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# Can biomarkers predict the risk of cardiovascular disease in patients with obstructive sleep apnea syndrome?

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**Ethics Committee Approval**
The study was approved by the Ethical Committee of Binali Yildirim University (Decree No: 21/06/2021, 08/11).
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

**Conflict of Interest**
No conflict of interest was declared by the authors.

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## Abstract

**Background/Aim:** Obstructive sleep apnea syndrome (OSAS) is a clinical syndrome characterized by recurrent partial or total obstruction of the upper airway. Cardiovascular disease (CVD) is more common in OSAS patients. Biomarkers can predict the progression of OSAS disease and the occurrence of CVD. Here we investigate the effects of age, gender, body mass index (BMI), comorbidities, neutrophil-to-lymphocyte ratio (NLR), lymphocyte-to-monocyte ratio (LMR), platelet-to-lymphocyte ratio (PLR), systemic inflammatory index (SII), atherogenic index of plasma (AIP), C-reactive protein-to-albumin ratio (CAR) and monocyte to HDL cholesterol (MHR) on the severity of OSAS and the occurrence of CVD in OSAS patients.

**Method:** This cross-sectional study included 172 OSAS patients presenting to Erzincan Binali Yildirim University Mengucek Gazi Training and Research Hospital, Sleep Service between 01.01.2021 and 31.08.2022. Polysomnography (PSG) was applied to patients participating in the study, and routine complete blood and biochemistry tests were performed. Comorbidities and demographic data of the participants were recorded.

**Results:** The frequency of CVD, chronic pulmonary disease (CPD) and hyperlipidemia increased as the severity of OSAS increased ($P$=0.005, $P$<0.001, $P$=0.016, respectively). As the severity of OSAS disease increased, only the MHR indices increased ($P$=0.009). When OSAS groups with and without CVD were examined, OSAS patients with CVD were older and had higher BMI ($P$<0.001, $P$=0.001, respectively). In addition, tended to be females with hyperlipidemia, diabetes mellitus (DM) and high Charlson Comorbidity Index (CCI) scores (all $P$<0.001). When the polysomnography of OSAS patients with CVD was evaluated, apnea-hypopnea index (AHI), non-rapid eye movement (NREM)-AHI (NREM-AHI), respiratory disorder index (RDI) and oxygen desaturation index (ODI) values were higher and sleep efficiency (SE) values were lower than patients with OSAS without CVD ($P$=0.002, $P$=0.002, $P$=0.003, $P$<0.001, $P$<0.001, respectively).

**Conclusion:** CVD is common in OSAS patients. As the severity of OSAS increases, the risk of developing CVD increases. Elderly female OSAS patients with hyperlipidemic, DM, high BMI, and Charlson Comorbidity Index (CCI) constitute a relatively risky group for CVD. OSAS patients with high AHI, NREM-AHI, RDI, ODI, and low SE values should be monitored more closely for CVD.

**Keywords:** OSAS, cardiovascular disease, biomarker, AHI

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## Introduction

Obstructive sleep apnea syndrome (OSAS) is a disease that affects 3–7% of the middle-aged population [1]. OSAS is a clinical syndrome characterized by recurrent partial or total upper airway obstruction. Its main symptoms are snoring, excessive daytime sleepiness, and witnessed apnea [2]. OSAS is diagnosed using nocturnal polysomnography (PSG) testing. The diagnostic criteria for OSAS are an apnea-hypopnea index (AHI) >5 on PSG in the presence of symptoms or an AHI >15 for an asymptomatic patient [3].

Comorbid conditions, such as obesity, smoking, hypertension, hyperlipidemia, metabolic syndrome, diabetes mellitus (DM), and insulin resistance, are more common in OSAS patients compared to the general population.

