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Impact of the severity of obstructive sleep apnea-hypopnea syndrome on the quality of life and exercise tolerance in hypertensive patients

Uticaj težine sindroma opstruktivne apneje-hipopneje u snu na kvalitet života i toleranciju napora kod obolelih od hipertenzije

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Abstract

Background/Aim. Obstructive sleep apnea-hypopnea syndrome (OSAHS) is associated with impaired quality of life (QoL) and reduced exercise tolerance. The aim of the study was to determine whether the severity of OSAHS influences the QoL and exercise tolerance in patients with moderate to severe OSAHS and arterial hypertension. **Methods.** The study included 115 consecutive patients with arterial hypertension and either moderate [40 (34.78%)] or severe [75 (65.22%)] OSAHS. Exercise tolerance was assessed using the exercise stress test, while QoL was evaluated with the Short Form-36 (SF-36) questionnaire. **Results.** The groups under study did not differ significantly in terms of age (54.80 ± 8.91 vs. 53.55 ± 9.53, *p* = 0.494) or sex distribution [females: 11 (27.50%) vs. 13 (17.33%), *p* = 0.201]. A high prevalence of cardiovascular risk factors was observed in the study population. Patients with severe OSAHS had significantly higher body mass index and neck circumference. Obesity was also more frequent among patients with severe OSAHS. In addition, this group demonstrated significantly higher apnea-hypopnea index, desaturation index, and time spent with oxygen saturation below 90%. There were no statistically significant differences in either exercise tolerance or the SF-36 parameters between the groups. Furthermore, no significant correlations were observed between apnea-hypopnea index, exercise tolerance, and QoL parameters. Multivessel coronary artery disease was detected in two asymptomatic patients. **Conclusion.** Patients with OSAHS overall exhibit multiple cardiovascular risk factors and are characterized by reduced QoL and decreased exercise tolerance. No significant correlation was found between OSAHS severity and exercise tolerance and QoL. The detection of multivessel coronary artery disease in asymptomatic patients in the study underscores the clinical significance of screening for coronary heart disease in patients diagnosed with OSAHS.

Keywords:
coronary artery disease; exercise tolerance; hypertension; quality of life; sleep apnea, obstructive; sleep apnea syndromes.

Apstrakt

Uvod/Cilj. Sindrom opstruktivne apneje-hipopneje u snu (*obstructive sleep apnea-hypopnea syndrome* – OSAHS) povezan je sa smanjenim kvalitetom života (*quality of life* – QoL) i smanjenom tolerancijom fizičkog napora. Cilj rada bio je da se utvrdi da li težina OSAHS-a utiče na QoL i toleranciju fizičkog napora kod bolesnika sa umerenim do teškim OSAHS-om i arterijskom hipertenzijom. **Metode.** Studija je obuhvatila 115 konsekutivnih bolesnika sa arterijskom hipertentijom koji su imali srednje tažak [40 (34,78%)] ili težak [75 (65,22%)] OSAHS. Tolerancija fizičkog napora procenjivana je korišćenjem testa fizičkim opterećenjem, dok je QoL procenjivan korišćenjem upitnika *Short-Form 36* (SF-36). **Rezultati.** Ispitivane grupe se nisu statistički značajno razlikovale prema starosti (54,80 ± 8,91 vs. 53,55 ± 9,53, *p* = 0,494) i polu [ženska populacija: 11 (27,50%) vs. 13 (17,33%), *p* = 0,201]. Uočena je visoka prevalencija kardiovaskularnih faktora rizika. Bolesnici sa teškim OSAHS-om imali su značajno višu vrednost indeksa telesne mase i veći obim vrata. Isto tako, kod bolesnika sa teškim OSAHS-om gojaznost je bila zastupljenija. Pored toga, ova grupa bolesnika imala je značajno više vrednosti indeksa apneje-hipopneje, indeksa desaturacije i trajanje perioda u kome je zabeležena sautracija kiseonika ispod 90%. Nije bilo statistički značajne razlike ni u toleranciji fizičkog napora, niti u parametrima upitnika SF-36 između grupa. Takođe, nije utvrđena značajna korelacija između indeksa apneje-

