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RESEARCH ARTICLE

Association Between Obstructive Sleep Apnea Severity and Cardiometabolic Index in the Context of Cardiovascular Risk

Obstrüktif Uyku Apne Şiddeti ile Kardiyometabolik İndeks Arasındaki İlişki: Kardiyovasküler Risk Bağlamında

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ABSTRACT

Objective: This study aimed to evaluate the relationship between the cardiometabolic index (CMI) and obstructive sleep apnea (OSA) and to investigate its potential clinical relevance in cardiovascular risk assessment.

Materials and Methods: A total of 158 patients who underwent polysomnography (PSG) with a preliminary diagnosis of sleep apnea between January 2022 and July 2023 were included. Demographic characteristics, anthropometric measurements, comorbidities, biochemical parameters, and CMI values were analyzed.

Results: Of the 158 patients, 112 (70.9%) were male. CMI values increased significantly with increasing OSA severity (p=0.03). A weak but statistically significant positive correlation was found between the apnea–hypopnea index (AHI) and CMI (r=0.21, p<0.001, 95% CI: 0.001–0.020). An absolute difference of 0.88 units in CMI was observed between patients with severe OSA and those with simple snoring. Although CMI values were higher in patients with cardiovascular disease, this difference did not reach statistical significance (p = 0.385).

Conclusion: There is a significant positive association between OSA severity and CMI. CMI may serve as a simple and practical marker reflecting an adverse cardiometabolic profile associated with cardiovascular risk in patients with OSA.

Keywords: Cardiometabolic index, cardiovascular disease, obstructive sleep apnea.

ÖZET

Amaç: Çalışmamız, Kardiyometabolik İndeks (KMİ) ile Obstrüktif Uyku Apne (OUA) arasındaki ilişkiyi değerlendirmeyi ve kardiyovasküler risk değerlendirmesindeki potansiyel klinik önemini araştırmayı amaçlamaktadır.

Gereç ve Yöntemler: Ocak 2022 ile Temmuz 2023 tarihleri arasında uyku apnesi ön tanısıyla polisomnografi (PSG) uygulanan toplam 158 hasta çalışmaya dahil edildi. Demografik veriler, antropometrik ölçümler, eşlik eden hastalıklar, biyokimyasal parametreler ve KMİ değerleri analiz edildi.

Bulgular: Çalışmaya 112'si erkek (%70,9) olmak üzere toplam 158 hasta alındı. Kardiyometabolik indeks değerlerinin, OUA şiddeti arttıkça anlamlı derecede yükseldiği saptandı (p=0,03). Apne-hipopne indeksi ile KMİ arasında düşük düzeyde pozitif korelasyon bulundu (r=0,21, p<0,001, %95 GA: 0,001-0,020). Ağır OUA olan hastalar ile basit horlama olan hastalar arasında KMİ değerlerinde 0,88 birimlik mutlak fark tespit edildi. Kardiyometabolik indeks, kardiyovasküler hastalığı olan hastalarda daha yüksek saptanmasına rağmen, aralarında anlamlı derecede fark bulunmadı (p=0,385).

Sonuç: Obstrüktif uyku apnesi şiddeti ile KMİ arasında anlamlı pozitif bir ilişki saptadık. Kardiyometabolik indeksin, uyku apnesi olan hastalarda kardiyovasküler riskle ilişkili olumsuz kardiyometabolik profili yansıtan basit ve pratik bir belirteç olarak kullanılabileceğini öne sürüyoruz.

Anahtar Kelimeler: Kardiyometabolik indeks, kardiyovasküler hastalık, obstrüktif uyku apnesi,

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INTRODUCTION

Obstructive sleep apnea (OSA) is a disorder characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxemia and sleep fragmentation (1). Epidemiological data indicate that OSA affects approximately 4% of men and 2% of women aged 30–60 years who present with significant daytime sleepiness, with prevalence expected to increase further in the aging population, particularly among individuals aged 65 years and older (2,3). The development of OSA is influenced by multiple anatomical and physiological factors that promote upper airway narrowing or instability. Among these, male sex and obesity represent the most prominent risk factors, while smoking, alcohol consumption, and the use of sedative or hypnotic agents further contribute to disease susceptibility (4). Clinically, OSA is characterized by loud snoring, witnessed apneas, excessive daytime sleepiness, nocturnal choking, and disrupted sleep architecture (5–7).