Recurrent episodes of nocturnal apnea in OSAS patients cause sympathetic system activation, increased oxidative stress, endothelial dysfunction, a spike in systemic hypertension, hypoxia, and hypercapnia [3]. Intermittent episodes of hypoxia resulting from temporary respiratory arrest and ischemia-reperfusion injury during sleep are the main physiological features of OSAS. Intermittent episodes of nocturnal hypoxemia also induce the generation of oxygen radicals that cause low-grade inflammation [4]. Oxidizing radicals and proteolytic enzymes accumulate leukocytes and platelets on blood vessel walls, leading to endothelial dysfunction [5]. Inflammation is one of the main factors contributing to the initiation and progression of atherosclerosis [6]. Studies have shown that autonomic and neurohumoral abnormalities in OSAS persist during the daytime, disrupting the general circadian blood pressure rhythm and increasing short- and long-term blood pressure variability [7-9]. Absolute blood pressure elevation and fluctuations seen in OSAS have been found to cause organ damage by promoting arterial remodeling, microvascular damage, hemodynamic instability, and vascular reactivity [10-13].

Endothelial dysfunction is involved in the pathophysiology of hypertension, DM, coronary artery disease (CAD), and congestive heart failure (CHF). Endothelial dysfunction is also common in OSAS patients [14]. For these reasons, cardiovascular diseases are more common in OSAS patients [15,16]. The chronic systemic inflammation seen in OSAS may play an important role in the progression of cardiovascular disease (CVD) [17]. Studies have shown that OSAS is an independent risk factor for CVD [18].

Recent studies show that WBC and neutrophil-to-lymphocyte ratio (NLR) are good indicators of inflammation [19-23]. Studies reveal platelet/lymphocyte ratio (PLR) as a new inflammatory marker in predicting CVD [22-24]. Unlike the separate analysis of CRP and albumin, the C-reactive protein/albumin ratio (CAR) can be used as a more reliable biomarker to predict the severity and prognosis of diseases [25]. CAR was found to be significantly higher in patients with high thrombus burden. CAR can be used as a marker of proinflammation, which is closely related to the prothrombotic state. In previous studies, the systemic inflammatory index (SII) indicates inflammatory status [26]. The atherogenic index of the plasma (AIP) can be defined as a marker of atherogenic oxidized small-density LDL-cholesterol and has predictive value for cardiovascular disease risk [27]. Patients with OSAS have higher AIP and triglyceride and lower HDL-cholesterol values. The monocyte/HDL-cholesterol ratio (MHR) is a practical, cost-effective marker for determining CVD risk and may indicate endothelial dysfunction [18,28].

In our study, we aimed to investigate the relationship between inexpensive, practical, and easily obtainable complete blood and biochemistry tests and NLR, MHR, LMR, and PLR values calculated from these tests with the severity of OSAS patients and to find out their contribution in predicting CVD risk. As far as we know, very few studies have been conducted on the CVD formation of CAR, SII, and AIP values in OSAS patients. In our study, we aimed to determine CAR, SII, and AIP levels' contributions to CVD risk in OSAS patients and the severity of OSAS. In addition, we aimed to determine the effects of age, gender, and body mass index (BMI) on disease progression and CVD formation in our OSAS patients. Finally, we aimed to investigate the relationship between the severity of OSAS and comorbid conditions.

## Materials and methods

This cross-sectional studies study includes 172 patients between 40 and 110 years old who were admitted to Erzincan Binali Yildirim University Mengucek Gazi Training and Research Hospital Sleep Department between 01.01.2021 and 31.08.2022 and diagnosed with OSAS according to the American Academy of Sleep Medicine (AASM). Patients younger than 18 years old, those with central sleep apnea syndrome, patients with upper respiratory tract resistance syndrome, and those with narcolepsy and movement disorders were excluded from the study. The study was carried out following the Declaration of Helsinki.

