



# Prevalence of target organ damage in hypertensive patients with coexisting obstructive sleep apnea

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## Abstract

**Introduction and Objective.** Obstructive sleep apnea (OSA) is associated with cardiovascular risk and higher mortality. Assessment of subclinical organ lesions is significant for prevention of clinically manifested complications which might result in death. The aim of the study was to assess the prevalence of subclinical target organ damage in hypertensive patients with OSA, and to establish whether these lesions are dependent on the intensity of sleep-disordered breathing.

**Materials and method.** The study covered 67 patients with OSA diagnosed on the basis of polygraphy. Depending on the apnea/hypopnea index (AHI), the patients were divided into two groups: G1 (n=32; AHI=5–30) and G2 (n=35; AHI>30). The control group C consisted of 31 hypertensive subjects with OSA risk estimated as low based on the Berlin Questionnaire (BQ). Each patient had the following parameters measured: intima-media thickness (IMT), ankle-brachial index (ABI), left ventricular mass index (LVMI), and estimated glomerular filtration rate (eGFR).

**Results.** The patients with severe OSA had significantly higher LVMI compared to the control group (130.99±44.6 g/m<sup>2</sup> versus 106.61±27.86 g/m<sup>2</sup>; p=0.0332). The G2 group had higher eGFR than C (104.7±17.96 ml/min/1.73m<sup>2</sup>, 88.85±17.68 ml/min/1.73m<sup>2</sup>; p=0.0058). Similar results of eGFR were observed between G1 and C (104.35±21.06 ml/min/1.73m<sup>2</sup>, 88.85±17.68 ml/min/1.73m<sup>2</sup>; p=0.0081). G1 and G2 did not differ significantly in terms of eGFR. The other measured parameters, such as IMT and ABI, did not differ significantly between OSA and controls.

**Conclusions.** Patients with severe OSA demonstrate signs of left ventricular hypertrophy, while early atherosclerotic lesions (ABI and IMT) were not more intense than in the control group. High eGFR levels may indicate hyperfiltration, which does not correlate with OSA intensity level.

## Key words

obstructive sleep apnea, systemic hypertension, target organ damage

## INTRODUCTION

Obstructive sleep apnea (OSA) is a chronic disorder of breathing during sleeping, caused by recurrent episodes of narrowing of the airways, which leads to hypopnea or apnea and numerous organ complications [1]. Acute and chronic cardiovascular diseases are the main causes of increased mortality in OSA patients [2]. The cardiovascular consequences of OSA include primarily arterial hypertension, arrhythmias, ischemic heart disease and congestive heart failure [3]. OSA may also be a potential risk factor for the development of Chronic Kidney Disease (CKD) [4].

The main goal of preventive medicine is to detect subclinical organ lesions as early as possible, which is reflected in the European guidelines. Due to the frequent coexistence of OSA and arterial hypertension, it is interesting to note that OSA *per se* does increase the risk of target organ damage (TOD) in the population of hypertensive OSA patients

compared to hypertensive subjects without sleep-disorder breathing. Common, easily detected markers of early damage include left ventricular hypertrophy, as expressed by the left ventricular mass index (LVMI), intima-media thickness (IMT) of the common carotid artery, ankle-brachial index (ABI) or eGFR [5].

## OBJECTIVE

The aim of the study is to assess the prevalence of subclinical target organ damage in hypertensive patients with coexisting obstructive sleep apnea.

