity class ($p = 0.008$ and $p = 0.02$, respectively), whereas AHI showed only a non-significant increasing trend ($p = 0.09$). Nocturnal hypoxemia was observed predominantly in obese patients and was most frequent in class III obesity (47%, $p = 0.04$). Both BMI (AUC 0.742) and AHI (AUC 0.805) were significantly associated with nocturnal hypoxemia, with no significant difference between ROC curves ($p = 0.41$).

Conclusion: Obesity severity is strongly associated with nocturnal hypoxemia in OSA, independent of AHI. BMI provides clinically meaningful information comparable to AHI in identifying patients at risk of nocturnal hypoxemia, supporting a complementary approach to OSA severity assessment.

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Introduction

Obstructive sleep apnea (OSA) is a prevalent sleep-related breathing disorder characterized by recurrent partial or complete upper airway collapse during sleep, resulting in intermittent hypoxemia and sleep fragmentation (1). According to population-based modelling by Benjafield et al., OSA affects an estimated 936 million adults globally, with 425 million suffering from moderate-to-severe disease (2). The severity of OSA is traditionally quantified using the apnea–hypopnea index (AHI), which reflects the frequency of respiratory events per hour of sleep (3). Although widely used in clinical practice, AHI does not fully capture the physiological burden of OSA, particularly the extent and depth of nocturnal oxygen desaturation.

Nocturnal hypoxemia has emerged as a critical pathophysiological mechanism linking OSA to adverse cardiovascular and metabolic outcomes (4). Growing evidence suggests that parameters such as mean nocturnal oxygen saturation and oxygen nadir may be more closely associated with hypertension, arrhythmias, stroke, and cardiovascular mortality than AHI alone (4-6). Consequently, there is increasing interest in identifying clinical factors that better predict the severity of nocturnal hypoxemia in patients with OSA.

Obesity is the most significant modifiable risk factor for OSA and plays a central role in the pathophysiology of upper airway obstruction and ventilatory impairment (7). Excess adipose tissue contributes to reduced upper airway caliber, impaired neuromuscular control, decreased lung volumes, and altered respiratory mechanics. In patients with severe obesity, these mechanisms may exacerbate nocturnal hypoxemia disproportionately, even in the absence of a corresponding increase in the frequency of apneic or hypopneic events (7).

Despite the well-established association between obesity and OSA severity, the relative contribution of obesity severity to nocturnal hypoxemia independent of conventional AHI-based classification remains insufficiently explored. In particular, it is unclear whether increasing obesity class is more strongly associated with nocturnal oxygen impairment than the conventional AHI-based classification of OSA severity.

Therefore, the aim of this study was to evaluate the association between obesity severity and nocturnal hypoxemia in patients with OSA, and to assess the extent to which obesity-related measures contribute to nocturnal oxygen impairment beyond conventional indices of disease severity.

Materials and Methods

Study design

This study was designed as a retrospective cross-sectional analysis of patients evaluated for OSA at the Department of Neurology, University Hospital Centre Osijek. Clinical, anthropometric, questionnaire-based, and polysomnographic data were extracted from electronic medical records over a predefined study period.

Study Population

Adult patients referred to the sleep disorders outpatient clinic who underwent full-night attended polysomnography and were diagnosed with OSA were eligible for inclusion. Data were collected over an eight-month period, from May 2021 to January 2022. A total of 70 patients were included in the final analysis. Patients with incomplete polysomnographic recordings or missing key anthropometric measurements were excluded. All consecutive adult patients who met the inclusion criteria and underwent full-night attended polysomnography during the predefined study period (May 2021 to January 2022) were included in the analysis. No a priori sample size calculation was performed due to the retrospective design of the study.

Anthropometric and Clinical Data

Demographic and clinical data obtained from hospital electronic medical records included age, sex, height, body weight, smoking status, alcohol consumption, employment status, and comorbidities, including arterial hypertension and diabetes mellitus. Body mass index (BMI) was calculated as body weight in kilograms divided by height in meters squared (kg/m^2).

Patients were categorized according to World Health Organization (WHO) BMI classification into normal weight, overweight, and obesity. Obesity was further classified into class I (30.0-34.9 kg/m^2), class II (35.0-39.9 kg/m^2), and class III (≥40.0 kg/m^2).

