Original article

doi:10.5633/amm.2026.0407

ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA SEVERITY AND CARDIOMETABOLIC COMORBIDITIES

Jelena Aleksić[1], Miloš Jovanović[2], Dragana Stojiljković[1], Zoran Stamenković[1]

[1]University Clinical Center Niš, Clinic for Pulmonology, Niš, Serbia

[2]University Clinical Center Niš, Clinic for Cardiovascular Diseases, Niš, Serbia

Contact: Jelena Aleksić

48/17 Studenička Str., Niš, Serbia

E-mail: jelenaaleksic29593@gmail.com

Obstructive sleep apnea (OSA) is a chronic sleep-disordered breathing condition associated with increased cardiovascular and metabolic risk. The aim of this study was to investigate the association between OSA severity and the presence and number of cardiometabolic comorbidities. A retrospective analysis was conducted on 113 patients with OSA (AHI ≥ 5) at the Department for Sleep-Related Breathing Disorders, Clinic for Pulmonology, University Clinical Center Niš, January–July 2024. Based on the apnea-hypopnea index (AHI), subjects were divided into three groups: mild (n = 24), moderate (n = 29), and severe OSA (n = 60). Anthropometric, polygraphy, and laboratory parameters were analyzed, along with comorbidity prevalence. The mean BMI was 34.71 ± 6.69 kg/m², with progressive increase from mild to severe OSA (29.91 vs. 36.67 kg/m², $p < 0.001$). Neck and waist circumference also increased significantly with disease severity (all $p < 0.001$). Minimum oxygen saturation decreased from 84.38% to 68.93% ($p < 0.001$). Arterial hypertension was the most prevalent comorbidity (73.5%), with significantly higher prevalence in severe OSA (81.7%) compared to mild (45.8%, $p = 0.003$). Overall, 87.6% of patients had at least one comorbidity, with a significant increase from mild (70.8%) to severe OSA (95.0%, $p = 0.010$). Laboratory cardiometabolic markers showed no significant differences between groups. OSA severity is strongly associated with obesity, nocturnal hypoxia, and the prevalence of hypertension, while the direct impact on lipid profile and glycemic control requires further evaluation.

Key words: obstructive sleep apnea, comorbidity, cardiometabolic risk

Originalni rad

doi:10.5633/amm.2026.0407

POVEZANOST TEŽINE OPSTRUKTIVNE SLEEP APNEJE SA KARDIOMETABOLIČKIM KOMORBIDITETIMA

Jelena Aleksić[1], Miloš Jovanović[2], Dragana Stojiljković[1], Zoran Stamenković[1]

[1]Univerzitetski Klinički centar Niš, Klinika za pulmologiju, Niš, Srbija

[2]Univerzitetski Klinički centar Niš, Klinika za kardiovaskularne bolesti, Niš, Srbija

Kontakt: Jelena Aleksić

Studenička 48/17 Niš-Pantelej

E-mail: jelenaaleksic29593@gmail.com

Opstruktivna sleep apneja (OSA) je hronični poremećaj disanja tokom sna udružen sa povećanim kardiovaskularnim i metaboličkim rizikom. Cilj studije bio je da se ispita povezanost težine OSA sa prisustvom i brojem kardiometaboličkih komorbiditeta. Sprovedena je retrospektivna analiza 113 pacijenata sa dijagnostikovanom OSA (AHI ≥ 5) lečenih na Odeljenju za respiratorne poremećaje sna Klinike za pulmologiju Univerzitetskog kliničkog centra u Nišu u periodu januar–jul 2024. Na osnovu apneja hipopneja indeksa (AHI), ispitanici su podeljeni u tri grupe: blaga (n = 24), umerena (n = 29) i teška OSA (n = 60). Analizirani su antropometrijski, poligrafski i laboratorijski parametri, kao i prevalencija komorbiditeta. Prosečan BMI iznosio je 34.71 ± 6.69 kg/m², sa progresivnim porastom od blage do teške OSA (29.91 vs. 36.67 kg/m², $p < 0.001$). Obim vrata i obim struka značajno su rasli sa težinom bolesti (svi $p < 0.001$). Minimalna saturacija kiseonikom opadala je od 84.38% do 68.93% ($p < 0.001$). Arterijska hipertenzija bila je najčešći komorbiditet (73.5%), sa značajno većom prevalencijom u teškoj OSA (81.7%) u poređenju sa blagom (45.8%, $p = 0.003$). Ukupno 87.6% ispitanika imalo je bar jedan komorbiditet, sa značajnim porastom od blage (70.8%) do teške OSA (95.0%, $p = 0.010$). Laboratorijski kardiometabolički markeri nisu pokazali značajne razlike među grupama. Težina OSA je snažno povezana sa gojaznošću, noćnom hipoksijom i prevalencijom hipertenzije, dok direktan uticaj na lipidni profil i glikemijsku kontrolu zahteva dalju evaluaciju.

