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SLEEP BREATHING PHYSIOLOGY AND DISORDERS • ORIGINAL ARTICLE

# Evaluation of apelin, chemerin, leptin, resistin and vaspin levels in patients with obstructive sleep apnoea syndrome

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## Abstract

**Background** The aim of this study was to investigate serum levels of apelin, chemerin, leptin, resistin, vaspin and advanced glycation end-products (AGEs) relationships with Obstructive Sleep Apnea Syndrome (OSA).

**Methods** 54 OSA patients and 34 healthy controls participated in this study. The levels of chemerin, leptin, resistin, vaspin and AGEs were determined in the serum by the ELISA method.

**Results** The chemerin median values were significantly higher *(p*=0,012) in the patients group when compared with the control. In contrast, the mean of AGEs in the patient's group was significantly decreased compared to the control group ($p=0.036$). A positive correlation was found between chemerin and apnea-hypopnea index (AHI) ($r=304$, $p=0.025$) and oxygen desaturation index (ODI) ($r=0.278$, $p=0.042$). A correlation was also observed among chemerin and haemoglobin ($r=0.228$, $p=0.032$) and iron ($r=0.339$, $p=001$). There was a positive correlation between apelin and C-reactive protein (CRP) ($r=0.226$, $p=0.034$). Leptin was positively correlated with body mass index (BMI), glucose, insulin, HbA1c, haemoglobin and iron.

**Conclusions** The result of this study demonstrate that chemerin and AGEs are related to OSA physiopathology. These two parameters may help in the diagnosis and progress of sleep apnea.

**Keywords** Obstructive sleep apnea · Apelin · Chemerin · Leptin · Resistin · Vaspin

## Introduction

Obstructive sleep apnea (OSA) is a widespread disorder with characterized specific symptoms such as recurrent cessation of airflow and desaturation during sleep. It has not been fully clarified, however, several risk factors have been determined in the pathogenesis of OSA. Morever obesity is the most known risk factor for OSA development, on the other hand, not all obese individuals develop OSA [1–3].

Adipokines are a group of bioactive polypeptides secreted by the white adipose tissue. These have important biological functions, such as the regulation of glucose, lipid metabolism and energy homeostasis. Evidence has revealed that some of these namely apelin, chemerin, leptin, resistin and vaspin are associated with obesity. The adipokines have been shown to have an important role in the development of obesity-related diseases such as OSA, insulin resistance, inflammation, hypertension, and cardiovascular and metabolic disorders [4]. Chemerin has been shown to exert pro- and anti-inflammatory properties in various lung disease models [5]. Chemerin also increases in different lung diseases, infectious, autoimmune, and age-related diseases. Due to its immunomodulatory role, it has emerged as an important therapeutic molecule. Leptin is an adipokine synthesised and secreted from many tissues, especially subcutaneous adipose tissue. Leptin, an appetite-suppressing hormone, is involved in oxidative stress, inflammation, thrombosis, and arteriosclerosis [6].

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Visceral adipose tissue-derived serpin (vaspin) is another adipokine related to the serine protease inhibitor family [7]. It has been shown that vaspin contributes to the development of insulin resistance (IR) and type 2 diabetes mellitus [8]. In the present study, vaspin was hypothesized to be a possible link between metabolic syndrome and OSA. Resistin is a 12 kDa protein that is related to systemic inflammation and is a potential biomarker for cardiovascular diseases. It has been reported that resistin increases in obese and diabetic patients and is associated with coronary artery disease (CAD) [9].

Abnormal glucose metabolism is associated with diabetes mellitus (DM), which involves complex organic reactions and the production of advanced glycation end-products (AGEs). These products have been noted to have relationships with various diabetic complications [6]. Production of AGEs causes different lesions and tissue damage in a large number of organs [7]. The lung is more target organ of AGEs because of its rich constitution of capillaries, collagen, and elastin fibers that are prone to deposit of AGEs [8].

Adipokines and AGEs have been studied in detail as they play an important role in many pathological and physiological processes [6]. In the present, we aimed to investigate the relationship between levels of chemerin, leptin, resistin, vaspin and AGEs in patients with OSA.