The pathophysiology of OSA is complex and involves a combination of intermittent hypoxia, oxidative stress, and systemic inflammation. Chronic intermittent hypoxemia and sleep fragmentation trigger inflammatory pathways and oxidative injury, which play a central role in disease progression (8). Importantly, these mechanisms are also implicated in the development of cardiovascular and metabolic disorders. A growing body of evidence has demonstrated that OSA severity is closely associated with major cardiovascular risk factors, including hypertension, diabetes mellitus, hyperlipidemia, and physical inactivity (9). These shared risk factors converge on common biological pathways, such as endothelial dysfunction, increased sympathetic activity, and accelerated atherosclerosis, thereby establishing OSA as an independent contributor to cardiovascular morbidity (10).

The cardiometabolic index (CMI) is a new measure first introduced by Wakabayashi et al. This index is calculated using the waist-to-height ratio (WHtR), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C), which are routinely measured in clinical practice (11). CMI is calculated as: CMI = (Triglycerides/HDL-cholesterol) x (Waist circumference/ Height). Given the shared cardiometabolic risk profile between OSA and cardiovascular disease, CMI appears to be a relevant marker for reflecting the adverse metabolic profile in these patients (12,13). By integrating measures of central adiposity and lipid metabolism, CMI provides a comprehensive assessment of cardiometabolic risk.

Given the substantial overlap between the pathophysiological mechanisms of OSA and cardiometabolic disorders, CMI may represent a clinically relevant marker for identifying adverse metabolic profiles in patients with OSA (12,13). In this context, the present study was designed to evaluate the relationship between CMI and OSA severity and to explore its potential role in cardiovascular risk assessment.

MATERİALS AND METHODS

This retrospective study included patients who were evaluated for suspected sleep apnea in the Chest Diseases Clinic and underwent overnight polysomnography (PSG) between January 2022 and July 2023. Demographic characteristics, anthropometric measurements, smoking status, comorbidities, complete blood count, lipid profile, hyperlipidemia treatment status, and PSG findings were obtained from the hospital's electronic medical records system and systematically analyzed. Patients were categorized into four groups according to their apnea–hypopnea index (AHI): simple snoring (AHI<5), mild OSA (AHI 5–15), moderate OSA (AHI 15–30), and severe OSA (AHI > 30). The primary objective was to investigate the relationship between OSA severity and the cardiometabolic index. The study was conducted in accordance with the principles of the Declaration of Helsinki. Due to its retrospective design, informed consent was not required. Ethical approval was obtained from the Health Sciences Ethics Committee of Balıkesir University Faculty of Medicine (Decision No: 2024/24, Date: 21/02/2024).

Statistical Analysis

All statistical analyses were performed using SPSS version 23.0. A priori power analysis conducted using G*Power indicated that a sample size of 158 patients would provide 85% power for the primary outcome (α=0.05, Cohen's f=0.54), while the power for secondary outcomes ranged between 70% and 75%. Continuous variables were expressed as mean ± standard deviation for normally distributed data and as median (IQR) for non-normally distributed data. Categorical variables were presented as counts and percentages. Normality was assessed using the Kolmogorov–Smirnov test. Comparisons between categorical variables were performed using the chi-square test, with Pearson's $\chi 2$ or Fisher's exact test applied as appropriate. For comparisons among more than two independent groups, one-way ANOVA was used for normally distributed variables, while the Kruskal–Wallis test was used for non-normally distributed variables. When overall group differences were statistically significant, post-hoc analyses were conducted to identify the specific groups contributing to the difference.

Correlation analyses were performed to evaluate relationships between continuous variables, using Pearson's correlation coefficient for normally distributed data and Spearman's rank correlation coefficient for non-normally distributed data. For comparisons between two independent groups with non-normally distributed variables, the Mann–Whitney U test was applied. A critical α value of 0.05 was considered significant.

RESULTS

A total of 158 patients were included in the study, of whom 112 (70.9%) were male. The baseline characteristics of the study population are presented in Table 1. According to OSA severity, 22 patients were classified as simple snoring, 37 as mild OSA, 37 as moderate OSA, and 62 as severe OSA. There was no significant difference in sex distribution across the groups (p= 0.79) (Table 2). However, age differed significantly among groups (p= 0.01), with post hoc analysis (Tukey HSD) indicating that patients in the simple snoring group were significantly younger than those in the moderate and severe OSA groups (M=-9.38 p=0.014, M=-8.20 p=0.023, respectively)

Table 1. General characteristics of all patients

Variable	n= 158
Age	52.91±11.7
Gender (M/F)	116/42
Comorbidity (Yes/No)	115/43
BMI	32.54±6.2
CMI	2.68±1.57

Data are presented as mean ± standard deviation. BMI: Body mass index, CMI: Cardiometabolic Index

(Table 4). A history of smoking was present in 22.1% (n = 35) of the study population, with no significant differences between OSA severity groups (p= 0.753) or according to cardiovascular disease status (p= 0.410).