Ključne reči: opstruktivna sleep apneja, komorbiditeti, kardiometabolički rizik

Introduction

Obstructive sleep apnea (OSA) is a chronic sleep-disordered breathing condition characterized by recurrent episodes of complete (apnea) or partial (hypopnea) collapse of the upper airway, leading to intermittent hypoxia, sleep fragmentation, and sympathetic nervous system activation [1,2]. According to the latest estimates, OSA affects nearly one billion people worldwide. A large number of patients remain undiagnosed [2]. It has been confirmed that OSA represents an independent risk factor for the development and progression of cardiovascular and metabolic diseases [3,4]. Intermittent hypoxia, the central pathophysiological mechanism of OSA, triggers a cascade of harmful processes including oxidative stress, endothelial dysfunction, systemic inflammation, and sympathetic nervous system activation [3,5]. These mechanisms contribute to the development of arterial hypertension, atherosclerosis, insulin resistance, and dyslipidemia [4,6]. Numerous epidemiological studies have confirmed a strong association between OSA and arterial hypertension (AH) [6,7,8], type 2 diabetes mellitus (DM) [9], dyslipidemia [10], and cardiovascular events such as myocardial infarction (MI) and cerebrovascular accident (CVA) [11]. Marin et al. demonstrated in a prospective study that untreated severe OSA significantly increases the risk of fatal and non-fatal cardiovascular events [12]. Furthermore, obesity represents both a cause and a consequence2 of OSA. Weight gain worsens upper airway collapse, while sleep fragmentation and hormonal imbalance associated with OSA further contribute to adipose tissue accumulation [1,13]. Although the relationship between OSA and individual cardiometabolic comorbidities is well documented, there is less data on how the presence and number of these comorbidities change depending on OSA severity, particularly in our population. The classification of OSA by the apnea-hypopnea index (AHI) into mild, moderate, and severe forms [14] allows assessment of disease severity, but the question remains whether this gradation is accompanied by a proportionally greater cardiometabolic burden. Understanding this association is of key importance for an individualized treatment approach and adequate overall risk assessment in patients with OSA.

Aim

The aim of this study was to determine cardiometabolic comorbidities in patients with confirmed sleep apnea and their association with obstructive sleep apnea severity.

Materials and methods

A retrospective analysis was conducted on 113 hospitalized patients diagnosed with obstructive sleep apnea (OSA) who were treated at the Department for Sleep-Related Breathing Disorders, Clinic for Pulmonology, University Clinical Center Niš, in the period from January 1, 2024 to July 1, 2024. The diagnosis of OSA was established based on respiratory polygraphy in accordance with the recommendations of the American Academy of Sleep Medicine [15]. The inclusion criterion was AHI ≥ 5 events per hour. Patients with acute infections, acute coronary syndrome, stroke within the previous 6 months, severe renal and hepatic insufficiency were excluded from the study. All subjects underwent nocturnal respiratory polygraphy [16]. The following parameters were analyzed: apnea-hypopnea index (AHI), oxygen desaturation index (ODI), minimum oxygen saturation (SpO2), mean nocturnal saturation, andtime spent with SpO2 < 90%. AHI represents the average number of apneas and hypopneas per hour of sleep. ODI denotes the number of desaturation episodes per hour. Minimum SpO2 is the lowest recorded oxygen saturation during sleep, while mean SpO2 is the average saturation throughout the entire recording. Time with SpO2 < 90% represents the percentage of total sleep time spent with saturation below 90%. Based on AHI values, subjects were divided into three groups: mild OSA (AHI 5–14.9/h), moderate OSA (AHI 15–29.9/h), and severe OSA (AHI ≥ 30/h) [12]. Anthropometric measurements were performed: body weight and height, body mass index (BMI, kg/m2), neck circumference, and waist circumference. Data on the presence of comorbidities were collected from medical records and patient history. For each subject, the total number of comorbidities was counted (from 8 defined categories: AH, DM, dyslipidemia, asthma, chronic obstructive pulmonary disease (COPD), cardiovascular diseases, hypothyroidism, and benign prostatic hyperplasia (BPH)). The distribution of the number of comorbidities by groups was presented, as well as the mean number (Mean ± SD). Venous blood samples were collected in the morning hours after an overnight fast. The following parameters were analyzed: total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, and glycosylated hemoglobin (HbA1c). For comparing the mean number of comorbidities between groups, the Kruskal-Wallis test was used ($p = 0.063$), which shows a trend but does not reach statistical significance. For the selection of the statistical test, the normality of data distribution was first assessed using the Shapiro-Wilk test in each group. If data in all groups were normally distributed ($p > 0.05$ on the Shapiro-Wilk test), †One-way ANOVA (parametric test) was applied. If at least one group deviated from normal distribution, *Kruskal-Wallis test (non- parametric alternative to ANOVA) was applied. For each parameter, the arithmetic mean (Mean) and standard deviation (SD) were presented, and the p-value indicated statistical significance of the difference between groups — if $p < 0.05$, the difference was considered statistically