## MATERIALS AND METHOD

**Materials.** The study included 98 patients, aged 23–70 years, with effectively treated systemic hypertension, hospitalized in the Department of Internal Diseases. The criteria for including patients in the study group was the clinical suspicion of OSA based on medical history, the

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Berlin Questionnaire (BQ) and the Epworth Sleepiness Scale (ESS). The exclusion criteria were: lack of the patient's written consent to participate in the study, mental illness, acute infectious diseases, acute cardiovascular diseases (myocardial infarction, stroke, decompensated heart failure), state after coronary artery bypass graft, blood pressure > 140/90 mmHg, presence of leg ulcers. Therefore, 76 patients with a high risk of apnea in BQ then diagnosed using the abbreviated version of polysomnography – polygraphy (PG). The following parameters were recorded during the study: air flow through the upper respiratory tract (using a nasal cannula with a pressure sensor), respiratory movements of the chest and abdomen, arterial blood oxygen saturation (using a pulse oximeter) and body position (Fig. 1). The diagnosis of OSA was based on the American Sleep Disorders Association recommendations [6]. Sleep recording time was 6 hours, according to the recommendations [6]. Accordingly, OSA was diagnosed when AHI (Apnea Hypopnea Index) was  $\geq 5$  and the presence of clinical symptoms confirmed (history,  $\geq 11$  points on the ESS scale) [6]. Based on the PG result, 9 patients who were not confirmed with OSA (AHI < 5) were excluded from further analysis. OSA was diagnosed in the remaining 67 patients. The patients were next divided into 2 groups: group G1 with mild and moderate severity of OSA (AHI = 5–30, n = 32) and group G2 with severe OSA (AHI > 30, n = 35). Both groups were patients with treated, well-controlled blood pressure, and most of patients were treated with at least 2 hypotensive agents. The control group C (n = 31) also consisted of patients with well-controlled BP ( $\leq 140/90$  mmHg), low level of daytime somnolence ( $\leq 10$  points in ESS) and low risk of sleep apnea in BQ.

**Methods. Echocardiography.** Transthoracic echocardiography (TTE) was performed in each test group (G1 and G2) and control group (C) to assess left ventricular mass (LVM). Echocardiography was performed with a Vivid 4 ultrasonograph. M-mode echocardiography was used to measure the left ventricular end-diastolic (LVEDD) and end-systolic (left ventricular end-systolic dimension (LVESD) of the left ventricle, interventricular septal thickness at end-diastole (IVSd) and left ventricular posterior wall thickness at end-diastole; LVPWd). Left ventricular muscle mass was calculated using the formula proposed by the American

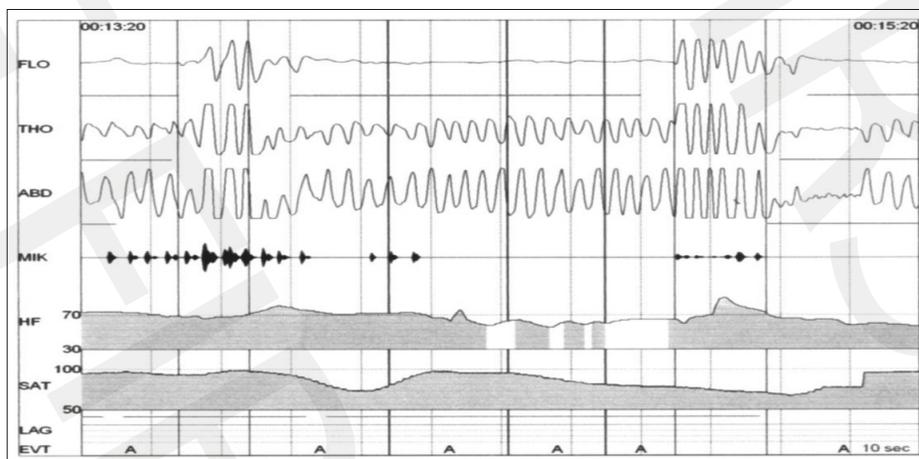
Society of Echocardiography, modified by Devereaux [7]. The body surface area (BSA) was calculated from the Mosteller formula:  $BSA [m^2] = 0.01666667 \times (\text{height in cm})^{0.5} \times (\text{body weight in kg})^{0.5}$ . Left ventricular mass index (LVMI) was calculated using formula:  $LVMI [g/m^2] = LVM [g] / BSA [m^2]$ .

Based on the recommendations of the European Society of Cardiology, the European Society of Hypertension and the Polish Society of Hypertension, left ventricular hypertrophy was diagnosed when the LVMI value was  $> 115 g/m^2$  BSA in men and  $> 95 g/m^2$  BSA in women [8].