Hyperlipidemia treatment was more frequently observed in patients with OSA compared to the control group; however, this difference did not reach statistical significance when all patients were considered (p= 0.514). Similarly, no significant differences were observed in treatment rates across OSA severity subgroups (p= 0.745). In contrast, hyperlipidemia treatment was significantly more common among patients with cardiovascular disease (p< 0.001). Comorbidities were present in 73.4% (n= 116) of patients. Cardiovascular disease was identified in 34.2%, hypertension in 58.9%, and diabetes mellitus in 30.4% of the cohort. The prevalence of cardiovascular disease did not differ significantly across OSA severity groups (p=0.151) (Table 2). The distribution of cardiovascular diseases according to groups is shown in Figure 1. Although CMI values were higher in patients with a history of cardiovascular disease

Table 2. Comparison of gender and comorbidities between groups

Variable	Simple snoring	Mild OSA	Moderate OSA	Severe OSA	p value
Gender (F/M)	6/16	11/26	13/24	16/46	0.796
Hypertension n (%)	7 (31.8)	23 (62.4)	24 (64.8)	39 (62.9)	0.051
Diabetes Mellitus n (%)	8 (36.3)	6 (16.2)	13 (35.1)	20 (33.8)	0.201
Cardiovascular Disease n (%)	4 (19.2)	11 (29.7)	12 (32.4)	27 (43.5)	0.151

Table 3. Comparison of age, laboratory parameters, and indices between groups

Variable	Simple snoring (n=22)	Mild OSA (n=37)	Moderate OSA (n=37)	Severe OSA (n=62)	p value
Age	45.86±11.99	52.84±12.50	55.24±11.01	54.06±10.80	0.017
BMI	27.51 (26.43-31.23)	29.9 (27.58-35.15)	31.14 (28.85-35.22)	33.14 (29.67-37.39)	0.003
CMI	1.91 (1.45-2.74)	2.27 (1.51-3.63)	2.05 (1.29-3.57)	2.79 (1.96-3.82)	0.031
HDL	47.09±9.56	46.64±8.08	52.10±13.02	45.37±8.66	0.012
LDL	125.50±36.25	131±24.14	118.02±40.63	132.39±35.19	0.435
Cholesterol	197.18±37.14	208.73±39.49	208.25±42.70	214.62±42.70	0.403
Triglyceride	144 (113.50-191.75)	178 (122-223.50)	162 (106.50-227)	173.50 (127.75-241.25)	0.219
Hemoglobin	14.42±1.07	14.07±1.50	13.76±1.64	14.03±1.90	0.535
Lymphocyte	2.40±0.77	2.48±0.78	2.47±0.60	2.42±0.95	0.971
Platelet	258.63±46.16	260.89±66.06	284.35±63.16	278.71±63.65	0.230

Data are presented as mean ± standard deviation, median (25th Percentile – 75th Percentile), BMI: Body mass index, CMI: Cardiometabolic Index, HDL: High density lipoprotein, LDL: Low density lipoprotein

Table 4. Tukey HSD Post-hoc test results for age and HDL across OSA Groups

Variable	Mean difference	%95 CI	p value
Age			
Simple snoring - Moderate OSA	-9.38	-(17.38 - 1.38)	0.014
Simple snoring - Severe OSA	-8.20	-(15.57 - 0.83)	0.023
HDL			
Moderate OSA - Severe OSA	6.74	0.36 – 13.11	0.035

HDL: High density lipoprotein

Table 5. Comparison of CMI with comorbidity, hypertension, and cardiovascular disease variables

Variable	Yes median (IQR)	No median (IQR)	p value
Comorbidity	2.10 (1.40-3.31)	3.02 (2.19-3.89)	0.002
Hypertension	2.12 (1.34-3.25)	2.76 (1.85-3.80)	0.022
Cardiovascular Disease	2.44 (1.57-3.66)	2.31 (1.60-3.18)	0.385