PSG evaluation of all patients was performed, and patients were grouped as mild (AHI: 5–15), moderate (AHI: 15–30), and severe OSAS (AHI) >30) according to AASM criteria (14). All patients underwent nocturnal PSG (55-channel polysomnography (Alice Sleepware; Philips Respironics, Pennsylvania, USA). Electrooculogram (two channels), electroencephalogram (four channels), electromyograms of submental muscles (one channel), anterior tibialis muscle of both legs (two channels), electrocardiograms, airflow measurements (with oronasal), thermistor and nasal cannula pressure transducer, and a snoring sensor to detect sleep and snoring vibrations were used in all patients. Using a finger probe, the thoracic and abdominal muscles' respiratory effort (two channels) was recorded using piezoelectric belts and arterial blood pressure, pulse, and oxyhemoglobin saturation (SaO2: 1 channel). Apnea was defined as a 90% reduction in airflow amplitude or 10 s relative to baseline amplitude. According to the AASM, hypopnea was defined as a 30% decrease in airflow amplitude relative to a recommended baseline and values associated with 4% oxygen desaturation lasting at least 10 s. In patients with OSAS, the number of AHI apneic plus hypopneic episodes was calculated per hour of sleep. The oxygen desaturation index (ODI) was defined as the number of measurements of -4% oxyhemoglobin desaturation within 10 s to <3 min from baseline. Mean oxygen saturation, minimum oxygen saturation, oxygen desaturation index, desaturation %,

AHI, REM AHI, NREM-AHI, sleep efficiency (SE), arousal index (AI), and respiratory disorder index (RDI) values of the patients were recorded.

Demographic characteristics, pulse rates, arterial blood pressure, sleep patterns, medical histories, cardiovascular and metabolic diseases, and drug use habits of the patients were also recorded. In the study, cardiovascular disease was diagnosed by a specialist cardiologist using an electrocardiogram, echocardiography, and coronary angiography and by evaluating the patient's medical history and drug use. CVD was used only for heart failure, coronary artery disease or arrhythmia diseases.

On the morning of PSG, patient venous blood samples were obtained after 12 h of fasting. Triglyceride, total cholesterol, LDL-cholesterol, HDL-cholesterol levels and leukocyte, neutrophil, lymphocyte and monocyte counts were determined by Sysmex XN_100 and Sysmex XN_2000 devices. Serum albumin level was analyzed using automated photometry kits (AU 2700 Backman Olympus spectrophotometric method). Serum CRP levels were measured by the nephelometric method (Siemens BN11 system). CAR was obtained by multiplying the CRP to albumin ratio by 100. The AIP value was calculated as the base 10 logarithmic conversion of the triglyceride to high-density lipoprotein-cholesterol ratio (TG / HDL-cholesterol). The systemic inflammation index (SII) was calculated using the neutrophil × platelet/lymphocyte formula. The ratio of monocytes/HDL-cholesterol found in MHR. Local Ethics Committee approval (Decree No: 21/06/2021, 08/11) was obtained from Binali Yildirim University for the study, and the study was performed per the ethical standards specified in the 1964 Declaration of Helsinki and its later amendments.

**Bias**

Patients with OSAS in all severity who applied to the OSAS clinic were included in the study sequentially. The cardiologist did not know the severity of OSAS in the patients.

**Study size**

All patients diagnosed as OSAS according to AASM criteria applied to Erzincan Binali Yıldırım University Mengucek Gazi Training and Research Hospital Sleep Service between 01.01.2021-31.08.2022 were included in the study, respectively.

**Statistical analysis**

Categorical variables were expressed as numbers and percentages, whereas continuous variables were summarized as mean, standard deviation, or median (Q1-Q3). The chi-square test was used to compare categorical variables between the groups. The normality of distribution for continuous variables was confirmed with the Shapiro-Wilk test. Depending on the distribution, the Student's t-test or Mann-Whitney U test was used to compare continuous variables between the two groups. The comparison between more than two groups was made by One-way ANOVA or the Kruskal-Wallis test. Logistic regression analysis was performed to determine significant predictors of CVD risk. In univariate analysis, variables significant at the $P$<0.25 level were entered in logistic regression analysis. All analyses were performed using IBM SPSS 20. A value of $P$<0.05 for all tests was considered statistically significant.