**Carotid artery ultrasonography.** All carotid examinations were performed using a GE Vivid 4. The intima-media thickness (IMT) was assessed for both the left and right common carotid arteries, 1–2 cm below the bifurcation. IMT was calculated as the average of 12 measurements (excluding plaque sites) – 6 measurements on the right and 6 measurements on the left. The presence of atherosclerotic plaque was defined as the presence of a focal strong thickening of the arterial wall with an IMT value  $\geq 1.5$  mm.

**Assessment of the Ankle-Brachial Index.** Measurement of the ankle-brachial index (ABI) was determined using a mercury sphygmomanometer and continuous wave Doppler. In order to obtain greater accuracy of the measurements and better interpretation of the results, measurements were taken on all 4 limbs. As standard, 2 pressure measurements were taken on each limb, and the higher value of the measured systolic pressure was chosen to calculate the ABI. The ABI index was calculated separately for the right side limbs ( $ABI_R$ ) and for the left side limbs ( $ABI_L$ ).

**Estimation of Glomerular Filtration Rate (eGFR).** The concentration of creatinine was determined by using Roche COBAS 6000 and COBAS INTEGRA 400 analyzers. The reference values for creatinine concentration were: 0.5–0.9 mg/dL for women and 0.7–1.2 mg/dL for men. Using the data on the patient's age, gender and race, as well as the determined creatinine concentration, the eGFR was calculated according to the abbreviated MDRD formula (Modification of Diet in Renal Diseases) [9].



**Figure 1.** An episode of obstructive sleep apnea with significant decrease in blood saturation. Explanations: visible loss of airflow in the airways (FLO), with preserved respiratory movements of the chest (THO) and abdomen (ABD), and large decrease in blood saturation (SAT).

Source. Own materials at the Department of Internal Diseases, Medical University of Lublin

**Statistical analysis.** Statistical analysis was performed using the PQStat statistical package, version 1.6.4.122. The analyzed data are presented on 2 measurement scales: nominal and interval. The data from the nominal scale was analyzed with the chi-square dependency test and the Fisher's exact test. The analyzed parameters from the interval scale are presented in the form of the mean value, median value, standard deviation, minimum and maximum value. Data were analyzed for normal distribution using the Kolmogorov-Smirnov test. Variables characterized by a normal distribution were analyzed with parametric tests, in other cases non-parametric tests were used. One-way analysis of variance (ANOVA) was used to compare the 3 study groups, after confirming the homogeneity of variance with the Levene test and the Brown-Forsythe test, in the remaining cases the groups were compared with the Kruskal-Wallis test. When there were significant differences between the groups, *post hoc* analysis was performed using the Tukey's test or Dunn Bonferroni's test.

## RESULTS

The polygraphy was performed on 76 patients hospitalized in the Department of Internal Diseases with suspected SDB (Sleep Disordered Breathing). On the basis of AHI values  $\geq 5$  and the presence of symptoms, breathing disorders during sleep were diagnosed in 67 patients. Central-type apnea was not observed in patients with SDB during polygraph examination; therefore, all patients were diagnosed with OSA. The majority of patients with OSA were men who constituted 81% of patients (54 people), while women constituted 19% of the study group (13 people). The mean age of the patients was  $49 \pm 11.9$  years. Minimum age in the study group – 23 years, maximum age – 70 years. The mean AHI value was  $35.7 \pm 24.9$ . The highest AHI value recorded was 85.5. Based on the value of the AHI index, 23 people (34%) were diagnosed with mild OSA (AHI = 5–15), in 9 (14%) moderate (AHI = 16–30), and severe (AHI > 30) in 35 people who accounted for over half of the study group (52%).