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hipopneje, tolerancije fizičkog napora i parametara QoL. Kod dvoje asimptomatskih bolesnika registrovano je postojanje višesudovne koronarne bolesti. **Zaključak.** Oboleli od OSAHS-a generalno imaju brojne kardiovaskularne faktore rizika, niži QoL i smanjenu toleranciju fizičkog napora. Nije dokazano postojanje značajne korelacije između težine OSAHS-a, tolerancije fizičkog napora i QoL. Otkrivanje višesudovne koronarne bolesti kod asimptomatskih bolesnika u istraživanju naglašava klinički značaj skrininga na koronarnu bolest srca kod obolelih od OSAHS-a.

Ključne reči:
koronarna bolest; vežbanje, tolerancija; hipertenzija; kvalitet života; apneja u snu, opstruktivna; apneja, spavanje poremećaji, sindromi.

Introduction

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a breathing disorder characterized by episodes of partial (hypopnea) or complete (apnea) airway collapse during sleep [1]. It represents the most prevalent sleep-breathing disorder [2]. The diagnosis of OSAHS is made by a sleep study, and the severity of OSAHS is usually estimated by the apnea-hypopnea index (AHI). AHI is defined as apneic or hypopnea events that occur during one hour of sleep [3].

The main risk factors for OSAHS are obesity, male gender, age (≥ 50 years), alcohol abuse, smoking, heredity, and craniofacial abnormalities [4]. On the other hand, OSAHS represents an independent risk factor for many cardiovascular diseases (CVDs). Changes in intrathoracic pressure, intermittent episodes of hypoxemia or hypercapnia, and repeated episodes of arousal seen in patients with OSAHS trigger increased sympathetic nervous system activation, oxidative stress, and systemic inflammation [5]. These pathophysiological mechanisms contribute to the increased risk of atrial fibrillation, coronary artery disease (CAD), stroke, heart failure, ventricular tachycardia, and sudden cardiac death [5, 6].

The most common CVD in OSAHS patients is arterial hypertension (HTA) [7]. There appears to be a linear relationship between the severity of OSAHS and the risk of HTA. Furthermore, this relationship seems bidirectional as 44% of HTA patients suffer from moderate to severe OSAHS, and the prevalence even increases in resistant HTA [5, 8]. This is why relevant guidelines suggest screening for OSAHS in patients with resistant or refractory HTA [9]. Likewise, specific HTA phenotypes, such as masked or nocturnal hypertension, are very prevalent in OSAHS patients [10, 11]. In addition, these patients often have an increased blood pressure (BP) variability, an exaggerated morning surge [10, 11], and an impaired circadian rhythm [10–12].

The quality of life (QoL) in patients with OSAHS is significantly impaired. Excessive daytime sleepiness, decreased concentration and memory, irritability, and decreased energy are the reasons why anxiety, depression, and cognitive impairment are very prevalent in OSAHS [13]. Furthermore, patients with OSAHS have lower exercise tolerance compared to healthy individuals, and it is not just because of physical limitations (obesity, decreased energy, etc.), but also due to psychological motivation [14].

The aim of the study was to determine whether the severity of OSAHS influences the QoL and exercise tolerance in patients with moderate to severe OSAHS and HTA.

Methods

Out of 410 consecutive patients with OSAHS, 115 hypertensive patients were included in this prospective study due to strict exclusion criteria. Among them, 40 (34.78%) patients had moderate OSAHS [AHI 15–29 episodes *per* hour (15–29/hr)], while 75 (65.22%) patients had severe OSAHS (AHI ≥ 30/hr). The diagnosis of OSAHS was made based on full-night respiratory polygraphy (RPG) using the Alice NightOne device from Philips Respironics (Eindhoven, Netherlands). Testing was performed at the sleep laboratory of the Clinic for Lung Diseases, University Clinical Center Niš, Niš, Serbia, during the patient's habitual sleep time, following the criteria from the American Academy of Sleep Medicine (AASM) Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea [15]. The parameters of RPG used in the study were AHI, oxygen desaturation index, total sleep time spent with oxygen saturation below 90%, and minimal, average, and maximal oxygen saturation, graded according to AASM recommendations and criteria.