Assessment of Daytime Sleepiness and OSA Risk

Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS), a validated self-administered questionnaire consisting of eight items scored from 0 to 3, yielding a total score ranging from 0 to 24. The STOP-BANG questionnaire was used to assess the risk of OSA. This instrument comprises eight dichotomous (yes/no) items related to snoring, tiredness, observed apnea, blood pressure, BMI, age, neck circumference, and sex. Patients were categorized as having low, intermediate, or high risk according to established scoring criteria.

Polysomnographic Assessment

All patients underwent full-night attended polysomnography using standardized recording techniques. Recordings included electroencephalography, electrooculography, electromyography, electrocardiography, airflow measurement, respiratory effort monitoring, pulse oximetry, body position, and snoring detection. Respiratory events were scored according to the American Academy of Sleep Medicine criteria (version 2.4) (1). Oxygen saturation parameters included mean nocturnal peripheral oxygen saturation (SpO_2), lowest oxygen saturation during sleep (SpO_2 nadir), and oxygen desaturation indices. Nocturnal hypoxemia was defined as SpO_2 <90% for any duration during sleep. This threshold was chosen because oxygen saturation values below 90% are widely considered clinically relevant and have been commonly used in previous studies and clinical guidelines to define sleep-related hypoxemia (8). OSA severity was classified based on the AHI as mild (5-14.9 events/h), moderate (15-29.9 events/h), or severe (≥30 events/h).

Statistical Analysis

Categorical variables were expressed as absolute numbers and percentages. Continuous variables were tested for normality using the Shapiro-Wilk test and are presented as medians with interquartile ranges due to non-normal distribution. Between-group comparisons were performed using the chi-square test or Fisher's exact test for categorical variables, and the Mann-Whitney U test or Kruskal-Wallis test with post hoc Conover analysis for continuous variables, as appropriate. All statistical tests were two-tailed, and a p-value <0.05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics, version 23.0.

Results

Baseline characteristics

A total of 70 patients with OSA were included in the analysis. The median age was 58 years (interquartile range [IQR] 47-64), and the majority of patients were male (76%). The median BMI was 32.55 kg/m^2 (IQR 29.55-38.80). Overall, 74.3% of patients were classified as obese, 20.0% were overweight, and 5.7% had normal body weight. Among obese patients, 42.3% had class I obesity, 32.7% class II obesity, and 25.0% class III obesity (Table 1).

Table 1. Baseline demographic and anthropometric characteristics of the study population

Variable	Median (IQR) / n (%)	Range
Age (years)	58 (47-64)	35-80
Height (cm)	176 (170-183)	159-199
Body weight (kg)	102 (89-121)	66-160
BMI (kg/m^2)	32.55 (29.55-38.80)	21.5-55.1
BMI category		
Normal weight	4 (5.7)	—
Overweight	14 (20.0)	—
Obese (total)	52 (74.3)	—
Class I obesity	22 (42.3)	—
Class II obesity	17 (32.7)	—
Class III obesity	13 (25.0)	—

Abbreviations: IQR - interquartile range; body mass index - BMI

Polysomnographic characteristics according to nutritional status

Significant differences in nocturnal oxygenation and snoring-related parameters were observed across nutritional status categories. Obese patients exhibited significantly lower mean nocturnal oxygen saturation and lower oxygen saturation nadir compared with overweight patients (p = 0.007 and p = 0.01, respectively). In addition, obese patients demonstrated a higher number of snoring events, longer snoring duration, and a greater proportion of total sleep time spent snoring compared with normal-weight and overweight individuals (all p < 0.01). The AHI increased significantly with worsening nutritional status (p = 0.02) (Table 2).