significant. Absolute frequencies and percentages of subjects with a given comorbidity were presented for each group. Cardiovascular diseases included: cerebrovascular accident, myocardial infarction, atrial fibrillation (AF), arrhythmias, tachycardia, and bradycardia. For comparison between groups, the chi-square test ($\chi 2$) was used.

Results

Of the total 113 hospitalized patients diagnosed with obstructive sleep apnea, 34 (30.1%) were women, while 79 (69.9%) were men. The age of the subjects ranged from 27 to 77 years, with a mean age of 53.36 ± 11.35 years.

Demographic and anthropometric characteristics of the entire sample are presented in Table 1. The mean body mass index was 34.71 ± 6.69 kg/m2, indicating that the majority of subjects belonged to class I obesity (BMI 30–34.9). The mean body weight was 104.40 ± 20.45 kg, with a mean height of 173.48 ± 9.47 cm. Elevated values of neck circumference (43.83 ± 4.89 cm) and waist circumference (115.50 ± 16.25 cm) were measured in patients, indicating a high prevalence of central obesity and adipose tissue accumulation in the upper airway region.

Table 1. Demographic and anthropometric characteristics of the respondents

Parameter	**Mean ± SD from 27 to 77 years old**
Age (years)	53.23 ± 11.31
BMI (kg/m²)	34.56 ± 6.73
Body mass (kg)	103.87 ± 21.02
Height (cm)	173.35 ± 9.59
Neck circumference (cm)	43.72 ± 5.02
Waist circumference (cm)	114.95 ± 16.80

* BMI-body mass index

The results of nocturnal respiratory polygraphy are presented in Table 2. The mean AHI was 34.43 ± 21.50 events/h, which classifies the subjects into the severe OSA category. The oxygen desaturation index (ODI) was 35.78 ± 24.83 events/h, indicating frequent desaturation episodes. The mean minimum oxygen saturation was 75.04 ± 12.48%, and the mean nocturnal saturation was 91.24 ± 4.45%. Subjects spent an average of 19.11 ± 23.89% of total sleep time with saturation below 90%. A large standard deviation was observed, indicating significant heterogeneity in the

degree of nocturnal hypoxemia among subjects, ranging from those with minimal desaturation to those with prolonged and severe hypoxemia.

Table 2. Polygraph parameters of the respondents

Parameter	Mean ± SD
AHI (events/h)	34.43 ± 21.50
ODI (events/h)	35.78 ± 24.83
Minimum SpO2 (%)	75.04 ± 12.48
Average SpO2 (%)	91.24 ± 4.45
Time SpO2 < 90% (%)	19.11 ± 23.89 111

* AHI- apnea-hypopnea index *ODI- oxygen desaturation index

Laboratory cardiometabolic parameters are presented in Table 3. The mean value of total cholesterol was 5.39 ± 1.22 mmol/L. LDL cholesterol (3.35 ± 0.82 mmol/L) was above the target value of 3.0 mmol/L, while triglycerides (2.40 ± 1.32 mmol/L) were significantly above the upper limit of the reference range (1.7 mmol/L). The HDL cholesterol value was 1.22 ± 0.57 mmol/L. The mean glycosylated hemoglobin (HbA1c) was 5.87 ± 0.93%.