## Methods

### Study population

In this study, 88 individual participants were included in order to obtain an effect size of 0.20, 5% type I error, and 95% power level. GPower 3.1 was used to calculate the sample size [10]. A total of 107 consecutive patients who were admitted to our clinic because of complaints of snoring during sleep or respiratory disorder at sleep were evaluated at the sleep center in four months. All participants underwent polysomnography. Exclusion criteria were the presence of any known history of cardiovascular disease, cerebrovascular accident, heart failure, hypertension, and diabetes mellitus. The participants who had blood pressure higher than 140/90 or taking antihypertensive medications were considered hypertensive patients. Having fasting blood glucose levels > 126 mg/dl or current use of antidiabetic medication was defined as diabetes mellitus.

Nineteen of them were excluded because of cardiovascular diseases/hypertension ($n$ = 10), diabetes mellitus ($n$ = 6), and the presence of any other comorbid diseases ($n$ = 3). Thirty-four healthy controls were included regarding polysomnography reports (AHI < 5). Excessive daytime sleepiness and AHI ≥ 5 were defined as OSA [10]. Participants were divided into two groups: control ($n$ = 34) and OSA group ($n$ = 54).

Physical examination was performed and body mass index was measured. Daytime sleepiness was evaluated by using the Epworth Sleepiness Scale.

### Blood sampling

Serum samples were separated by centrifugation at 825 g for 10 min and then serums were kept at −80 °C until analysis. Serum levels of chemerin (Elabscience, China, Cat No: E-El-H0698), leptin (DRG International GmbH, 2017), resistin (Affymetrixe Bioscience Inc., 2016), vaspin (Elabscience, China, Cat No: E-EL-H1762) and AGEs (Elabscience, China, Cat No: E-EL-0102) were determined by ELISA using commercially available kits according to the manufacturer's protocols.

### Polysomnography

Full-night polysomnography was done with a 62-channel Embla N7000 device (Medcare Flage, Iceland). Specific signals of interest for sleep scoring included EEG, EOG, chin EMG, ECG, bilateral anterior tibial muscle EMG, nasal airflow, respiratory effort (thorax and abdomen movements), oxygen saturation, tracheal microphone, and body position. Sleep records were scored according to standard criteria of the American Academy of Sleep Medicine (AASM) [11]. Apnea was determined as a reduction of at least 90% in airflow amplitude for ≥ 10 s. Hypopnea is defined as a reduction of at least 30% in airflow for ≥ 10 s with an oxygen desaturation of ≥ 3% or accompanying arousal. Patients were categorized in terms of OSA severity as follows: an AHI < 5 events/h was accepted normal (control groups), ≥ 5 and < 30 were considered mild-to-moderate OSA, and ≥ 30 was accepted severe OSA [12]. Written informed consent was obtained from all participants. The study was performed according to the ethics guidelines of the Declaration of Helsinki, and the study protocol was approved by the Local Ethics Committee.

### Statistical analysis

In this study, analyses were conducted with 54 volunteers diagnosed with sleep apnea and 34 healthy volunteers who formed the control group. Patient results were evaluated with SPSS v. 25 (Statistical Package for the Social Sciences) program. Descriptive categorical variables were reported as n (%). Continuous numerical variables were expressed as mean ± standard deviation or median (minimum-maximum). Differences in continuous numerical variables between groups were assessed based on the normality of the

data distribution, using either Student's t-test or Kruskal-Wallis analysis. For comparisons of multiple groups, post-hoc Bonferroni correction was applied following One-way ANOVA or Kruskal-Wallis analyses. Correlation analyses were performed using Spearman's analysis. Binary logistic regression analysis was conducted to examine independent risk factors for disease development risk. A significance threshold was considered at $p<0.05$.

## Results

The demographic and laboratory data of the control and patients group are given in Tables 1 and 2. There were no difference between the groups in terms of age and gender. BMI level was significantly higher in the patient group comparede to control group ($p$=0001). Besides this, the levels of AHI, ODI, and T90 levels were significantly higher, whereas minimum oxygen saturation (min SpO2) levels were significantly lower in patients group compared to the control (Tables 1 and 2).