## Results

One-hundred-seventy-two OSAS patients were included in our study. Age distributions were similar in all OSAS severity groups ($P$=0.302). The gender distribution did not affect the OSAS severity and showed a similar distribution in control, mild, moderate, and severe OSAS groups ($P$=0.369). BMI value, CCI score, and DM frequency were similar in all OSAS severity groups ($P$=0.312, $P$=0.062, $P$=0.663, respectively). CVD, CPD, and hyperlipidemia were seen more frequently as the severity of OSAS increased ($P$=0.005, $P$<0.001, $P$=0.016, respectively). As the severity of OSAS disease increased, only the MHR indices increased, while NLR, LMR, PLR, SII, CAR, and AIP did not change ($P$=0.009, $P$=0.134, $P$=0.060, $P$=0.796, $P$=0.307, $P$=0.187, $P$=0.333, respectively) (Table 1).

Table 1: Age, gender, comorbidities, laboratory values and indices of patients are seen in control and all OSAS severity groups.

| | Mild OSAS (n=67) | Medium OSAS (n=52) | Severe OSAS (n=53) | *P*-value |
|---|---|---|---|---|
| **Age** | 51.60 (9.36) | 54.31 (10.87) | 54.02 (11.76) | 0.302* |
| **Gender (M/F)** | 42/25 | 27/25 | 34/19 | 0.369$^{+}$ |
| **BMI** | 32.0 (5.9) | 33.4 (7.3) | 33.7 (6.1) | 0.312* |
| **CCI** | 1.0 (0.0-2.0) | 1.0 (0.0-2.0) | 1.0 (1.0-2.0) | 0.062** |
| **CVD %** | 21 (46.3%)$^{a}$ | 31 (59.6%)$^{a,b}$ | 40 (75.5%)$^{b}$ | 0.005$^{+}$ |
| **CPD %** | 24 (35.8%)$^{a}$ | 26 (50.0%)$^{a}$ | 39 (73.6%)$^{b}$ | <0.001$^{+}$ |
| **DM %** | 21 (31.3%) | 20 (38.5%) | 20 (37.7%) | 0.663$^{+}$ |
| **Hyperlipidemia %** | 11 (16.4%)$^{a}$ | 8 (15.7%)$^{a,b}$ | 18 (35.8%)$^{b}$ | 0.016$^{+}$ |
| **NLR** | 1.7 (1.3-2.1) | 1.6 (1.1-2.0) | 1.7 (1,5-2.4) | 0.134** |
| **LMR** | 3.2 (2.7-4.6) | 3.7 (2.7-4.6) | 2.5 (1.7-4.3) | 0.060** |
| **PLR** | 108.1 (88.6-128.5) | 102.3 (83.5-124.0) | 105.7 (84.6-123.5) | 0.796** |
| **SII** | 418.7 (333.0-621.9) | 376.9 (327.6-518.6) | 457.3 (316.8-644.9) | 0.307** |
| **CAR** | 7.5 (7.1-7.8) | 7.7 (7.3-9.3) | 7.5 (7.4-7.7) | 0.187** |
| **AIP** | 0.20 (0.03-034) | 0.22 (-020-0.36) | 0.24 (0.6-0,45 | 0.333** |
| **MHR** | 0.02 8 (0.1-0.2)$^{a}$ | 0.01 (0.01-0.02)$^{a}$ | 0.2 (0.01-0.03)$^{b}$ | 0.009** |

*One-way ANOVA, **Kruskal Wallis test and $^{+}$Chi-square test was performed. Results were presented as Mean (SD) or Median (Q1-Q3). BMI: body mass index, CCI: Charlson comorbidity index, CVD: cardiovascular disease, CPD: chronic pulmonary disease, DM: diabetes mellitus, NLR: neutrophil-to-lymphocyte ratio, LMR: lymphocyte-to-monocyte ratio, PLR: platelet-to-lymphocyte ratio, SII: systemic inflammatory index, CAR: C-reactive protein-to-albumin ratio, AIP: atherogenic index of plasma, MHR: monocyte to HDL cholesterol ratio.