Table 3. Laboratory parameters of the subjects

Parameter	Mean ± SD
Total Cholesterol (mmol/L)	5.39 ± 1.22
LDL (mmol/L)	3.35 ± 0.82
HDL (mmol/L)	1.22 ± 0.57
Triglycerides (mmol/L)	2.40 ± 1.32
HbA1c (%)	5.87 ± 0.93

* HbA1c- glycosylated hemoglobin

Subjects are presented according to AHI index severity in Table 4. The most prevalent was the severe form of OSA (AHI ≥ 30) in 60 subjects (53.1%). Moderate OSA (AHI 15–29.9) was present in 29 subjects (25.7%), while mild OSA (AHI 5–14.9) was recorded in 24 subjects (21.2%).

Table 4. Patient groups according to OSA severity based on AHI

Weight	OSA N (%)
Mild (AHI 5–14.9)	24 (21.2%)
Moderate (AHI 15–29.9)	29 (25.7%)
Severe (AHI ≥ 30)	60 (53.1%)

Anthropometric and metabolic parameters in the study groups according to AHI index severity are presented in Table 5. The results showed statistically significant differences for all anthropometric parameters. BMI progressively increased from 29.91 ± 4.83 kg/m2 in mild to 36.67 ± 6.34 kg/m2 in severe OSA ($p < 0.001$), neck circumference from 40.38 ± 3.03 cm to 45.67 ± 4.75 cm ($p < 0.001$), and waist circumference from 103.79 ± 12.49 cm to 121.69 ± 13.95 cm ($p < 0.001$). Among polygraphy parameters, ODI increased from 10.14 ± 7.26 in mild to 50.71 ± 23.34 events/h in the severe group ($p < 0.001$), while minimum SpO2 progressively decreased from 84.38 ± 4.73% to 68.93 ± 12.65% ($p < 0.001$), indicating increasingly pronounced intermittent hypoxia with increasing OSA severity.

Table 5. Comparative analysis by severity of OSA

Parameters	**Mild**	**Moderate**	**Severe**	**P value**
BMI (kg/m²)	29.91 ± 4.83	34.68 ± 6.85	36.67 ± 6.34	<0.001*
Neck Circumference (cm)	40.38 ± 3.03	43.18 ± 4.78	45.67 ± 4.75	<0.001†
waist circumference (cm)	103.79 ± 12.49	113.39 ± 17.62	121.69 ± 13.95	<0.001†
ODI (events/h)	10.14 ± 7.26	26.11 ± 11.72	50.71 ± 23.34	<0.001*
Minimum SpO2	84.38 ± 4.73	79.93 ± 9.38	68.93 ± 12.65	<0.001*
Total Cholesterol	5.84 ± 0.91	5.06 ± 1.45	5.39 ± 1.15	0.161
LDL (mmol/L)	3.67 ± 0.78	3.18 ± 1.05	3.32 ± 0.68	0.298
HDL (mmol/L)	1.60 ± 1.05	1.08 ± 0.24	1.14 ± 0.32	0.063
Triglycerides	2.28 ± 1.28	2.15 ± 0.78	2.60 ± 1.56	0.430
HbA1c (%)	5.58 ± 0.40	6.16 ± 1.40	5.79 ± 0.65	0.483

Laboratory cardiometabolic parameters — total cholesterol (p = 0.161), LDL (p = 0.298), HDL (p = 0.063), triglycerides (p = 0.430), and HbA1c (p = 0.483) — showed no statistically significant differences between groups, although a decreasing trend in HDL cholesterol was observed from mild (1.60 ± 1.05 mmol/L) to severe OSA (1.14 ± 0.32 mmol/L) with borderline significance.