**General characteristics of the studied groups.** For further analysis, the patients were divided into 2 groups of similar size. Group G1 consisted of 32 patients with mild or moderate disease severity (AHI = 5–30), while group G2 consisted of 35 patients with severe respiratory disorders (AHI > 30). The control group (C) consisted of 31 patients with a low risk of developing OSA in BQ and no clinical symptoms suggesting OSA (ESS  $\leq 10$  points). The characteristics of the studied groups (G1 and G2) and group C are presented in Table 1.

### Detailed group analysis in terms of parameters tested.

The lowest LVMI values were recorded in group C and the highest in group G2. The results in the G1 group did not differ significantly ( $p > 0.05$ ) from the results in the other 2 groups. *Post hoc* analysis with Tukey's test showed that significant differences were found between the control group and the G2 group ( $p = 0.0332$ ) (Tab. 2). There were no statistically significant differences in the values of IMT,  $ABI_R$  and  $ABI_L$  between the groups. The lowest values of eGFR were observed in the control group, while higher in both OSA groups. Highly statistically significant differences in Tukey's test were observed for group G1 compared to group C ( $p = 0.0081$ ) and

**Table 1.** Characteristics of the studied groups in terms of anthropometric parameters and laboratory test results

	C (n = 31)	G1 (n = 32)	G2 (n = 35)	P value
Gender – Female	6	10	3	0.0639
Gender – Male	25	22	32	
Age [years]	$53.13 \pm 10.13$	$50.31 \pm 11.54$	$47.49 \pm 12.32$	0.1392
BMI [kg/m <sup>2</sup> ]	$30.05 \pm 6.15$	$33.47 \pm 6.48$	$33.08 \pm 4.66$	0.0412
Creatinine [mg/dl]	$0.93 \pm 0.16$	$0.79 \pm 0.15$	$0.84 \pm 0.15$	0.0037
eGFR [ml/min/1.73m <sup>2</sup> ]	$88.85 \pm 17.68$	$104.35 \pm 21.06$	$104.70 \pm 17.96$	0.0028
$ABI_R$	$1.14 \pm 0.18$	$1.19 \pm 0.14$	$1.19 \pm 0.14$	0.4181
$ABI_L$	$1.15 \pm 0.10$	$1.15 \pm 0.16$	$1.17 \pm 0.14$	0.8512
IMT [mm]	$0.88 \pm 0.30$	$1.01 \pm 0.19$	$0.97 \pm 0.25$	0.1332
LVMI [g/m <sup>2</sup> ]	$106.61 \pm 27.86$	$117.69 \pm 41.09$	$130.99 \pm 44.60$	0.0426

**Table 2.** LVMI values in studied groups

Descriptive statistics	C (n = 31)	G1 (n = 32)	G2 (n = 35)
Mean [g/m <sup>2</sup> ]	106.61	117.69	130.99
Median	103	108.24	118.26
Standard deviation	27.86	41.09	44.60
Minimum	56.56	70.61	62.93
Maximum	169.48	274.80	259.14
ANOVA	F Statistics	3.2640	
	p value	0.0426	
Post-hoc (Tukey HSD)	C	0.4968	0.0332
	G1	0.4968	0.3448
	G2	0.0332	0.3448

**Table 3.** Creatinine levels in studied groups

Descriptive statistics	C (n = 31)	G1 (n = 32)	G2 (n = 35)
Mean [mg/dL]	0.93	0.79	0.84
Median	0.90	0.79	0.82
Standard deviation	0.16	0.15	0.15
Minimum	0.65	0.48	0.57
Maximum	1.3	1.21	1.35
ANOVA	F statistics	5.9894	
	P value	0.0037	
Post-hoc (Tukey HSD)	C	0.0026	0.0773
	G1	0.0026	0.3682
	G2	0.0773	0.3682

for group G2 also compared to the control group ( $p = 0.0058$ ). The eGFR values in both study groups (G1 and G2) did not differ significantly (in Tukey's test,  $p = 0.9972$ ) (Tab. 3).