Patients with known CAD, heart failure, severe valvular disease or artificial valve, chronic kidney disease (defined as estimated glomerular rate below 60 mL/min/1.73m^2), patients younger than 40 years or older than 80 years, and patients with impaired physical or mental condition were not included in the study.

After finishing the sleep study, all patients were hospitalized at the Department of Cardiovascular Diseases at the Institute for Treatment and Rehabilitation "Niška Banja", Niš, where clinical assessment and anthropometric measurements were performed. Data on cardiovascular risk (CVR) factors, including HTA, dyslipidemia, stress, smoking, diabetes mellitus (DM), obesity, physical inactivity, and family history of CVD, were also collected. During hospitalization, laboratory assessment, Short-Form 36 (SF-36) Health Status Survey, and exercise stress test (EST) were performed. The ESTs were done on the treadmill (3017 Full Vision Drive, Newton, Kansas, USA) according to the Bruce protocol. Seven patients could not perform EST due to extreme obesity (weight > 130 kg). Tests were limited by submaximal heart rate, symptoms, ischemic changes on electrocardiogram (horizontal or downsloping ST segment depression ≥ 1 mm), complex ventricular arrhythmia (couplets of ventricular premature beats or ventricular tachycardia), hypertensive reaction defined as a sudden increase of systolic BP (values ≥ 220 mmHg), or decrease in systolic BP (values >10 mmHg), or patient request to stop.

Psychological dimensions and the QoL were assessed by the validated SF-36 questionnaire, which has been culturally adapted and validated for the Serbian-speaking population [16]. All patients were divided into two groups: one with moderate OSAHS (AHI 15–29/h) and one with severe OSAHS (AHI ≥ 30/h). All data were analyzed based on the severity of OSAHS.

The subjects' written consent was obtained, according to the Declaration of Helsinki. The study was approved by the Ethics Committee of the Institute for Treatment and Rehabilitation "Niška Banja", Niš (No. 3560/1, from March 29, 2023), and the Ethics Committee of the Faculty of Medicine, University of Niš, Niš (No. 12-1760-1/2-4, from February 20, 2024).

Statistical analysis

Data were analyzed using the SPSS software, version 20.0 (SPSS Inc., Chicago, IL, USA). Categorical data were expressed as frequencies and percentages, while quantitative data were presented as means ± standard deviations. Data distribution was tested using the Kolmogorov-Smirnov test. Means of normally distributed data were compared using the Student's *t*-test, while the Mann-Whitney *U* test was used for data whose distribution deviates significantly from normal distribution. The Chi-square test was used to compare frequencies. In correlation analysis, Pearson's correlation was used for normally distributed variables, while for data whose distribution deviates significantly from normal distribution, Spearman's rank correlation was used. Statistical significance was accepted for $p < 0.05$.

Results

The study included 115 patients (age 53.98 ± 9.3 years) with HTA and moderate or severe OSAHS. Patients were divided into two groups according to the severity of OSAHS: 40 (34.78%) patients with moderate OSAHS and 75 (65.22%) patients with severe OSAHS. All parameters were compared between the groups.

All sleep study parameters were significantly better in patients with moderate OSAHS than in those with severe OSAHS (Table 1). Patients with severe OSAHS had significantly higher values of AHI (51.84 ± 16.97 vs. 23.08 ± 4.63, $p < 0.001$), oxygen desaturation index (53.15 ± 22.17 vs. 24.98 ± 10.22, $p < 0.001$), and total sleep time spent with oxygen saturation below 90% (36.38 ± 27.09 vs. 18.56 ± 27.86, $p < 0.001$) compared to moderate OSAHS. On the other hand, patients with moderate OSAHS had significantly higher values of minimum (92.72 ± 11.11 vs. 67.41 ± 13.28, $p < 0.001$) and average oxygen saturation (92.3 ± 3.29 vs. 89.56 ± 4.18, $p < 0.001$) compared to those with severe OSAHS.