The prevalence of cardiometabolic comorbidities by OSA severity groups is presented in Table 6. AH was the most common comorbidity in the entire sample, present in 83 subjects (73.5%). The distribution of AH showed a statistically significant difference between groups (p = 0.003). In the mild OSA group, hypertension was present in 45.8%, in moderate 79.3%, and in severe OSA 81.7% (p < 0.001). Diabetes mellitus was present in 21 subjects (18.6%), and cardiovascular diseases (CVA, arrhythmias, MI, AF, tachycardia) in 21 subjects (18.6%), without statistically significant differences between groups. Dyslipidemia was identified in 11 subjects (9.7%), asthma in 15 (13.3%), and BPH in 8 (7.1%). A total of 99 subjects (87.6%) had at least one comorbidity, with a statistically significant increase in prevalence from mild (70.8%) to severe OSA (95.0%, chi-square test p = 0.010), suggesting that more severe disease carries a greater cumulative comorbidity burden. The distribution of the number of comorbidities by OSA severity groups is presented in Table 7. Of the total number of subjects, 14 (12.4%) had no comorbidity, 50 (44.2%) had one, 34 (30.1%) two, and 15 (13.3%) three or more comorbidities. The mean number of comorbidities showed an increasing trend with OSA severity — from 1.08 ± 0.91 in mild,through 1.62 ± 1.22 in moderate, to 1.60 ± 0.84 in the severe group, with borderline statistical significance (Kruskal-Wallis p = 0.063). A difference was obtained in the percentage of patients without any comorbidity. In mild OSA, 29.2% were without associated diseases, in the severe group without comorbidities there were only 5.0%, which is statistically significantly lower than the number of respondents 23 (38.3%) who had two simultaneous comorbidities with severe OSA.

Table 6. Prevalence of comorbidities by severity of OSA

Parameter	Total	Mild	Moderate	Severe	p value
Arterial hypertension	83 (73.5%)	11 (45.8%)	23 (79.3%)	49 (81.7%)	**0.003**
Diabetes mellitus	21 (18.6%)	2 (8.3%)	7 (24.1%)	12 (20.0%)	0.311
Dyslipidemia	11 (9.7%)	1 (4.2%)	3 (10.3%)	7 (11.7%)	0.573
Asthma	15 (13.3%)	5 (20.8%)	2 (6.9%)	8 (13.3%)	0.330
COPD	3 (2.7%)	1 (4.2%)	2 (6.9%)	0 (0.0%)	0.145
Cardiovascular diseases	21 (18.6%)	4 (16.7%)	8 (27.6%)	9 (15.0%)	0.346
Hypothyroidism	7 (6.2%)	2 (8.3%) 0	1 (3.4%)	4 (6.7%)	0.745
BHP	8 (7.1%)	0 (0.0%)	1 (3.4%)	7 (11.7%)	0.115
Presence of ≥1 comorbidity	**99 (87.6%)**	**17 (70.8%)**	**25 (86.2%)**	**57 (95.0%)**	**0.010**

* COPD - chronic obstructive pulmonary disease * BPH - benign prostatic hyperplasia

Table 7. Number of comorbidities by severity of OSA

Number of comorbidities	Total	Mild	Moderate	Severe
0	14 (12.4%)	7 (29.2%)	4 (13.8%)	3 (5.0%)
1	50 (44.2%)	10 (41.7%)	13 (44.8%)	27 (45.0%)
2	34 (30.1%)	5 (20.8%)	6 (20.7%)	23 (38.3%)
≥3	15 (13.3%)	2 (8.3%)	6 (20.7%)	7 (11.7%)
Mean ± SD	**1.50 ± 0.99**	**1.08 ± 0.91**	**1.62 ± 1.22**	**1.60± 0.84**

Discussion

The results of our study show that with increasing obstructive sleep apnea severity, there is a significant worsening of both anthropometric and polygraphy parameters, along with a statistically significantly higher prevalence of AH and greater overall comorbidity burden in severe OSA. Laboratory cardiometabolic markers do not show statistically significant differences between groups. The mean BMI in the entire sample was 34.71 ± 6.69 kg/m2, indicating that the majority of subjects

