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Research Article

Selected Biomarkers in Patients with Obstructive Sleep Apnea Syndrome Before and After Continuous Positive Airway Pressure Treatment

İbrahim Arslan[1]*, Hülya Eyigör[1], Kadir Çetin[1], Ömer Tarık Selçuk[1], Serkan Özben[2], Tomris Özben[3] and Hatice Yılmazçoban[1]

[1]Antalya Education and Research Hospital, Department of Ear Nose and Throat, Antalya, Turkey

[2]Antalya Education and Research Hospital, Department of Neurology, Antalya, Turkey

[3]Akdeniz University Hospital, Department of Medical Biochemistry, Antalya, Turkey

***Corresponding Author:** İbrahim Arslan, Antalya Education and Research Hospital, Department of Ear Nose and Throat, Antalya, Turkey.

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Abstract

Objective: Obstructive sleep apnea syndrome (OSAS) is associated with cardiovascular diseases such as hypertension and arrhythmia, metabolic diseases such as diabetes, and cerebrovascular diseases such as thromboembolism. Continuous positive airway pressure (CPAP) is effectively used in the treatment of patients with moderate and severe OSAS. In this study, we aimed to measure serum biomarker levels of nesfatin-1, salusin alpha, salusin beta, and galectin-3 in patients with moderate and severe OSAS, evaluate their relationship with disease severity, and examine the changes in these biomarkers after a long-term CPAP therapy.

Method: A two-center prospective study was performed. A total of 23 patients diagnosed with OSA were included in this study. A control group was formed with 21 healthy volunteers. Serum concentration levels of nesfatin-1, salusin alpha and beta, and galectin-3 were determined using the enzyme-linked immunosorbent assay method. The patients who received CPAP therapy were followed up every three months for one year, and their compliance with treatment was measured using the internal data stores of the CPAP device.

Results: In this study, we found nesfatin-1, salusin alfa, salusin beta and galectin-3 levels were significantly lower in patients with OSAS compared to the healthy controls ($p = 0.025$, $p = 0.049$, $p = 0.002$ and $p = 0.043$, respectively). There was a statistically significant negative relationship between Apnea-Hipopnea Index (AHI) and the nesfatin-1 levels ($p = 0,006$). There was also no significant change in biomarker levels among the patients with OSAS before CPAP and after 12 months of therapy ($p > 0.05$).

Conclusion: The data of our study show a clear negative correlation between nesfatin-1, salusin alfa, salusin beta, galectin-3 levels and OSAS. Furthermore, serum nesfatin-1 levels were negatively correlated with the presence of OSAS and AHI score. Serum nesfatin-1 level may be use of as a new biomarker for early detection and risk assesment of OSAS and its severity. CPAP treatment did not alter levels of these circulating markers.

Keywords: Obstructive Sleep Apnea Syndrome; Continuous Positive Airway Pressure; Nesfatin-1; Salusin Alfa; Salusin Beta; Galectin-3

Abbreviation

AHI: Apnea-Hypopnea İndex; BMI: Body Mass İndex; CPAP: Continuous Positive Airway Pressure; OSAS: Obstructive Sleep Apnea Syndrome

Introduction

Obstructive sleep apnea syndrome (OSAS) is one of the most common sleep-related respiratory disorders across the world, with an increasing prevalence among individuals aged 30-69 years [1-3]. OSAS is a syndrome characterized by apneas and hypopneas that arise from repetitive obstruction of the upper airway while respiratory effort continues throughout sleep. During episodes of apnea resulting from upper airway obstruction, individuals experience arousal reactions accompanied by desaturation in arterial blood, an increase in negative intrathoracic pressure, and activation of the sympathetic nervous system. Subsequently, abnormal responses are observed in the cardiovascular, hormonal, and metabolic systems [4]. Therefore, OSAS is associated with cardiovascular diseases such as hypertension and arrhythmia, metabolic diseases such as diabetes, and cerebrovascular diseases such as thromboembolism [5]. Many studies have shown a relationship between OSAS and atherosclerosis, oxidative stress, endothelial dysfunction, and lipid metabolism [6-11].

OSAS and serum biomarkers

Continuous positive airway pressure (CPAP) is effectively used in the treatment of patients with moderate and severe OSAS. CPAP therapy has been shown to improve endothelial dysfunction, reduce systemic inflammation, inhibit erythropoiesis, and cause changes in some metabolic parameters, such as hemoglobin A1c [12,13].