The rate of OSAS patients without CVD comorbidity was 40.69% (n=70). The rate of patients with CVD comorbidity was 59.30% (n=102). We observed that OSAS patients with CVD were more commonly female, elderly, and with high BMI values ($P$=0.004, $P$<0.001, $P$<0.001, respectively). It was determined that the CCI score was higher, and hyperlipidemia and DM were more common in OSAS patients with CVD ($P$<0.001, $P$<0.001. $P$<0.001, respectively). When laboratory indices were examined in patients with and without CVD, it was observed that NLR, LMR, PLR, SII, CAR, AIP, and MHR were at similar values ($P$=0.366, $P$=0.396, $P$=0.689, $P$=0.361, $P$=0.803, $P$=0.691, $P$=0.994, respectively). When OSAS groups with and without CVD were examined polysomnographically, it was seen that AHI, NREM-AHI, RDI and ODI values were higher ($P$=0.002, $P$=0.002, $P$=0.003, and $P$<0.001, respectively) and SE values were lower ($P$<0.001) in the OSAS group with CVD, while REM-AHI and AI were similar in both groups (P=0.882 and $P$=0.321, respectively) (Table 2, Figure 1). Logistic regression analysis was performed using the Forward LR method to determine the potential risk factors for CVD, and variables were reported in Table 3. While age, BMI, and NREM-AHI increase, the risk of CVD increases. Also, having hyperlipidemia increased the risk of CVD by 3.9 times (95% CI: 1.3–11.6). For SE, having high sleep efficiency decreased CVD risk (OR: 0.95 [0.92–0.98]).

Table 2: Age, gender, comorbidities, indices, and polysomnographic findings are seen in OSAS patients who do not develop CVD and who develop CVD.

| | CVD - (n=70) | CVD + (n=102) | *P*-value |
|---|---|---|---|
| **Age** | 48.0 (10.3) | 56.8 (9.3) | <0.001* |
| **Gender (M/F)** | (51/19) | (52/50) | 0.004$^{+}$ |
| **BMI** | 30.3 (4.4) | 34.8 (7.0) | <0.001* |
| **Hyperlipidemia** | 7 (10.0%) | 31 (30.7%) | 0.001$^{+}$ |
| **DM** | 13 (18.6%) | 48 (47.1%) | <0.001$^{+}$ |
| **CCI** | 0.5 (0.1-1.0) | 1 (1.0-2.0) | <0.001** |
| **NLR** | 1.65 (1.26-2.00) | 1.71 (1.33-2.25) | 0.366** |
| **LMR** | 3.18 (2.35-4.65) | 3.17 (2.00-4.49) | 0.396** |
| **PLR** | 106.36 (84.33-123.89) | 105.22 (84.58-128.03) | 0.689** |
| **SII** | 396.90 (319.17-522.00) | 437.03 (326.24-604.58) | 0.361** |
| **CAR** | 7.50 (7.13-8.68) | 7.5 (7.32-7.89) | 0.803** |
| **AIP** | 0.24 (0.07-0.36) | 0.22 (0.01-0.39) | 0.691** |
| **MHR** | 0.02 (0.01-0.02) | 0.02 (0.01-0.02) | 0.994** |
| **AHI** | 14.3 (9.0-26.3) | 21.3 (12.0-53.0) | 0.002** |
| **REM-AHI** | 25.3 (13.5-45.6) | 28.2 (9.3-50.4) | 0.882** |
| **NREM-AHI** | 12.6 (7.9-27.6) | 21.4 (10.6-53) | 0.002** |
| **SE%** | 78.9 (70.9-88.4) | 73.0 (59.5-80.0) | <0.001** |
| **AI** | 10.3 (6.8-18.7) | 12.0 (6.7-24.7) | 0.321** |
| **RDI** | 14.6 (9.1-26.9) | 21.6 (12.0-53.0) | 0.003** |
| **ODI** | 6.8 (4.0-11.1) | 9.9 (5.6-23.6) | 0.001** |