According to Tables 1 and 2, significant differences were seen for cholesterol, eGFR, RBC, HGB, HCT whereas no significant differences were found for iron, UIBC, FT3, MCHC, PDW, PLT, NE%, NE#, LY%, LY#.

There was a positive correlation between chemerin and AHI ($r$=304, $p$=0.025) and ODI ($r$=0.278, $p$=0.042). A correlation was also found among chemerin and haemoglobin ($r$=0.228, $p$=0.032) and iron ($r$=0.339, $p$=001) (Table 3). A positive correlation had been analysed between apelin and CRP ($r$=0.226, $p$=0.034). Leptin was positively correlated with BMI, glucose, insulin, HbA1c, haemoglobin and iron (Table 4; Fig. 1).

The median (min.-max.) chemerin value of the patient group was determined as 0.023 (0.020–0.369) ng/ml. The median (min.-max.) chemerin value of the control group was 0.008 (0.006–0.535) ng/ml. The chemerin value of the patient group was significantly higher than the control group ($p$=0.012) (Fig. 2).

The serum level of AGEs in the control was 2051.7±431.6 ng/ml whereas in the OSA group was 1837.3±469.9 ng/ml. The results of the patient group were found to be significantly lower ($p$=0.036) than the control group. No significant differences were found between the groups in related to levels of apelin, chemerin, leptin, resistin, and vaspin (Table 1).

In the analysis adjusted for age and gender, it was found that BMI, LDL and HbA1c was independent risk factors for OSAS (Table 5).

**Table 1** Biochemical parameters according to the groups

| | Control ($n$:34) | OSA ($n$:54) | $p$ values |
|---|---|---|---|
| | Mean±SD; Median (min-max) | Mean±SD; Median (min-max) | |
| AHI (events/hour) | | 15,4 (5–90,5) | |
| ODI (events/hour) | | 66,5 (7–516) | |
| Age (years) | 44,03±11,01 | 48,57±11,32 | 0,067 |
| Gender (male/female) | 22 (64.7) | 32 (59.3) | 0.609 |
| Resistin (ng/mL) | 5,12±1,88 | 4,65±2 | 0,279 |
| AGEs (AU) | 2051,69±431,59 | 1837,28±469,87 | 0,036 |
| Apelin (pg/mL) | 1,76 (0,66−4,46) | 1,82 (0,73−4,75) | 0,625 |
| Chemerin (pg/mL) | 0,01 (0,01−0,54) | 0,02 (0,02−0,37) | 0,012 |
| Vaspin (pg/mL) | 0,27 (0–0,75) | 0,28 (0,03−1,2) | 0,107 |
| Leptin (pg/mL) | 3,44 (0,25−11,83) | 4,22 (0,78−34,89) | 0,176 |
| BMI (kg/m²) | 23,1 (19–39,3) | 30,85 (23,5–47,2) | <0,001 |
| Glucose (mg/dL) | 88,5 (73–124) | 101 (76–125) | <0,001 |
| HbA1c (mmol/mol) | 5,3 (4,9−5,9) | 5,7 (5–5.9.9) | <0,001 |
| HbA1c IFCC (mmol/mol) | 34 (30–41) | 39 (3,3–84) | <0,001 |
| Cholesterol (mg/dL) | 177,71±46,03 | 204,5±47,64 | 0,011 |
| Triglyceride (mg/dL) | 117,5 (42–482) | 152 (44–377) | 0,002 |
| HDLC (mg/dL) | 45 (17–78) | 46 (31–90) | 0,554 |
| LDLC (mg/dL) | 104,4 (64,6–166) | 129,3 (41–206) | 0,053 |
| VLDLC (mg/dL) | 23,5 (8,4–96,4) | 30,4 (10,8–172,4) | 0,001 |
| eGFR (mL/min/1.73 m²) | 101,9±11,44 | 88,88±16,53 | <0,001 |
| Iron (µg/dL) | 84,62±38,09 | 87,78±36,84 | 0,7 |
| UIBC(µg/dL) | 269,18±62,3 | 262,81±72,78 | 0,674 |
| TProt (g/dL) | 7,4 (6,8–8,1) | 7,3 (6,4–8,5) | 0,329 |
| Urea (mg/dL) | 24 (14–43) | 27 (15–69) | 0,006 |
| Creatinine (mg/dL) | 0,8 (0,6−1,1) | 0,9 (0,7−1,6) | 0,012 |
| Uric acid (mg/dL) | 4,6 (3,1–8,6) | 5,7 (4,3–9,5) | <0,001 |
| IgE (IU/mL) | 27,6 (4,2–480,1) | 45,2 (0,45–2456,47) | 0,001 |
| TSH (µIU/mL) | 2,15 (0,31−6,33) | 1,59 (0,59−19,36) | 0,017 |
| FT3 (pg/mL) | 3,83±0,49 | 3,76±0,37 | 0,517 |
| Ferritin (ng/mL) | 24,7 (4,3–83,7) | 46,2 (2,5–295,4) | 0,002 |
| Insulin (µIU/mL) | 7,13 (2,54−20,48) | 12,59 (3,53–60,12) | <0,001 |
| HOMA-IR | 1,6 (0,5–5,26) | 3,12 (0,76−21,08) | <0,001 |