We then compared CVR factors and anthropometric parameters between the groups (Table 2). Patients with severe OSAHS had significantly higher neck circumference (46.23 ± 3.58 vs. 43.97 ± 4.20, $p = 0.004$) and body mass index (BMI) values (36.79 ± 5.72 vs. 34.25 ± 5.88,

Table 1

Sleep study parameters

Parameters	OSAHS		Total	*Z*	*p*-value
	moderate	severe			
Apnea-hypopnea index *per* hour	23.08 ± 4.63	51.84 ± 16.97	41.83 ± 19.59	-8.808	< 0.001
Oxygen desaturation index *per* hour (%)	24.98 ± 10.22	53.15 ± 22.17	43.35 ± 23.16	-6.404	< 0.001
Time spent with oxygen saturation below 90% (%)	18.56 ± 27.86	36.38 ± 27.09	30.32 ± 28.51	-3.659	< 0.001
Minimal oxygen saturation (%)	92.72 ± 11.11	67.41 ± 13.28	76.22 ± 66.96	-3.770	< 0.001
Average oxygen saturation (%)	92.3 ± 3.29	89.56 ± 4.18	90.48 ± 4.1	-3.951	< 0.001

OSAHS – obstructive sleep apnea-hypopnea syndrome. All values are given as mean ± standard deviation.

Table 2

Cardiovascular risk factors and anthropometric measurements

Parameters	OSAHS		Total	$t/Z/\chi^2$	*p*-value
	moderate	severe			
Gender, female	11 (27.50)	13 (17.33)	24 (20.87)	1.633[a]	0.201
Smoking	23 (57.50)	39 (52.00)	62 (53.91)	0.318[a]	0.573
Physical inactivity	14 (35.00)	35 (46.67)	49 (42.61)	1.452[a]	0.228
Obesity	31 (77.50)	71 (94.67)	102 (88.70)	7.667[a]	**0.006**
Stress	13 (32.50)	15 (20.00)	28 (24.35)	2.213[a]	0.137
Diabetes mellitus	6 (15.00)	25 (33.33)	31 (26.96)	4.453[a]	0.035
Heredity	25 (62.50)	42 (56.00)	67 (58.26)	0.453[a]	0.501
Dyslipidaemia	30 (75.00)	50 (66.67)	80 (69.57)	0.856[a]	0.355
Age, years	54.8 ± 8.91	53.55 ± 9.53	53.98 ± 9.30	0.687	0.494
Waist circumference (cm)	118.33 ± 15.11	123.42 ± 12.36	121.65 ± 13.53	-1.919	0.058
Neck circumference (cm)	43.97 ± 4.20	46.23 ± 3.58	45.46 ± 3.94	-2.970	**0.004**
Body mass index (kg/m^2)	34.25 ± 5.88	36.79 ± 5.72	35.90 ± 5.88	-2.242	**0.027**
Number of cardiovascular risk factors	4.60 ± 1.55	4.75 ± 1.23	4.70 ± 1.35	-0.638	0.524

OSAHS – obstructive sleep apnea-hypopnea syndrome. All values are given as numbers (percentages) or mean ± standard deviation. The bold values indicate a significance level of *p* < 0.05.
***Note*: [a] Student's *t*-test was used.**