## DISCUSSION

The aim of this study was to assess the impact of OSA on the development of subclinical target organ damage in hypertensive OSA patients. On the basis of the conducted analyzes, it was shown that all 3 groups (C, G1, G2) did not

differ in terms of age and gender, which allowed elimination of the influence of these factors on the test results. Group G2 was shown to have a significantly higher LVMI index, indicative of myocardial hypertrophy, compared to the control group. However, no significant differences were found between the groups in terms of IMT and ABI values. Among patients with OSA, higher eGFR values have also been reported, which may indicate renal hyperfiltration.

**Left ventricular hypertrophy.** In the group of patients with severe OSA (AHI > 30), higher LVMI values were observed compared to the control group (LVMI –  $130.99 \pm 44.6$  g/m<sup>2</sup> in the G2 group and  $106.61 \pm 27.86$  g/m<sup>2</sup> in the control group;  $p = 0.0332$ ). In the analyzed study, abnormal LVMI values were found in 32% of patients in the control group (n=10), in the G1 group in 44% (n=14), while in the G2 group, abnormal LVMI results were observed in more than half of the patients – 51% (n=18 people). The above results are consistent with the results of the study by Dursunoglu et al., who studied 67 patients with suspected sleep disorder breathing, who were divided into 3 groups depending on the severity of respiratory disorders: mild (AHI=5–14), moderate (AHI=15–29) and with severe disease (AHI ≥ 30). The groups did not differ in terms of age, gender and BMI. The authors showed that in the group of patients with severe severity of OSA, left ventricular hypertrophy and LVMI were higher in the group of patients with severe severity of disease, compared to the group of patients with mild severity of disease (LVMI in the group of with severe disease –  $144.7 \pm 39.8$  g/m<sup>2</sup> versus LVMI with mild disease –  $100.5 \pm 42.3$  g/m<sup>2</sup>;  $p = 0.001$ ). However, they did not show statistically significant differences in LVMI between the group with severe and moderate disease severity (LVMI –  $144.7 \pm 39.8$  g/m<sup>2</sup> versus  $126.5 \pm 41.2$  g/m<sup>2</sup>;  $p > 0.05$ , respectively) [10].

The demonstration of high values of the LVMI in the group of normotensive patients suggests that in patients with OSA, the causes of LVH may be other than arterial hypertension. It has been proven that in patients with OSA there is an increase in the level of inflammatory factors, for example, Galectine-3, that may be responsible for LVH [11]. Thus, it is possible that OSA may have an influence on the left ventricular mass, regardless of the coexisting hypertension. This thesis was confirmed by Aslan et al. who confirmed that left ventricular diastolic dysfunction, left ventricular hypertrophy and left atrial dilatation occur in patients with OSA even before the development of hypertension and other cardiovascular diseases [12]. Also, Hedner et al. showed that the left ventricular mass index was about 15% higher in normotensive patients with OSA, compared to the normotensive control group [13].

**Intima Media Thickness.** In the presented study, no statistically significant differences in the IMT values were found between the groups. The mean IMT values were: 0.88 mm, 1.01 mm and 0.97 mm, respectively, for groups C, G1 and G2. These results are in contradiction with the results of other studies assessing the value of IMT in patients with OSA. Most authors showed an independent influence of OSA on IMT. Kostrzewska et al. found that increased carotid intima-media thickness in severe OSA was accompanied by higher systolic and diastolic blood pressures, compared with both moderate OSA and control subjects [14]. In 2014, Ali et al. reviewed 52 studies that assessed the presence of

subclinical features of atherosclerosis among OSA patients using various diagnostic methods (FMD, PWV, IMT) [15]. Minoguchi et al. assessed IMT and serum inflammatory markers in patients with OSA. Thirty-six men with newly diagnosed OSA (age, 23–60 years) and 16 obese male control subjects (age, 25–66 years) were enrolled in this study and underwent polysomnography. In conclusion, patients with OSA had increased carotid IMT and increased serum levels of CRP, IL-6, and IL-18. The carotid IMT was significantly correlated with serum levels of CRP, IL-6, and IL-18 and the severity of OSA [16].