Nesfatin-1 is a protein that contains 82 amino acids derived from nucleobindin-2. In a study conducted on rats, intracerebroventricular injection of nesfatin-1 was reported to suppress food intake at night and decrease body weight [14], which suggests that nesfatin-1 plays an important role in the pathogenesis of obesity. It has also been shown that nesfatin-1 has anti-inflammatory effects [15]. Salusins, namely salusin alpha and salusin beta, are peptides consisting of 28 and 20 amino acids, respectively. Synthesis and expression of salusins occur in human, mouse, and rat tissues in the kidney, central nervous system, and vascular system [16]. While salusins are also expressed in coronary atherosclerotic plaques, the expression of salusin alpha is observed at a lower rate than that of salusin beta [17]. OSAS and atherosclerosis share many risk factors, including obesity, age, male gender, metabolic syndrome, smoking, increased high-sensitivity C-reactive protein levels, and insulin resistance [18-21]. Galectin-3 is a member of the β-galactoside-binding lectin family and is a peptide with a molecular weight of 30-35 kDa. It contains a special amino terminal region rich in proline and glycine. It functions as a paracrine signal, leading to macrophage and fibroblast proliferation and fibrosis [22]. High plasma galectin-3 levels have been associated with increased mortality secondary to cardiovascular diseases [23-25]. Similarly, the risk of cardiovascular diseases, such as heart failure, coronary artery disease, and atrial fibrillation, is known to increase in OSAS [26-28]. There are studies showing a relationship between galectin-3 and some types of cancer, although controversial results have been reported [29-34].

Considering OSAS and the associated systemic diseases, studies have been conducted on serum biomarkers to evaluate the severity, prognosis, and response to treatment of OSAS; however, it remains controversial which biomarkers have superior diagnostic or prognostic features. In this study, we aimed to measure serum biomarker levels of nesfatin-1, salusin alpha and beta, and galectin-3 in patients with moderate and severe OSAS, evaluate their relationship with disease severity, and examine the changes in these biomarkers after CPAP therapy.

Methods

The study included 23 patients who underwent polysomniography at the sleep laboratory of our clinic from January 2021 through November 2022 and were diagnosed with moderate and severe OSAS based on their apnea-hypopnea index (AHI) values (>15-30 and 30, respectively). AHI, age, gender, body mass index, and chronic disease parameters were recorded in these patients. Individuals with a history of malignancy and comorbidities, such as severe mental illness, hearth failure (HF), and uncontrolled hypertension, were not included in the study. A control group was formed with 21 healthy volunteers with an AHI value of <5 and no history of chronic disease. The medical files of all participants were screened. All participants were also examined in detail at our clinic, and those with nasal pathologies such as septum deviation, nasal polyps, and nasal valve stenosis were excluded.

This study conducted in accordance with the principles stated in the Declaration of Helsinki, and ethical approval was obtained from the Ethical Committee of Antalya Training and Research Hospital (decision dated 13.02.2020, and numbered 3/25). Written informed consent was obtained from all participants.

Sample collection and biomarker tests

After an overnight sleep study following an eight- to 12-hour fast, venous blood samples were taken from individuals in both groups in the morning and transferred to gel biochemistry tubes that did not contain any preservatives or anticoagulant substances to avoid hemolysis. In the OSAS group, the blood sampling procedure was repeated 12 months after CPAP therapy. The collected blood samples were kept at room temperature for one hour and then centrifuged at 1,600 rpm for 10 minutes. Subsequently, the serum portion was collected and stored at -80 °C.

Serum concentration levels of nesfatin-1, salusin alpha and beta, and galectin-3 were determined using the enzyme-linked immunosorbent assay method according to the manufacturer's procedure (ELISA Kit, Elabscience®, Houston, TX, USA).

Treatment compliance and follow-up

The patients who received CPAP therapy were followed up every three months for one year, and their compliance with treatment was measured using the internal data stores of the CPAP device. Regular CPAP use was defined as the use of the device on 70% of nights from the initiation of therapy to the subsequent medical examination, with an average of four hours of device use per night [35].

Statistical analysis

Descriptive statistics were presented with frequency, percentage, mean, standard deviation, median, minimum, maximum, 25th percentile (Q_1), and 75th percentile (Q_3) values. In the analysis of categorical data, the Pearson chi-square test was used since the percentage of cells with an expected value of less than 5 was less than 20%. The assumption of normality was checked using the Shapiro-Wilk test. In the analysis of the difference between the numerical data of the two groups, the independent-samples t-test was employed if the data conformed to the normal distribution and the Mann-Whitney U test otherwise. For the comparison of the numerical data before and after the procedure, the Wilcoxon signed-rank test was utilized because the data did not comply with the normal distribution. Relationships between numerical data were evaluated with the non-parametric Spearman correlation test and the parametric Pearson correlation test. Analyses were performed with SPSS v. 23.0. $P < 0.05$ was considered statistically significant.