Table 3

Laboratory measurements

Parameters	OSAHS		Total	Z	p-value
	moderate	severe			
Cholesterol, mmol/L	5.27 ± 1.08	5.08 ± 1.17	5.15 ± 1.14	-0.781	0.435
LDL, mmol/L	2.96 ± 1.12	3.07 ± 1.06	3.03 ± 1.08	-0.209	0.835
HDL, mmol/L	1.18 ± 0.31	1.07 ± 0.27	1.11 ± 0.29	-1.647	0.099
Triglyceride, mmol/L	2.33 ± 1.44	2.03 ± 0.94	2.13 ± 1.14	-0.523	0.601
Glucose, mmol/L	5.73 ± 1.20	5.84 ± 0.81	5.8 ± 0.96	-1.204	0.229
AST, U/L	21.65 ± 6.92	21.35 ± 8.40	21.45 ± 7.89	-0.650	0.516
ALT, U/L	29.25 ± 14.9	29.29 ± 15.45	29.28 ± 15.19	-0.015	0.988
Creatinine, µmol/L	91.30 ± 15.99	93.97 ± 15.93	93.04 ± 15.93	-0.341	0.733
Urea, mmol/L	5.08 ± 1.28	5.40 ± 1.43	5.29 ± 1.38	-1.087	0.277
Uric acid	354.88 ± 77.71	379.31 ± 85.60	370.81 ± 83.42	-1.577	0.115
eGFR	114.05 ± 35.16	129.53 ± 3 2.54	124.15 ± 34.13	-2.646	**0.008**
Modified eGFR*	91.64 ± 25.05	100.19 ± 22.75	97.24 ± 23.81	-1.969	**0.049**
Sedimentation	20.55 ± 18.92	19.56 ± 13.88	19.90 ± 15.74	-0.062	0.951
Leukocyte	7.16 ± 1.65	7.46 ± 1.58	7.36 ± 1.60	-0.722	0.470
Erythrocyte	4.85 ± 0.45	4.87 ± 0.44	4.86 ± 0.44	-0.279	0.780
Hematocrit	0.43 ± 0.04	0.43 ± 0.03	0.43 ± 0.04	-0.553	0.580
Hemoglobin	143.13 ± 14.95	144.45 ± 12.40	143.99 ± 13.29	-0.264	0.791
Thrombocyte	262.30 ± 52.48	254.91 ± 55.20	257.48 ± 54.15	-0.810	0.418

LDL – low-density lipoprotein; HDL – high-density lipoprotein; AST – aspartate aminotransaminase; ALT – alanine aminotransaminase; eGFR – estimated glomerular filtration rate. For other abbreviations, see Table 1.
All values are given as mean ± standard deviation. The bold values indicate a significance level of $p < 0.05$.
***Note*: * used for an overweight patient.**

Table 4

Exercise stress test

Parameters	OSAHS		Total	χ^2/Z	p-value
	moderate	severe			
Submaximal heart rate achieved	26 (68.42)	59 (84.29)	85 (78.70)	3.698[a]	0.054
Ischemia	3 (7.50)	3 (4.00)	6 (5.22)	0.646[a]	0.421
Arrhythmias	2 (5.26)	7 (10.00)	9 (8.33)	0.723[a]	0.395
Level	2.71 ± 1.14	2.61 ± 1.04	2.65 ± 1.07	-0.259	0.796
Duration (min)	6.53 ± 3.25	6.14 ± 2.95	6.28 ± 3.05	-0.384	0.701
Double product before test	9,541.05 ± 1,718.26	9,876.86 ± 1,877.82	9,758.7 ± 1,822.31	-1.017	0.309
Double product after test	21,409.5 ± 5,804.29	22, 177.73 ± 6,896.41	21,910.52 ± 6,521.74	-1.398	0.162

OSAHS – obstructive sleep apnea-hypopnea syndrome; min – minutes.
All values are given as numbers (percentages) or mean ± standard deviation.
***Note*: [a] Chi-square test was used.**

$p = 0.027$) compared to those with moderate OSAHS. In addition, a significantly higher proportion of patients with severe OSAHS suffered from obesity (94.67% vs. 77.5%, $p = 0.006$).

No significant differences were observed in the investigated laboratory parameters between the groups except for creatinine clearance (129.53 ± 32.54 vs. 114.05 ± 35.16, $p = 0.008$) and creatinine clearance adjusted for overweight patients (100.19 ± 22.75 vs. 91.64 ± 25.05, $p = 0.049$). Both parameters were statistically higher in patients with severe OSAHS (Table 3).

The ESTs were performed in 108 patients (38 with moderate, and 70 with severe OSAHS). The differences in exercise tolerance among the groups were shown in Table 4. There were no significant differences in the examined parameters among the groups.