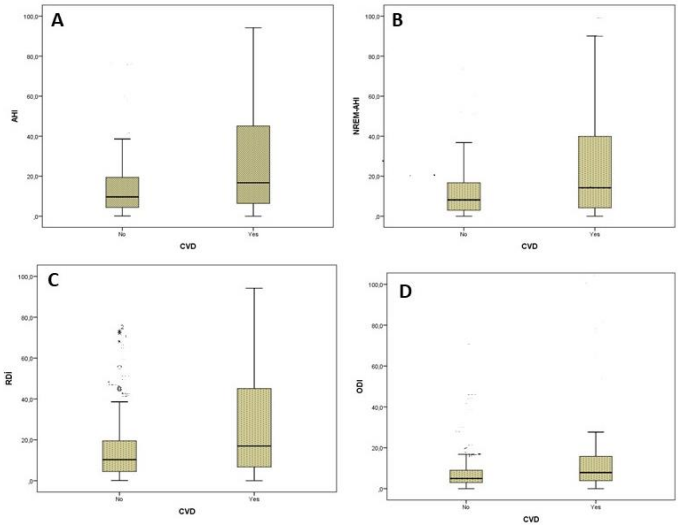
Student t-test, **Mann Whitney U test and $^{+}$Chi-square test were performed. Results were presented as Mean (SD) or Median (Q1-Q3). BMI: body mass index, CCI: Charlson comorbidity index, DM: diabetes mellitus, NLR: neutrophil-to-lymphocyte ratio, LMR: lymphocyte-to-monocyte ratio, PLR: platelet-to-lymphocyte ratio, SII: systemic inflammatory index, CAR: C-reactive protein-to-albumin ratio, AIP: atherogenic index of plasma, MHR: monocyte to HDL cholesterol ratio, AHI: apnea-hypopnea index, NREM-AHI: non-rapid eye movement AHI, RDI: respiratory disorder index, ODI: oxygen desaturation index, CVD: cardiovascular disease.

Table 3: Logistic regression results for CVD in patients with obstructive sleep apnea syndrome.

| | OR | 95% CI for OR | | *P*-value |
|---|---|---|---|---|
| | | Lower | Upper | |
| **Age (years)** | 1.1 | 1.035 | 1.131 | <0.001 |
| **BMI (kg/m$^2$)** | 1.2 | 1.062 | 1.257 | 0.001 |
| **Hyperlipidemia** | 3.9 | 1.298 | 11.642 | 0.015 |
| **NREM-AHI** | 1.03 | 1.012 | 1.052 | <0.001 |
| **SE%** | 0.95 | 0.92 | 0.98 | 0.005 |

OR-Odds Ratio, CI- confidence interval, BMI-body mass index, NREM-AHI-non-rapid eye movement AHI, SE%-sleep efficiency. CVD- Cardiovascular Disease

Figure 1: Polysomnographic findings are seen in OSAS patients who do not develop CVD and in groups that develop CVD. AHI (A), NREM-AHI (B), RDI (C), and ODI (D).




AHI: apnea-hypopnea index, NREM-AHI: non-rapid eye movement AHI, RDI: respiratory disorder index, ODI: oxygen desaturation index, CVD: cardiovascular disease

## Discussion

OSAS is characterized by recurrent narrowing of the upper airway causing intermittent oxyhemoglobin desaturation, sleep fragmentation, and daytime sleepiness.

The risk of CVD is increased in OSAS patients due to intermittent hypoxia, high BMI, excessive free radical formation, and the frequent coexistence of additional diseases, such as DM and hyperlipidemia. Predicting CVD is vital in these patients. Demographic data of patients, comorbidities, laboratory values, and polysomnographic measurements can be helpful in this regard. In a study of 2353 people, it was found that OSAS was more severe, especially in older men [29]. Our study observed that OSAS patients with CVD were older, but no significant relationship was found between OSAS severity and age.

Studies have shown that the incidence of OSAS in men is twice that of women [30]. In our study, gender did not affect the severity of OSAS. Interestingly, the frequency of CVD was higher in female OSAS patients than in males.