Table 2. Polysomnographic characteristics according to nutritional status

Polysomnographic parameter	Normal weight	Overweight	Obese	p*
Mean oxygen saturation (%)	93 (89.25-93.75)	93 (92-94)	91 (89-92.75)	0.007ǂ
Oxygen saturation nadir (%)	80.5 (75.75-88.25)	83.5 (77.75-87.25)	76 (65.25-80.75)	0.01ǂ
Snoring events (n)	74 (27.25-144)	168.5 (59.5-210)	269.5 (160.5-395.75)	0.001ǂ
Snoring duration (min)	22.35 (2.98-41.2)	29.65 (16.95-52.4)	71.55 (40.53-100.15)	0.002ǂ
Snoring time (% of sleep)	1.68 (0.39-9.9)	9.15 (5.35-15.55)	19.25 (8.25-30.68)	0.009ǂ
AHI (events/h)	12.65 (8.93-50.73)	32.35 (21.2-40.03)	43.4 (30.43-65.95)	0.02ǂ

Abbreviations: apnea–hypopnea index - AHI
Data are presented as median (interquartile range).
* Kruskal-Wallis test (post hoc Conover).
ǂ At p < 0.05, obese patients had significantly lower values compared with overweight patients.
ǂ At p < 0.05, obese patients had significantly different values compared with all other nutritional status groups.

Polysomnographic characteristics according to obesity class

When obese patients were stratified according to obesity class, a progressive impairment in nocturnal oxygenation was observed. Patients with class III obesity had significantly lower mean nocturnal oxygen saturation and lower oxygen saturation nadir compared with those with class I obesity (p = 0.008 and p = 0.02, respectively). Although snoring burden and AHI tended to increase with higher obesity classes, these differences did not reach statistical significance (Table 3).

Table 3. Nocturnal oxygenation, snoring, and AHI according to obesity class

Parameter	Class I	Class II	Class III	p
Mean SpO_2 (%)	92 (89,75 - 93,25)	91 (89 - 92)	89 (84 - 91)	**0.008**
SpO_2 nadir (%)	79 (73,25 - 83,75)	76 (64 - 79,5)	66 (54 - 78)	**0.02**
Snoring time (% TST)	16,95 (6,58 - 31,3)	17,2 (4,05 - 28,1)	24,9 (16,85 - 31,85)	0,27
AHI (events/h)	35,1 (23,78 - 62,58)	50,9 (33,35 - 60,1)	64,9 (32,7 - 80,75)	0,09

Abbreviations: apnea–hypopnea index - AHI; SpO_2 - peripheral oxygen saturation. Data are presented as median (interquartile range).

Nocturnal hypoxemia and obesity

The prevalence of nocturnal hypoxemia differed significantly according to nutritional status. Hypoxemia was observed predominantly in obese patients (p = 0.02). Within the obese subgroup, nocturnal hypoxemia was most frequent among patients with class III obesity (47%), compared with class I (29%) and class II obesity (24%) (p = 0.04) (Table 4)..

Table 4. Nutritional status and obesity class according to presence of nocturnal hypoxemia

Nutritional status	No hypoxemia	Hypoxemia	Total	P*
Overall nutritional status				0.02
Normal weight	3 (6)	1 (6)	4 (6)	
Overweight	14 (27)	0 (0)	14 (20)	
Obese	35 (67)	17 (94)	52 (74)	
Obesity class (n = 52)				0.04
Class I obesity	17 (49)	5 (29)	22 (42)	
Class II obesity	13 (37)	4 (24)	17 (33)	
Class III obesity	5 (14)	8 (47)	13 (25)	

Data are presented as n (%).
* Fisher's exact test.

Comparative performance of BMI and AHI in identifying nocturnal hypoxemia

ROC analysis demonstrated that both BMI and AHI were significant diagnostic indicators of nocturnal hypoxemia, with higher discriminatory performance observed for AHI (AUC = 0.805) compared with BMI (AUC = 0.742). However, the difference between the two ROC curves was not statistically significant (P = 0.41) (Table 5, Figure 1).

Table 5. Receiver operating characteristic (ROC) analysis of BMI and AHI index for nocturnal hypoxemia

	AUC	95% CI	Sensitivity	Specificity	*cut off*	Youden index	*P value*
BMI	0.742	0.623 – 0.839	83.3	59.6	> 32.3	0.430	**<0.001**
AHI	0.805	0.693 – 0.890	83.3	69.2	>42.5	0.526	**<0.001**
BMI vs. AHI	Difference between areas = 0.063			95% CI: -0.088 to 0.214			0.41