A total of 113 (39 with moderate and 74 with severe OSAHS) patients completed the SF-36 questionnaire. Two patients did not complete the questionnaire, and their data were not included in the study. The physical component summary (PCS) was higher in the moderate OSAHS group, while the mental component summary (MCS) was higher in the severe OSAHS group (Table 5), but without statistical significance.

After excluding DM, obesity, and stress, a correlation analysis was performed between AHI and EST level, PCS, and MCS (Table 6). No significant correlation was observed between AHI and EST level, PCS, or MCS.

Table 5

The quality of life assessed by the SF-36 questionnaire

Parameters	OSAHS		Total	Z	p-value
	moderate	severe			
Physical functioning	51.88 ± 26.33	53.69 ± 25.90	53.05 ± 25.95	-0.494	0.621
Limitations due to physical health	44.23 ± 43.48	45.61 ± 40.83	45.13 ± 41.57	-0.339	0.735
Limitations due to emotional problems	52.99 ± 43.07	60.81 ± 39.16	58.11 ± 40.53	-0.870	0.384
Energy/fatigue	49.10 ± 19.60	51.15 ± 19.65	50.44 ± 19.57	-0.585	0.559
Emotional well-being	66.26 ± 18.28	67.46 ± 18.11	67.04 ± 18.10	-0.348	0.728
Social functioning	66.35 ± 27.23	66.05 ± 25.04	66.15 ± 25.70	-0.150	0.881
Pain	52.31 ± 29.28	55.47 ± 25.86	54.38 ± 27.00	-0.647	0.518
General health	50.00 ± 18.25	48.92 ± 17.79	49.29 ± 17.88	-0.219	0.827
Health change	40.38 ± 24.07	49.32 ± 28.66	46.24 ± 27.39	-1.519	0.129
Physical component summary	65.59 ± 13.29	63.77 ± 13.69	64.40 ± 13.52	-0.531	0.595
Mental component summary	76.83 ± 16.16	79.16 ± 14.55	78.35 ± 15.1	-0.901	0.367

SF-36 – Short-Form 36. OSAHS – obstructive sleep apnea-hypopnea syndrome.
All values are given as mean ± standard deviation.

Table 6

Correlation between AHI and EST, MCS, and PCS

Index		EST level	PCS	MCS
AHI	*R*	-0.0120	0.086	0.098
	p	0.902	0.387	0.324

AHI – apnea-hypopnea index; EST – exercise stress test; PCS – physical component summary; MCS – mental component summary.

Discussion

The study evaluated 115 patients with moderate or severe OSAHS and HTA, focusing on disease severity, CVR profile, QoL, and exercise tolerance. As expected, patients with severe OSAHS had higher BMI and neck circumference, in line with previous research linking obesity and anthropometric markers to OSAHS severity [17–21]. A high prevalence of CVR factors was observed, underscoring the substantial cardiovascular burden in this population. However, no significant differences were detected between moderate and severe OSAHS in QoL measures or exercise tolerance. These findings suggest that disease severity may not reliably predict exercise tolerance or perceived health status. Finally, detection of ischemic changes and confirmed CAD in some patients further highlights the necessity of cardiovascular screening in this population.

The study population comprised predominantly males [91 (79.13%)], with only 24 (20.87%) female participants. Our results are in concordance with the results of previous trial [22]. The higher prevalence of OSAHS in males can be explained by several factors. Women less frequently report typical OSAHS symptoms like snoring or witnessed apneas [23]. On the other hand, the male respiratory tract is longer and more prone to collapse compared to the female respiratory tract [24]. Nevertheless, the most important explanation for sex-related differences in OSAHS prevalence is the protective role of progesterone and estrogen, which enhance upper airway dilator muscle function and reduce the likelihood of developing OSAHS [25]. As anticipated, all assessed sleep study parameters showed significantly better values in moderate OSAHS than in severe OSAHS.

Patients with severe OSAHS had significantly higher neck circumference and BMI values, and a greater proportion of them were obese compared to those with moderate OSAHS. This is in concordance with previous research [17–21]. The obesity and high BMI values significantly increase the chance of OSAHS development [17], and a positive correlation between BMI and OSAHS severity has been found—the higher the BMI, the higher the AHI [18]. Moreover, it has been shown that neck circumference can predict both the presence and severity of OSAHS in snoring patients [19], and that neck circumference positively correlates with OSAHS severity [20]. Some evidence suggests that this correlation is even stronger than that observed between OSAHS and general obesity [21].