The relationship between IMT and OSA was not confirmed in all clinical observations. In particular, the results of more recent studies suggest the influence of other factors on the development of atherosclerosis rather than OSA itself. Tan et al. examined a large group of OSA patients (156 patients, 142 men and 14 women), excluding patients with existing atherosclerosis or diseases that could cause it. Although they showed higher values of IMT in the group of patients with OSA ( $0.66 \pm 0.14$  versus  $0.58 \pm 0.13$  mm;  $p < 0.002$ ), the factors having a big impact on the values of IMT were: age, fasting glucose, concentration of LDL-C and hsCRP [17]. A study by King et al. produced similar conclusions. Chinese men with polysomnography-diagnosed OSA were subgrouped into mild-moderate (n = 28) and severe (n = 54) OSA groups on the basis of apnea hypopnea index (AHI) scores. The control group consisted of 30 healthy men. One of the assessed factors was IMT. The patients with severe OSA had a significantly higher carotid IMT and levels of inflammatory factors (IL-6 and hs-CRP). This suggests that arterial endothelial damage and inflammation may play important roles in the development of atherosclerosis in OSA patients [18]. Another study was conducted by Theodoropoulos et al. among 40 patients with similar cardiovascular risk, assessed on the basis of the Framingham scale, and with no clinically evident cardiovascular disease. The authors did not find that the severity of newly diagnosed OSA was correlated with the IMT value and with the severity of inflammation assessed on the basis of CRP concentration [19]. The results of the studies cited above suggest that the increase in IMT is rather influenced by diseases comorbid with OSA, such as obesity, hypertension, smoking or diabetes [19].

**Ankle-Brachial Index.** In the presented study, no significant differences were observed in the mean values of the ABI index between the analyzed groups. These results are consistent with the results of the studies by Steiropoulos et al., who in a group of 20 non-smokers with newly diagnosed OSA, without comorbidities, did not observe any differences in the ABI value compared to the appropriate gender and age-matched control group [20].

Nagayoshi et al. in a research group consisting of 1,844 participants (mean age – 63.0 years, 54.1% were women) who had home polysomnography and ABI measurements performed in 2010–2013, identified a total of 101 (5.4%) having a PAD (Peripheral Arterial Disease) (ABI < 0.90). Almost half of this group (n=49) were black people. Sleep apnea severity was not significantly associated with the occurrence of PAD. In cross-sectional analyzes, severe sleep apnea [(AHI) ≥ 30 versus AHI < 5] was associated with a higher incidence of PAD only among blacks [multivariate adjusted prevalence ratio (95% CI): 2.29 (1.07–4, 89); Interaction  $p = 0.05$ ] [21].

In the presented study, the mean values of  $ABI_R$  and  $ABI_L$  were slightly higher in the groups of patients with severe OSA compared to the control group, but these differences were not statistically significant. There was a trend towards vessel stiffness. Schaefer et al. confirmed the increased vascular stiffness in patients with OSA, measured by PWV (Pulse Wave Velocity) [22]. It should be mentioned that the clinical consequences of increased arterial stiffness are increased left ventricular load, and hence increased myocardial oxygen demand and left ventricular hypertrophy. In the presented study, in the group of patients with severe severity of OSA, in which tendencies towards vascular stiffness were observed (the highest mean ABI values), higher LVMI values were also shown compared to the other groups. Despite the high prevalence of PAD in the OSA population (98%) [22], proven in studies, the ABI index is not a sufficiently sensitive indicator for detecting this disease in patients with OSA.