belong to the obesity category (BMI ≥ 30). Comparative analysis showed a progressive increase in BMI with OSA severity — from 29.91 kg/m2 in mild to 36.67 kg/m2 in the severe group ($p < 0.001$). The same pattern was observed for neck circumference (40.38 vs. 45.67 cm, $p < 0.001$) and waist circumference (103.79 vs. 121.69 cm, $p < 0.001$). These findings are consistent with the well-established relationship between obesity and OSA [1,2]. Visceral obesity, manifested by increased waist circumference, contributes to upper airway narrowing through fat deposits in the parapharyngeal space [17], while increased neck circumference directly indicates mechanical compression of the airway. Peppard et al. demonstrated that a 10% increase in body weight increases the risk of moderate to severe OSA sixfold [13], which is consistent with our findings. Regarding polygraphy parameters, as expected, ODI was significantly higher in the severe OSA group (50.71 ± 23.34) compared to mild OSA (10.14 ± 7.26, $p < 0.001$), reflecting a greater degree of intermittent hypoxia during sleep. Minimum oxygen saturation progressively decreased with OSA severity — from 84.38% in mild to 68.93% in the severe group ($p < 0.001$). This finding is clinically significant because intermittent hypoxia represents one of the key pathophysiological mechanisms by which OSA contributes to cardiovascular risk [3]. Repeated cycles of hypoxia and reoxygenation trigger oxidative stress, endothelial dysfunction, and systemic inflammation, which are mediators in the development of atherosclerosis, arterial hypertension, and metabolic syndrome [3,5]. Regarding the lipid profile, no statistically significant difference was found between groups of different OSA severity. Total cholesterol ($p = 0.161$), LDL ($p = 0.298$), HDL ($p = 0.063$), and triglycerides ($p = 0.430$) did not differ significantly. Nevertheless, the HDL cholesterol value shows a decreasing trend from mild (1.60 mmol/L) to severe OSA (1.14 mmol/L) with borderline significance ($p = 0.063$), which could reach statistical significance in a larger sample. A significant proportion of subjects were on statin or other lipid-lowering therapy, which may mask differences in the lipid profile. The relationship between OSA and dyslipidemia may be mediated by obesity as a common confounding factor, rather than directly by OSA severity [4]. Trzepizur et al. demonstrated that nocturnal intermittent hypoxemia is independently associated with metabolic dyslipidemia [10], but this association does not necessarily follow the AHI classification. Despite HbA1c of 5.87 ± 0.93%, differences between groups were not statistically significant. Nevertheless, it is known that intermittent hypoxia characteristic of OSA leads to insulin resistance through sympathetic nervous system activation and increased cortisol levels [9], suggesting that a longitudinal study could demonstrate a stronger association. Comorbidity analysis showed that AH was the most common comorbidity in the entire sample (73.5%), witha statistically significant difference between groups ($p = 0.003$) — prevalence increased from 45.8% in mild to 81.7% in severe OSA. This finding is consistent with the known pathophysiological mechanisms by

which OSA contributes to the development of hypertension. Intermittent hypoxia activates the sympathetic nervous system, increases peripheral vascular resistance, and leads to endothelial dysfunction [6,7]. Nieto et al. demonstrated a significant association between sleep-disordered breathing and hypertension in a large population-based study [6], while Pedrosa et al. identified OSA as the most common secondary cause of resistant hypertension [7]. Diabetes mellitus was present in 18.6% of subjects, without a statistically significant difference between groups ($p = 0.311$). Cardiovascular diseases (CVA, arrhythmias, MI, AF, tachycardia) were present in 18.6% of subjects, with higher prevalence in the moderate OSA group (27.6%), but without statistical significance ($p = 0.346$). Particularly noteworthy is the finding that the percentage of patients with at least one comorbidity significantly increases with OSA severity — from 70.8% in mild to 95.0% in the severe group ($p = 0.010$), confirming the clinical significance of severe OSA as a factor of cumulative cardiometabolic risk [16]. The mean number of comorbidities shows an increasing trend with OSA severity — from 1.08 in mild to 1.60 in the severe group — with borderline significance (Kruskal-Wallis $p = 0.063$). Our results are consistent with the review by Drager et al., which demonstrated a strong association between OSA severity and obesity and nocturnal hypoxemia, but a variable relationship with metabolic markers [10]. The study by Marin et al. also confirmed that severe OSA independently increases cardiovascular risk [11], with complex and multiple intermediary mechanisms. Our findings suggest that the relationship between OSA severity and cardiometabolic risk is mediated primarily by obesity and the degree of nocturnal hypoxia, while the direct impact on lipid profile and glycemic control requires further evaluation.

Conclusion

Based on the analysis of 113 patients with obstructive sleep apnea, the following conclusions can be drawn: the majority of subjects had severe OSA — more than half of the subjects (53.1%) had an AHI ≥ 30. OSA severity is strongly associated with obesity. BMI, neck circumference, and waist circumference progressively increase with OSA severity (all $p < 0.001$), confirming obesity as the dominant risk factor. The degree of nocturnal hypoxia significantly worsens with OSA severity. ODI increases from 10.14 in mild to 50.71 in severe OSA, while minimum SpO2 decreases from 84.38% to 68.93% (all $p < 0.001$), indicating progressively greater cardiovascular risk. AH is the most common comorbidity (73.5%), with statistically significantly higher prevalence in severe OSA (81.7%) compared to mild (45.8%, $p = 0.003$). The percentage of patients with at least one comorbidity significantly increases with OSA severity — from 70.8% to 95.0% ($p = 0.010$). Laboratory cardiometabolic markers show no significant differences between groups of different OSA severity.