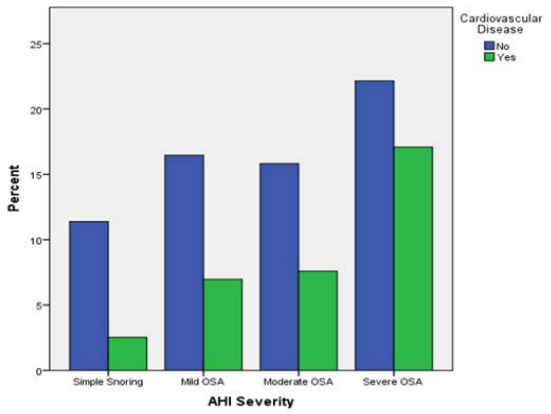

Figure 1. Incidence of cardiovascular disease by case group

[2.44 (1.57–3.66)] compared to those without [2.31 (1.60–3.18)], no significant difference was observed between the groups. In addition, no association was found between the number of comorbidities and CMI (p= 0.88).

Body mass index (BMI) differed significantly across groups (p = 0.03), with higher values observed in patients with more severe OSA. Post hoc analysis demonstrated that patients with severe OSA had significantly higher BMI compared to those with simple snoring (p< 0.001). Additionally, BMI was significantly higher in patients with hypertension and cardiovascular disease (p= 0.00, p= 0.00, respectively), and a weak positive correlation was observed between BMI and the number of comorbidities (r=0.26, p=0.00). No significant association was found between BMI and CMI (p= 0.25). CMI values differed significantly according to OSA severity (p= 0.03) (Table 3). Post hoc analysis revealed that patients with severe OSA had significantly higher CMI values compared to those with simple snoring (p<0.001), whereas no significant differences were observed between other group pairs. No significant difference in CMI was found between patients receiving and not receiving hyperlipidemia treatment (p=0.842).

A low-level, positive, and statistically significant correlation was found between AHI and CMI (r=0,21, p<0,001, 95% CI: 0,001-0,020). No statistically significant differences were observed between OSA severity groups in terms of LDL cholesterol, total cholesterol, or triglyceride levels (p=0.435, p=0.403, p=0.219, respectively) (Table 3). However, HDL cholesterol levels differed significantly across groups (one-way ANOVA, p=0.012). Post hoc analysis using the Games–Howell test demonstrated that HDL levels were significantly higher in the moderate OSA group compared to the severe OSA group (p = 0.035) (Table 4), while no other pairwise differences reached statistical significance.

DISCUSSION

In the present study, the relationships between obstructive sleep apnea (OSA) severity, cardiometabolic index (CMI), and related clinical parameters were comprehensively evaluated. Our findings demonstrated a significant association between OSA severity and CMI, with higher CMI values observed in patients with more severe disease. In addition, a weak but statistically significant positive correlation between the apnea–hypopnea index (AHI) and CMI was identified, suggesting that increasing disease severity is accompanied by a progressively adverse cardiometabolic profile. OSA is increasingly recognized not only as a sleep-related breathing disorder but also as a systemic condition with substantial cardiovascular implications. The underlying pathophysiological mechanisms include chronic intermittent hypoxemia, oxidative stress, systemic inflammation, and sustained sympathetic nervous system activation. These processes contribute to endothelial dysfunction and accelerated atherosclerosis, thereby increasing cardiovascular risk independently of traditional risk factors (10). Furthermore, these mechanisms are closely linked to disturbances in lipid metabolism; OSA-related hypoxemia and sympathetic overactivity may promote dyslipidemia, particularly through alterations in HDL metabolism and triglyceride clearance (8,14). In this context, CMI, which integrates measures of central adiposity and lipid parameters, appears to be a relevant composite marker reflecting the adverse cardiometabolic milieu in patients with OSA.

In our cohort, the prevalence of cardiometabolic comorbidities was notably high, with 34.2% of patients having cardiovascular disease, 58.9% hypertension, and 30.4% diabetes mellitus. These findings are consistent with previous reports demonstrating a higher prevalence of such conditions among patients with OSA compared to the general population (10). Moreover, prior studies have reported a direct relationship between OSA severity and the prevalence of cardiometabolic diseases (15). In line with this evidence, our data also indicated an increasing prevalence of cardiovascular disease with increasing OSA severity, although this trend did not reach statistical significance. Body mass index (BMI) was significantly associated with OSA severity in our study, with higher BMI values observed in more severe disease groups. This finding supports the well-established role of obesity as a major predisposing factor for OSA (10,15–17). Increased adiposity contributes to upper airway collapsibility and plays a central role in the pathogenesis of OSA (18). Additionally, we observed that BMI was significantly higher in patients with hypertension and cardiovascular disease, and that the number of comorbidities showed a weak positive correlation with BMI. These results suggest that excess body weight represents a shared and important risk factor linking OSA and cardiovascular disease. Consistent with this, Drager et al. reported that OSA and obesity synergistically increase cardiovascular risk (13). In contrast, no statistically significant relationship was found between the number of comorbidities and CMI in our study. This finding suggests that cardiometabolic risk may not be solely determined by the presence of comorbid conditions, but also by their severity and management. Although higher CMI values were observed in patients with cardiovascular disease, this difference did not reach statistical significance.