Results

Of the 44 patients participating in the study, 47.7% were in the control group, and 52.3% were in the patient group. Distribution of the chronic diseases in patients with OSAS is shown in Table 1. Nineteen patients with OSAS had various comorbidities, with the most common being obesity. Table 2 presents the comparison of gender, chronic disease status, age, body mass index (BMI), and polysomnography (PSG) AHI values between the control and patient groups. There were no statistically significant differences between the two groups in terms of the distribution of gender ($p = 0.599 > 0.05$). The rate of patients with chronic diseases was found to be 82.6% in the OSAS group. Upon comparing age, BMI (kg/m^2), and AHI values between the groups, statistically significant differences were observed ($p < 0.0001$). The mean age, BMI, and AHI values were higher in the OSAS group than in the control group.

OSAS group (n = 23)	
Chronic disease	19 (82.6)
Obesity	11 (47.8)
Hypertension	5 (21.7)
Asthma	4 (17.4)
COPD	4 (17.4)
DM	3 (13.0)
Rheumatoid arthritis	2 (8.7)
CAD	1 (4.3)
Hypothyroidism	1 (4.3)

Table 1: Distribution of the chronic diseases in OSAS group.

Abbreviations: OSAS, obstructive sleep apnea syndrome; COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus; CAD, coronary artery disease.

	Control group (n = 21)	OSAS group (n = 23)	p-value
	Mean ± SD (min-max)	Mean ± SD (min-max)	
	Median (Q_1-Q_3)	Median (Q_1-Q_3)	
Gender			
Female, n (%)	7 (33.3)	6 (26.1)	0.599[1]
Male, n (%)	14 (66.7)	17 (73.9)	
Chronic disease			
Absent, n (%)	21[a] (100)	4b (17.4)	<0.0001[1]
Present, n (%)	0[a] (0)	19[b] (82.6)	
Age (years)	34.9 ± 8.52 (26-53)	53.74 ± 8.68 (41-73)	<0.0001[2]
	32 (29-36)	52 (47-62)	
BMI (kg/m^2)	25.89 ± 1.35 (23.2-28.4)	32.3 ± 6.01 (21.7-39.8)	<0.0001[2]
	25.8 (25-26.7)	30.4 (27.5-38)	
AHI	1.43 ± 0.98 (0-3)	35.86 ± 11.52 (12.9-66.9)	<0.0001[2]
	1 (1-2)	38.2 (27.7-41.2)	

Table 2: Descriptive statistics of the OSAS and control groups.

[1]Pearson chi-square test, [2]Mann-Whitney U test. [a,b]Different letters in the same row indicate statistically significant differences in column values ($p < 0.05$). OSAS: obstructive sleep apnea syndrome, SD: standard deviation, BMI: body mass index, AHI: apnea-hypopnea index.

Table 3 shows the relationships between the AHI, BMI, and gender variables and the nesfatin-1, salusin alpha, salusin beta, and galectin-3 biomarker values in the OSAS group. Accordingly, there was a statistically significant negative relationship only between AHI and the nesfatin-1 value (r = -0.551, p = 0.006) (Figure 1).

		Nesfatin-1 pg/ml	Salusin alpha pg/ml	Salusin beta pg/ml	Galectin-3 ng/ml
AHI	r	-.551**	0.151	-0.215	-0.026
	p	0.006	0.491	0.325	0.906
	n	23	23	23	23
BMI (kg/m^2)	r	0.164	-0.236	0.087	0.037
	p	0.454	0.278	0.691	0.868
	n	23	23	23	23
Gender	r	0.131	0.206	0.134	0.152
	p	0.551	0.346	0.543	0.49
	n	23	23	23	23

Table 3: Relationship between the polysomnography AHI, BMI, and gender variables and the nesfatin-1, salusin alpha, salusin beta, and galectin-3 biomarker values in the OSAS group.

AHI: apnea-hypopnea index, BMI: body mass index, r: correlation coefficient, n: number of observations.