The association between OSA severity and cardiometabolic comorbidities is mediated primarily through obesity, intermittent hypoxia, and arterial hypertension, while the direct impact on lipid profile and glycemic control requires further evaluation in prospective studies.

References

1. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. Am J Respir Crit Care Med. 2002;165(9):1217-1239. https://doi.org/10.1164/rccm.2109080

2. Benjafield AV, Ayas NT, Eastwood PR et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. Lancet Respir Med.2019;7(8):687-698. https://doi.org/10.1016/S2213-2600(19)30198-5.

3. Lévy P, Kohler M, McNicholas WT et al. Obstructive sleep apnoea syndrome. Nat Rev Dis Primers. 2015;1:15015. https://doi.org/10.1038/nrdp.2015.15

4. Drager LF, Togeiro SM, Polotsky VY et al. Obstructive sleep apnea: a cardiometabolic risk in obesity and the metabolic syndrome. J Am Coll Cardiol. 2013;62(7):569-576. https://doi.org/10.1016/j.jacc.2013.05.045

5. Dempsey JA, Veasey SC, Morgan BJ et al. Pathophysiology of sleep apnea. Physiol Rev. 2010;90(1):47-112. https://doi.org/10.1152/physrev.00043.2008

6. Nieto FJ, Young TB, Lind BK, Shahar E et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. JAMA. 2000;283(14):1829-1836. https://doi.org/10.1001/jama.283.14.1829

7. Pedrosa RP, Drager LF, Gonzaga CC et al. Obstructive sleep apnea: the most common secondary cause of hypertension associated with resistant hypertension. Hypertension. 2011;58(5):811-817. https://doi.org/10.1161/hypertensionaha.111.179788

8. Stojanovic M, Deljanin-Ilić M, Ristic L, Stamenkovic Z et al. The impact of the severity of obstructive sleep apnea-hypopnea syndrome on the quality of life and exercise tolerance in patients with hypertension.Military Medic Pregl. 2026; 83(2): 119–125. https://doi.org/10.3390/medicina61122221

9. Punjabi NM, Shahar E, Redline S et al. Sleep Heart Health Study Investigators. Sleep-disordered breathing, glucose intolerance, and insulin resistance: the Sleep Heart Health Study. Am J Epidemiol. 2004;160(6):521-530. https://doi.org/10.1093/aje/kwh261

10. Trzepizur W, Le Vaillant M, Meslier N et al. Independent association between nocturnal intermittent hypoxemia and metabolic dyslipidemia. Chest. 2013;143(6):1584-1589. https://doi.org/10.1378/chest.12-1652

11. Marin JM, Carrizo SJ, Vicente E et al. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway

pressure: an observational study. Lancet. 2005;365(9464):1046-1053. https://doi.org/10.1016/s0140-6736(05)71141-7

12. Stojanovic M, Deljanin-Ilic M, Ristic L, Stamenkovic Z et al. Challenges in assessing cardiovascular risk in obstructive sleep apnea-hypopnea syndrome - applicability of existing scores. Military Medical Examination. 2025, vol. 82, br. 8, str. 481-489. https://doi.org/10.2298/VSP250422053S

13. Peppard PE, Young T, Palta M et al. Longitudinal study of moderate weight change and sleep-disordered breathing. JAMA. 2000;284(23):3015-3021. https://doi.org/10.1001/jama.284.23.3015

14. Berry RB, Budhiraja R, Gottlieb DJ et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. J Clin Sleep Med. 2012;8(5):597-619. https://doi.org/10.5664/jcsm.2172

15. Collop NA, Anderson WM, Boehlecke B et al. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. J Clin Sleep Med. 2007;3(7):737-747. https://pmc.ncbi.nlm.nih.gov/articles/PMC2556918/

16. Schwab RJ, Pasirstein M, Pierson R et al. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. Am J Respir Crit Care Med. 2003;168(5):522-530. https://doi.org/10.1164/rccm.200208-866oc

17. Bonsignore MR, Baiamonte P, Mazzuca E et al. Obstructive sleep apnea and comorbidities: a dangerous liaison. Multidiscip Respir Med. 2019;14:8. https://doi.org/10.1186/s40248-019-0172-9