A high prevalence of CVR factors was noticed in OSAHS patients, including obesity (88.70%), dyslipidaemia (69.57%), smoking (53.91%), heredity (58.26%), physical inactivity (42.61%), and DM (26.96%). These findings align with previous research, which highlights high CVR in patients with OSAHS [26, 27]. However, standard CVR assessment tools may underestimate or inadequately represent CVR associated with OSAHS [28].

Several factors contribute to impaired QoL in patients with OSAHS [29, 30]. Depression [29], poor sleep quality [30], comorbidities, excessive daytime sleepiness [30], and cognitive dysfunction [31] are frequently observed in this population and are associated with reduced QoL. Surprisingly, the severity of OSAHS does not influence the QoL [32]. Our study confirmed these findings as all investigated parameters obtained by the SF-36 questionnaire showed no significant differences between the groups. Furthermore, there were no statistically significant differences in PCS and MCS between the groups. These findings may be attributed to several factors, including potential adaptation mechanisms in patients with more severe OSAHS, the subjective nature of QoL assessments, and the limited sample size of the study.

Patients with OSAHS have lower levels of physical activity compared to individuals without OSAHS, and regular physical activity has been shown to reduce the risk of OSAHS development [33]. Obesity, daytime sleepiness, fatigue, and decreased energy contribute to reduced exercise tolerance in OSAHS patients. It might be hypothesized that exercise tolerance decreases with increasing OSAHS severity. Exercise tolerance was assessed using EST rather than cardiopulmonary exercise testing (CPET), the latter being a more sensitive method for evaluating exercise tolerance in patients with OSAHS [34]. In the present study, there were no statistically significant differences in exercise tolerance on EST between the investigated groups. Moreover, the anticipated negative correlation between AHI and exercise tolerance was not observed in our analysis. These findings are consistent with previous studies reporting weak or absent associations between AHI and objective measures of exercise tolerance, particularly in men [35], suggesting that OSAHS severity is not always a reliable predictor of exercise tolerance [36]. Finally, it should be noted that we used EST rather than CPET, which is considered a more sensitive method for assessing exercise tolerance in patients with OSAHS [37].

Six (5.22%) patients of the 109 who underwent EST exhibited ischemic changes on the electrocardiogram. Three of these patients were referred for stress echocardiography, which revealed no exercise-induced wall motion abnormalities. Conversely, three patients underwent coronary angiography, with one demonstrating multivessel CAD. Moreover, one patient was directly referred for coronary angiography due to high CVR. Multivessel CAD was also identified in this patient. These findings underscore the importance of screening for CAD in patients with OSAHS [38].

The RPG was performed instead of respiratory polysomnography, which remains the gold standard for OSAHS diagnosis. This decision was necessitated by the lack of readily available respiratory polysomnography facilities within our medical center. Although it has certain limitations, RPG remains a valuable and widely used tool for assessing respiratory events during sleep, particularly in resource-constrained settings [39]. In addition, exercise tolerance was assessed using EST instead of CPET, although CPET represents a more sensitive and comprehensive method for evaluating exercise tolerance in patients with OSAHS. The predominance of male (79.13%) participants reduces the generalizability of our results to female patients.

Conclusion

Patients with obstructive sleep apnea-hypopnea syndrome overall exhibit multiple cardiovascular risk factors and are characterized by reduced quality of life and decreased exercise tolerance. Nevertheless, in our sample, increasing severity of obstructive sleep apnea-hypopnea syndrome did not correspond to significant differences in quality-of-life domains or exercise tolerance. Importantly, we detected multivessel coronary artery disease in asymptomatic patients, which underscores the clinical importance of coronary artery disease screening in patients diagnosed with obstructive sleep apnea-hypopnea syndrome.

Conflict of interest

The authors declare no conflict of interest.

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