One possible explanation is the more frequent use of lipid-lowering therapies in these patients, which may have attenuated differences in CMI values. Taken together, these findings suggest that CMI may still have potential utility as a marker for cardiometabolic risk, particularly in patients with OSA.

A key finding of our study is the significant association between OSA severity and CMI. Post hoc analysis revealed an absolute difference of 0.88 units in CMI between patients with severe OSA and those with simple snoring. In addition, the presence of a weak but significant linear correlation between AHI and CMI further supports this relationship. The role of CMI as a predictor of cardiovascular risk has been increasingly investigated in recent years. Wakabayashi et al. originally defined CMI as a composite marker reflecting both adiposity and lipid abnormalities (19), while Shi et al. reported its utility in predicting coronary artery disease risk (20). In this context, our findings suggest that increasing OSA severity is associated with a more unfavorable cardiometabolic profile, as reflected by elevated CMI values. Previous studies have identified OSA as a risk factor for cardiovascular, cerebrovascular, and metabolic diseases (13). In a longitudinal study, Cai et al. demonstrated that CMI is an effective predictor of cardiovascular disease risk in patients with OSA and hypertension, with a mean follow-up of 6.83 years (12). These findings support our results and further indicate that CMI may provide additional value in cardiovascular risk stratification among patients with OSA.

Interestingly, our lipid profile analysis did not reveal significant differences in LDL cholesterol, total cholesterol, or triglyceride levels across OSA severity groups, whereas HDL cholesterol levels differed significantly. This finding contrasts with some previous studies reporting lipid abnormalities in OSA patients. The relationship between OSA and lipid metabolism is complex and likely influenced by multiple factors, including obesity, insulin resistance, systemic inflammation, and pharmacological treatments (8,13). Although intermittent hypoxemia may theoretically impair lipid metabolism via increased sympathetic activity (14), our findings suggest that concurrent treatments, particularly lipid-lowering therapies in a population with a high burden of cardiovascular disease, may have mitigated these differences. CMI has been described as a marker reflecting both abdominal obesity and dyslipidemia (11). In our study, no significant association was observed between BMI and CMI, suggesting that CMI may capture aspects of cardiometabolic risk that are not fully reflected by general adiposity measures. Supporting this, Wang et al. reported that CMI may better reflect visceral adiposity and may therefore be a more sensitive indicator of cardiometabolic risk compared to BMI (21). In addition, our study also revealed a significant association of BMI with cardiovascular disease and hypertension. All these findings support the importance of obesity in patients with OSA and its potential role in increasing cardiovascular risk.

From a clinical perspective, CMI offers several practical advantages. It is derived from routinely measured clinical parameters—waist circumference, height, triglycerides, and HDL cholesterol—without requiring additional diagnostic procedures. Moreover, the lack of association between CMI and BMI in our findings suggests that CMI may provide complementary information beyond traditional anthropometric indices. Therefore, CMI may represent a simple, accessible, and clinically useful tool for evaluating cardiometabolic risk in patients with OSA, particularly in resource-limited settings.

Study Limitations

Nevertheless, several limitations should be acknowledged. The retrospective, single-center, and cross-sectional design limits the ability to establish causal relationships. Although the sample size was sufficient for the primary outcome, the statistical power for secondary analyses was moderate, and thus these findings should be interpreted with caution. In addition, the high prevalence of comorbidities and the potential confounding effects of lipid-lowering therapies may have influenced the observed associations. Finally, the long-term prognostic value of CMI in patients with OSA was not assessed.

CONCLUSION

In conclusion, our study demonstrated that increasing OSA severity is associated with higher CMI values and a more adverse cardiometabolic profile. These findings support the potential role of CMI as a simple and practical marker for cardiovascular risk assessment in patients with OSA. However, further large-scale, prospective studies are needed to better define its independent predictive value and clinical applicability.

DECLARATIONS

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