Abbreviations: AUC - Area under the curve; CI - confidence interval; apnea–hypopnea index - AHI; body mass index – BMI; SpO_2 - peripheral oxygen saturation. Bold denotes statistical significant

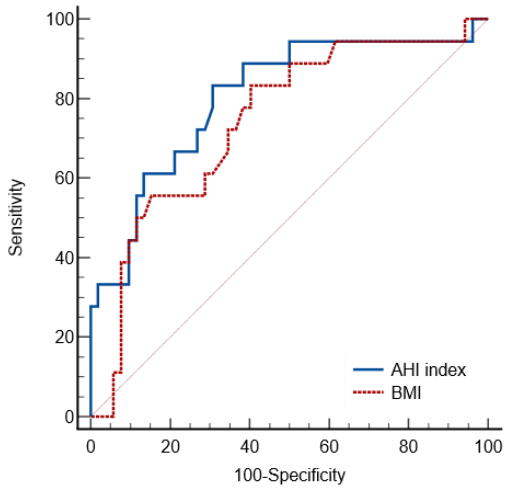

Figure 1. ROC curves of BMI and AHI for nocturnal hypoxemia.

Discussion

In this study, obesity was strongly associated with the severity of nocturnal hypoxemia in patients with OSA. Although worsening nutritional status was accompanied by increasing AHI values, measures of nocturnal oxygenation showed a more pronounced and consistent deterioration with increasing obesity severity. These findings support the concept that obesity contributes to the hypoxemic burden of OSA beyond the frequency of respiratory events alone.

We observed a stepwise decline in mean nocturnal oxygen saturation and oxygen saturation nadir across increasing obesity classes. Notably, patients with class III obesity exhibited the most severe nocturnal oxygen impairment, despite only a trend toward higher AHI values. These findings are consistent with emerging evidence indicating that adiposity-related factors play a central role in determining hypoxemic burden during sleep. Recent data suggest that measures of visceral adiposity may be particularly relevant, even in individuals without overt obesity, highlighting the importance of fat distribution rather than body mass alone in the development of nocturnal hypoxemia (9). Similarly, studies examining pulmonary function in patients with OSA have demonstrated that impaired lung volumes and altered respiratory mechanics are closely associated with distinct nocturnal hypoxemia patterns, supporting the concept that obesity-related reductions in functional residual capacity and ventilation-perfusion mismatch contribute to deeper and more sustained oxygen desaturation during sleep (10).

Importantly, nocturnal hypoxemia in obese patients has been shown to have clinically meaningful consequences. Kendzerska and colleagues demonstrated that the interaction between obesity and nocturnal hypoxemia is associated with an increased risk of adverse cardiovascular outcomes, independent of traditional OSA severity measures (11). Our findings extend these observations by showing that increasing obesity severity is associated with progressively worse nocturnal oxygenation, even when differences in AHI are less pronounced.

In addition to impaired oxygenation, obese patients demonstrated a significantly greater snoring burden, reflected by higher snoring frequency, longer snoring duration, and a greater proportion of total sleep time spent snoring. These findings are consistent with increased upper airway tissue load and altered airway mechanics in obesity. The progressive increase in snoring burden across obesity classes further supports the link between excess adiposity and upper airway dysfunction during sleep (12).

Although AHI increased with worsening nutritional status, the correlation between AHI and nocturnal oxygen saturation was relatively modest. This observation underscores an important limitation of AHI as a sole descriptor of disease severity, as it quantifies event frequency but does not account for event duration, depth of desaturation, or cumulative hypoxemic burden.

Importantly, this limitation of AHI has been demonstrated in large population-based cohorts. In the Sleep Heart Health Study, measures of sleep-related intermittent hypoxemia, but not sleep fragmentation, were independently associated with all-cause and cardiovascular mortality, whereas the

prognostic value of AHI was more modest (13). Building on these observations, Azarbarzin and colleagues subsequently showed that hypoxic burden—an integrated measure reflecting the depth and duration of respiratory event-related oxygen desaturation—was a strong predictor of cardiovascular disease-related mortality, whereas AHI showed limited prognostic value (14).

In contrast, obesity in our cohort showed a strong and consistent association with both hypoxemia and snoring-related parameters, suggesting that anthropometric measures may provide complementary information regarding disease impact.