**Glomerular Filtration Rate.** In the presented study, the groups differed significantly in the eGFR value ( $p = 0.0028$ ). The lowest eGFR values were observed in the control group ( $88.85 \pm 17.68$  ml/min/ $1.73$  m<sup>2</sup>), while higher values in both study groups ( $104.35 \pm 21.06$  and  $104.70 \pm 17.96$  ml/min/ $1.73$  m<sup>2</sup> for groups G1 and G2, respectively). Statistically significant differences in Tukey's test were observed for group G1 ( $p=0.0081$ ) and for group G2 ( $p=0.0058$ ), compared to the control group. Therefore, the results of the current study are inconsistent with the results of studies assessing the impact of OSA on renal function, which showed a decrease in eGFR among patients with OSA [23]. Most studies suggest that the presence of OSA and nocturnal hypoxia may lead to worsening of kidney function. One potential mechanism is activation of the renin-angiotensin system by OSA, and this effect may be attenuated by CPAP therapy. [23]. The observed higher values of eGFR in both groups of OSA patients may indicate the presence of glomerular hyperfiltration in this patient population. Increased glomerular filtration is observed, for example, in the early stages of diabetes development and is associated with a high risk of developing diabetic nephropathy. While the initial increase in GFR is favourable, in the long run it contributes to the hardening of the glomeruli, damage to the tubulointerstitial tissue and a decrease in glomerular filtration [24].

Canales et al. performed a retrospective cohort study in 855 participants from the Wisconsin Sleep Cohort Study, a large 20-year population-based study of sleep apnea, who had at least one polysomnogram and serial measurements of serum creatinine over time. They compared the slope of estimated glomerular filtration rate (eGFR) change and odds of rapid eGFR decline ( $>2.2$  mL/minute/ $1.73$  m<sup>2</sup>/year) for those with and without sleep apnea. Among healthy middle-aged adults, the presence of sleep apnea at baseline did not accelerate kidney function decline, compared with those without sleep apnea over time [25]. In the presented study, the influence of OSA on the severity of renal dysfunction as measured by the eGFR value calculated on the basis of the MDRD formula was not observed. The eGFR values in both study groups (G1 and G2) did not differ significantly from each other (in Tukey's test;  $p=0.9972$ ). The lowest creatinine values were recorded in the G1 group ( $AHI=5-30$ ) compared to the control group ( $p=0.0026$ ). Thus, hyperfiltration occurs even among patients with mild or moderate severity of OSA.

## CONCLUSIONS

The highest values of the left ventricular mass index (LVMI) were found in the group of patients with severe OSA. Regardless of the severity of OSA disorders, no influence of OSA on the severity of atherosclerotic processes was observed. An increased value of the eGFR index was observed in the population of OSA patients, also among patients with mild and moderate OSA. In the present study, the influence of other factors that may influence the occurrence of hyperfiltration in patients with OSA cannot be completely eliminated. The finding of an increase in the estimated glomerular filtration rate (eGFR), calculated using the MDRD formula, may indicate that patients with OSA are hyperfiltrating.