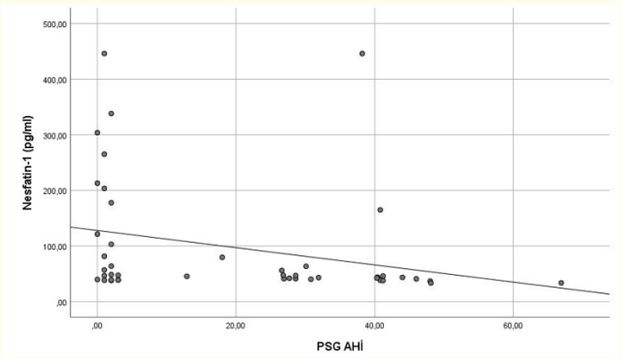

Figure 1: Relationship between the AHI score and nesfatin-1 (pg/ml).

The comparison of biomarker values between the groups is given in Table 4. There was a statistically significant difference between the nesfatin-1 values of the OSAS and control groups ($p = 0.025 < 0.05$). The mean nesfatin-1 value of the OSAS group was lower than that of the control group. The mean salusin alpha and beta values also statistically significantly differed between the two groups ($p = 0.049 < 0.05$ and $p = 0.002 < 0.05$, respectively). Lastly, the galectin-3 value of the OSAS group was statistically significantly lower compared to that of the control group ($p = 0.043 < 0.05$).

	Control group (n = 21)	**OSAS group (n = 23)**	
	Mean ± SD (min-max) Median (Q_1-Q_3)	Mean ± SD (min-max) Median (Q_1-Q_3)	p
Nesfatin-1 (pg/ml)	130.98 ± 120.21 (38.14-445.92)	67.67 ± 86.71 (33.56-445.81)	0.025[2]
	63.87 (39.81-203.53)	43.31 (40.32-47.61)	
Salusin alpha (pg/ml)	116.5 ± 105.21 (17.03-311.37)	61.47 ± 73.18 (14.31-311.97)	0.049[1]
	75.46 (23.46-211.94)	37.74 (27.73-52.06)	
Salusin beta (pg/ml)	176.13 ± 197.44 (16.39-692.53)	74.72 ± 201.34 (3.3-953.38)	0.002[2]
	72.2 (28.84-277.25)	21.42 (12.03-33.91)	
Galectin-3 (ng/ml)	7.63 ± 8.82 (0.63-32.87)	3.23 ± 6.6 (0.54-31.38)	0.043[2]
	3.06 (1.03-11.86)	1.29 (0.85-2.26)	

Table 4: Comparison of biomarker values between the groups.

[1]Independent-samples t-test, [2]Mann-Whitney U test. OSAS: obstructive sleep apnea syndrome, SD: standard deviation.

Table 5 presents the comparison of the biomarker values before and after CPAP treatment in the OSAS group. Accordingly, there were no statistically significant differences between the before and after CPAP treatment measurements of nesfatin-1, salusin alpha, salusin beta, and galectin-3 values ($p = 0.543 > 0.05$, $p = 0.201 > 0.05$, $p = 0.563 > 0.05$, and $p = 0.99 < 0.05$, respectively).

	Before CPAP treatment **n = 23**	**After CPAP treatment** **n = 23**	
	Mean ± SD (min-max)	Mean ± SD (min-max)	p
	Median (Q_1-Q_3)	Median (Q_1-Q_3)	
Nesfatin-1 (pg/ml)	67.67 ± 86.71 (33.56-445.81)	62.5 ± 68.84 (32.03-366.7)	0.543
	43.31 (40.32-47.61)	42.63 (38.47-53.32)	
Salusin alpha (pg/ml)	61.47 ± 73.18 (14.31-311.97)	48.69 ± 58.8 (13.79-290.07)	0.201
	37.74 (27.73-52.06)	32.47 (21.02-50.3)	
Salusin beta (pg/ml)	74.72 ± 201.34 (3.3-953.38)	43.01 ± 87.09 (3.04-426.34)	0.563
	21.42 (12.03-33.91)	21.11 (8.98-35.57)	
Galectin-3 (ng/ml)	3.23 ± 6.6 (0.54-31.38)	2.88 ± 5.13 (0.46-24.94)	0.99
	1.29 (0.85-2.26)	1.24 (0.65-2.93)	

Table 5: Comparison of before and after CPAP treatment biomarker values in the OSAS group.

All analyses were conducted using the Wilcoxon signed-rank test. CPAP: continuous positive airway pressure, OSAS: obstructive sleep apnea syndrome, SD: standard deviation.