Receiver operating characteristic analysis demonstrated that both BMI and AHI were significantly associated with nocturnal hypoxemia. Although AHI showed numerically higher discriminatory performance, the difference between AHI and BMI was not statistically significant. This finding suggests that BMI, a simple and readily available clinical measure, may provide clinically meaningful information comparable to AHI in identifying patients at risk of nocturnal hypoxemia. This observation is consistent with previous studies demonstrating that measures reflecting hypoxemic burden may better capture the physiological impact of sleep-disordered breathing than AHI alone (11,14,15).

These results should not be interpreted as replacing AHI, but rather as highlighting the complementary value of anthropometric measures in characterizing hypoxemic burden. Given the established prognostic importance of nocturnal hypoxemia, incorporating obesity-related measures alongside conventional event-based indices may improve risk stratification in patients with OSA.

Several limitations should be acknowledged. The relatively small sample size, particularly the limited number of normal-weight patients, represents an important limitation of this study and may reduce the robustness of subgroup comparisons. Therefore, findings related to specific BMI categories, especially normal-weight individuals, should be interpreted with caution. The study population was derived from a single tertiary centre, which may introduce referral bias. Additionally, other factors influencing nocturnal oxygenation, such as pulmonary function or body fat distribution, were not systematically assessed. Prospective studies with larger cohorts are warranted to further explore the mechanistic links between obesity and hypoxemia in OSA. Finally, the cross-sectional nature of the analysis precludes assessment of longitudinal outcomes related to hypoxemic burden.

Taken together, our findings support a more comprehensive approach to OSA assessment that extends beyond event frequency to include obesity severity and nocturnal oxygenation, particularly in patients with severe obesity. From a clinical perspective, these findings suggest that incorporating obesity severity alongside conventional AHI-based classification may improve identification of patients at increased risk of clinically relevant nocturnal hypoxemia.

Disclosure

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Pretilost i noćna hipoksemija u bolesnika s opstruktivnom apnejom u spavanju

Sažetak

Cilj: Ispitati povezanost stupnja debljine i noćne hipoksemije u bolesnika s opstruktivnom apnejom u spavanju (OSA) te usporediti sposobnost indeksa tjelesne mase (BMI) i indeksa apneja-hipopneja (AHI) u prepoznavanju noćne hipoksemije.

Ispitanici i metode: U retrospektivnu presječnu studiju uključeno je 70 odraslih bolesnika s dijagnosticiranom OSA koji su podvrgnuti cjelonoćnoj polisomnografiji. Analizirani su antropometrijski podaci, uključujući BMI i stupanj debljine, te polisomnografski parametri. Noćna hipoksemija definirana je kao zasićenje kisikom (SpO_2) < 90%. Analizirane su povezanosti nutritivnog statusa s noćnom oksigenacijom, hrkanjem i AHI-jem. ROC analizom procijenjena je diskriminacijska sposobnost BMI-ja i AHI-ja za noćnu hipoksemiju.

Rezultati: Medijan dobi iznosio je 58 godina (IQR 47-64), a 76% ispitanika bili su muškarci. Debljina je bila prisutna u 74,3% bolesnika (42,3% stupanj I, 32,7% stupanj II, 25,0% stupanj III). S porastom stupnja debljine zabilježeno je postupno smanjenje srednje noćne saturacije kisika i najniže saturacije ($p = 0,008$ i $p = 0,02$), dok je porast AHI-ja bio bez statističke značajnosti ($p = 0,09$). Noćna hipoksemija najčešće je bila prisutna u bolesnika s debljinom, osobito stupnja III (47%, $p = 0,04$). I BMI (AUC 0,742) i AHI (AUC 0,805) pokazali su značajnu povezanost s noćnom hipoksemijom, bez značajne razlike između ROC krivulja ($p = 0,41$).

Zaključak: Stupanj debljine snažno je povezan s noćnom hipoksemijom u OSA, neovisno o AHI-ju. BMI pruža klinički vrijedne informacije usporedive s AHI-jem u prepoznavanju bolesnika s rizikom za noćnu hipoksemiju te ima komplementarnu ulogu u procjeni težine bolesti.

Ključne riječi: opstruktivna apneja u spavanju; debljina; noćna hipoksemija