## REFERENCES

- Gottlieb DJ, Punjabi NM. Diagnosis and Management of Obstructive Sleep Apnea: A Review. *JAMA*. 2020;323(14):1389–1400.
- Semelka M, Wilson J, Floyd R. Diagnosis and Treatment of Obstructive Sleep Apnea in Adults. *Am Fam Physician*. 2016;94(5):355–60.
- Dredla BK, Castillo PR. Cardiovascular Consequences of Obstructive Sleep Apnea. *Curr Cardiol Rep*. 2019;21(11):137.
- Lin C-H, Lurie RC, Lyons OD. Sleep apnea and chronic kidney disease. A state of the art review. *Chest* 2020;157(3):673–685.
- Williams B, Mancia G, Spiering W, et al. 2018 ESH/ESC guidelines for the management of arterial hypertension. *Eur Heart J*. 2018; 39(33):3021–3104.
- Ito E, Inoue Y. International Classification of Sleep Disorders, Third Edition. American Academy of Sleep Medicine. *Nihon Rinsho*. 2015; 73(6):916–923.
- Corona-Villalobos CP, Sorensen L, Pozios I, et al. Left ventricular wall thickness in patients with hypertrophic cardiomyopathy: a comparison between cardiac magnetic resonance imaging and echocardiography. *Int J Cardiovasc Imaging* 2016;32(6):945–954.
- Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging*. 2015;16(3):233–70.
- Levey AS, Inker LA, Coresh J. GFR estimation: from physiology to public health. *Am J Kidney Dis*. 2014;63(5):820–34.
- Dursunoglu D, Dursunoglu N, Evrengul H, et al. Impact of obstructive sleep apnoea on left ventricular mass and global function. *Eur Respir J*. 2005;26:283–288;
- Cicco S, Castellana G, Marra L, Di Lecce V, Carratù P, Prete M, Ranieri G, Resta O, Carpagnano GE, Racanelli V, Dragonieri S, Vacca A. Galectin-3 and neutrophil-to-lymphocyte ratio are indicative of heart remodelling and disease severity in patients with obstructive sleep apnoea. *Sleep Med*. 2021;82:117–124.
- Aslan K, Deniz A, Cayli M, Bozdemir H, Sarica Y, Seydaoglu G. Early left ventricular functional alterations in patients with obstructive sleep apnea syndrome. *Cardiol J*. 2013;20(5):519–25.
- Hedner J, Ejnell H, Caidahl K. Left ventricular hypertrophy independent of hypertension in patients with obstructive sleep apnea. *J Hypertens*. 1990;8(10):941–946.
- Kostrzewska M, Piorunek T, Hoffmann K, et al. Carotid artery intima-media thickness in hypertensive patients with obstructive sleep apnea. *Adv Exp Med Biol*. 2015;839:61–66.
- Ali SS, Oni ET, Warraich HJ, et al. Systematic review on noninvasive assessment of subclinical cardiovascular disease in obstructive sleep apnea: new kid on the block! *Sleep Med Rev*. 2014;18(5): 379–391.
- Minoguchi K, Yokoe T, Tazaki T, et al. Increased Carotid Intima-Media Thickness and Serum Inflammatory Markers in Obstructive Sleep Apnea. *Am J Respir Crit Care Med*. 2005;172:625–30.
- Tan TY, Liou CW, Friedman M, et al. Factors associated with increased carotid intima-media thickness in obstructive sleep apnea/hypopnea syndrome. *Neurologist* 2012;18(5):277–281.
- Kong D, Qin Z, Wang W, Kang J. Effect of obstructive sleep apnea on carotid artery intima media thickness related to inflammation. *Clin Invest Med*. 2017;40(1):E25–E33.

19. Theodoropoulos K, Lykouras D, Karkoulas K, et al. Association between the severity of newly diagnosed obstructive sleep apnea and subclinical carotid atherosclerosis in patients without overt cardiovascular disease. *Eur Rev Med Pharmacol Sci.* 2017;21(7):1568–1575.
20. Steiropoulos P, Bogiatzi C, Archontogeorgis K, et al. Is there evidence of early vascular disease in patients with obstructive sleep apnoea without known comorbidities? Preliminary findings. *Open Cardiovasc Med J.* 2013;7:61–68.
21. Nagayoshi M, Lutsey PL, Benkeser D, et al. Association of sleep apnea and sleep duration with peripheral artery disease: The Multi-Ethnic Study of Atherosclerosis (MESA). *Atherosclerosis* 2016;251:467–475.
22. Schaefer CA, Adam L, Weisser-Thomas J, et al. High prevalence of peripheral arterial disease in patients with obstructive sleep apnoea. *Clin Res Cardiol.* 2015;104(9):719–26.
23. Lin C-H, Perger E, Lyons OD. Obstructive sleep apnea and chronic kidney disease. *Curr Opin Pulm Med.* 2018;24(6):549–554.
24. Kanbay M, Ertuglu LA, Afsar B, et al. Renal hyperfiltration defined by high estimated glomerular filtration rate: A risk factor for cardiovascular disease and mortality. *Diabetes Obes Metab.* 2019;21(11):2368–2383.
25. Canales MT, Hagen EW, Barnett et al. Sleep Apnea and Kidney Function Trajectory: Results From a 20-Year Longitudinal Study of Healthy Middle-Aged Adults. *Sleep.* 2018;41(1):181.