Discussion

OSAS has been found to be potentially linked to hypertension and other cardiovascular complications, cerebrovascular events, metabolic events, and less well-known obstetric complications. Given the collective impact of these conditions, OSAS imposes a significant socioeconomic burden [36]. Therefore, considering OSAS and the associated systemic diseases, there is a need for diagnostic and prognostic biomarkers for the management of this disorder.

In this study, we measured serum nesfatin-1, salusin alpha and beta, and galectin-3 levels in patients with moderate and severe OSAS, evaluated their relationship with disease severity, and examined the changes in these biomarkers after CPAP therapy.

Nesfatin-1 is a protein that has been determined to have an anti-inflammatory effect in addition to being a satiety regulator [14,15]. Shen., *et al*. found nesfatin-1 levels to be significantly lower in patients with OSAS [37]. Araz., *et al*. detected a negative correlation between disease severity based on the AHI score and the nesfatin-1 level in OSAS (38). Similarly, we observed that the nesfatin-1 level was significantly lower in patients with OSAS and there was a significant negative correlation between the AHI score and nesfatin-1.

Salusin alpha and beta are bioactive peptides synthesized in the vascular endothelium and kidneys. Salusin alpha is an endogenous ACAT-1 inhibitor and exhibits antiatherogenic and anti-inflammatory effects, while salusin beta acts as a proatherogenic and pro-inflammatory molecule that promotes the proliferation of vascular smooth muscle cells and the formation of macrophage foam cells [39].

Salusin alpha levels have been reported to be lower in patients with coronary artery disease, acute coronary syndrome, and stable angina pectoris when compared to a healthy control group [40]. In a study investigating the effect of salusins on atherosclerosis, salusin beta did not have any effect, while salusin alpha had a preventive role in the pathogenesis of atherosclerosis by reducing total cholesterol [41]. In our review of the literature, we did not find any study examining salusin levels in patients with OSAS. There are some shared risk factors between OSAS and atherosclerosis, including obesity, advanced age, male gender, metabolic syndrome, smoking, elevated levels of high-sensitivity C-reactive protein, and insulin resistance. These parameters have been shown to be characteristic of both disorders [17-21]. Therefore, it seems noteworthy to investigate the relationship between salusins and OSAS. In the current study, we found that salusin alfa and beta levels significantly decreased in patients with OSAS.

Galectin-3 has been identified as a biomarker that is implicated in fibrosis and inflammation, contributing to the pathogenesis and advancement of heart failure. Additionally, it has been associated with increased morbidity and mortality [42]. Although there are a limited number of studies evaluating OSAS and galectin-3 together, Slouka., *et al*. reported serum level of galectin-3 levels in patients with OSAS did not differ significantly from healthy individuals [43]. Pusuroglu., *et al*. found that the serum galectin-3 level was significantly higher in patients with OSAS and showed significant association between serum galectin-3 concentrations and coronary atherosclerosis. They also found a positive correlation between OSAS severity and galectin-3 levels [44]. In our study, we found galectin-3 levels to be significantly lower in the OSAS group compared to the healthy control group, we did not detect a significant correlation between galectin-3 and disease severity. Kondratavičienė., *et al*. reported that they found galectin-3 levels higher in patients with OSAS and the serum galectin-3 level decreased significantly after three months of CPAP therapy [45]. In our study, a long-term, 12-month CPAP therapy we did not find any significant change in serum galectin-3 levels.

This study has certain limitations. Similar to the majority of individuals diagnosed with OSAS, a significant proportion of the patients included in our study presented with comorbidities. Since there were only a very few patients with OSAS who did not have any comorbidities, we were not able to compare the serum biomarker levels according to the presence of comorbidities; thus, we could not discuss the effect of chronic diseases on the investigated biomarkers. The other limitation of this study is the relatively small patient sample (those who used the CPAP device and had good compliance).

Conclusion

The data of our study show a clear negative correlation between nesfatin-1, salusin alfa, salusin beta, galectin-3 levels and OSAS. Furthermore, serum nesfatin-1 levels were negatively correlated with the presence of OSAS and OSAS severity. Serum nesfatin-1

level may be use of as a new biomarker for early detection and risk assesment of OSAS and its severity. CPAP treatment did not alter levels of these circulating markers. It is obvious that new biomarkers are needed to evaluate the effectiveness of CPAP therapy.

Acknowledgments

None.

Conflict of Interest

The authors declare that they have no conflict of interest.

Data Availability

The data that support the findings of this study are available upon request.

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