A study showed that BMI and DM are independent risk factors for OSAS, and central obesity may be more dangerous than BMI in this regard [29]. Another study showed that BMI is ineffective in determining the severity of OSAS [31]. In our study, BMI was similar in all OSAS severity groups. However, OSAS patients with CVD had higher BMI values.

A study showed that the most common comorbidities in OSAS patients were obesity, HT, and DM. CHF, Deep vein thrombosis (DVT), Pulmonary thromboembolism (PTE), and hypothyroidism were more common, especially in the severe OSAS group [30]. Similarly, in our study, the frequency of CVD, CPD, and hyperlipidemia was higher in the severe OSAS group than in the mild OSAS group. In addition, in our study, it was observed that hyperlipidemia and DM were more common, and the CCI score was higher in the OSAS group with CVD.

It has been reported that the relationship between OSAS and triglyceride and HDL-cholesterol is independent of obesity [32]. Another study determined that the TG\HDL-cholesterol ratio was associated with the severity of OSAS [33]. The coexistence of OSAS and dyslipidemia may be affected by genetic factors, diet, medications used by the patient, and comorbidities. Intermittent hypoxia and disruption of sleep structure may cause dyslipidemia in patients with OSAS. Sleep disturbance can also cause overeating [34]. It has been shown that REM sleep disorder more commonly causes dyslipidemia than NREM sleep disorder [35]. In our study, it was observed that the frequency of hyperlipidemia increased as the severity of OSAS increased, similar to the literature. In addition, hyperlipidemia was more common in OSAS patients with CVD.

Uygur et al. [36] found a correlation between NLR and OSAS severity and showed that NLR is an independent risk factor for CVD in OSAS patients. LMR can be used as an inflammation marker. Another study has reported a negative correlation between LMR and CAD [37]. And another study found a correlation between OSAS severity and SII values [26]. It has been shown that there is an increase in CVD risk in parallel with the increase in MHR in OSAS patients, and MHR was found to be higher in OSAS patients with CVD [38,39]. Among the indices examined in our study, only the MHR index increased with the severity of OSAS. The NLR, LMR, PLR, SII, CAR, AIP, and MHR indices were similar in OSAS patients with and without CVD.

Campos-Rodriges et al. [40] found that untreated OSAS patients had a significantly increased risk of CVD. Wang et al. [41] found a higher CVD risk in moderate and severe OSAS than in mild OSAS patients. Loke et al. [42] found that an increase in AHI of 10 or more increased the risk of CVD by 36%. In our study, it was observed that the rate of having CVD increased as the severity of OSAS increased, similar to the literature. In addition, in our study, the incidence of CVD in OSAS patients was positively correlated with AHI, NREM-AHI, RDI, and ODI

scores and negatively correlated with SE scores. AI and REM-AHI values were found to be similar in both groups.

**Limitations**

Our study had several limitations. A relatively small number of patients were included in our study. The fact that patients were not asked about their smoking and alcohol use is another limitation of my study. The fact that the duration of additional diseases that the OSAS patients have is not considered is another limitation.

**Conclusion**

The frequency of CVD is increased in OSAS patients because of intermittent hypoxia and common risk factors with CVD. As the severity of OSAS increases, the risk of developing CVD increases. Here, we found that as OSAS severity increases, CVD, CPD, and hyperlipidemia may increase, and increased NLR, SII, and MHR and decreased LMR were associated with OSAS severity. Elderly and hyperlipidemic female OSAS patients with DM and high CCI and BMI values constitute a higher risk group for CVD. In addition, OSAS patients with high AHI, NREM-AHI, RDI, ODI, and low SE values should be monitored more closely for CVD. One of the important causes of mortality in OSAS is CVD. Predicting the risk of CVD with OSAS is vital. The patient's age, gender, BMI, and additional diseases can give an idea of the formation of CVD. In addition, polysomnographic examination of patients with OSAS can be life-saving in terms of CVD risk

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