## Discussion

Sleep-disordered breathing is a common health problem, of which one of the most common is OSA. The present study investigated whether adipokines such as apelin, chemerin, leptin, resistin, vaspin and AGEs could be used as biomarkers for the diagnosis and efficacy of the OSA treatment. PSG is mostly used as the gold standard method in the diagnosis of the disease. However, this method is expensive and time-consuming and requires experienced personnel and special equipment. Therefore, the assessment of specific laboratory parameters, particularly adipokines, may contribute to the

**Table 2** Hematological parameters according to the groups

| | Control (*n*:34) | OSA (*n*:54) | *p* values |
|---|---|---|---|
| | Mean ± SD; Median (min-max) | Mean ± SD; Median (min-max) | |
| WBC | 6,65 (4,4–11,3) | 7,35 (5–12,7) | 0,017 |
| NE | 56,84 ± 6,79 | 56,26 ± 6,73 | 0,694 |
| NE# | 3,99 ± 1,07 | 4,43 ± 1,19 | 0,084 |
| LY | 33,47 ± 5,82 | 33,29 ± 6,48 | 0,894 |
| LY# | 2,34 ± 0,62 | 2,6 ± 0,7 | 0,08 |
| MO | 7 (4,5–14,4) | 7,45 (4,5–11,7) | 0,247 |
| MO# | 0,5 (0,3 − 0,9) | 0,6 (0,3–8,5) | 0,012 |
| EO | 1,45 (0,6–123) | 2,1 (0,5–10,5) | 0,019 |
| EO# | 0,1 (0–1) | 0,2 (0–1,3) | 0,015 |
| RBC | 4,85 ± 0,53 | 5,2 ± 0,53 | 0,004 |
| HGB | 13,53 ± 1,55 | 14,27 ± 1,72 | 0,042 |
| HCT | 41,2 ± 4,37 | 43,47 ± 4,6 | 0,024 |
| MCHC | 32,78 ± 0,79 | 32,78 ± 0,95 | 0,972 |
| PDW | 16,52 ± 0,52 | 16,59 ± 0,65 | 0,577 |
| PLT | 262 ± 63,16 | 263,7 ± 63,23 | 0,902 |
| MCV | 84,25 (65,6–98,8) | 85,2 (58,2–94,6) | 0,827 |
| MCH | 28,1 (20,3–32,5) | 28,4 (17,7–31,7) | 0,712 |
| MPV | 9,05 (7,4–11,5) | 8,6 (6,8–12,2) | 0,121 |
| PCT | 0,23 (0,17 − 0,32) | 0,22 (0,13 − 0,46) | 0,526 |
| RDW | 13,3 (12,2–17,4) | 13,65 (11,9–18,6) | 0,415 |

estimation of OSA risk in a clinical setting. Although these biomarkers cannot replace polysomnography, they may serve as supportive tools by providing preliminary information regarding disease severity and the likelihood of sleep apnea.

Generally, profiles of patients with OSA are associated with obesity. Obesity is a well known and an important factor causing upper airway blockage [13]. White adipose tissue function also plays an important role in the pathogenesis of OSA. In additions to physiological functions such as neutral fat storage, energy storage, energy supply and temperature regulation, adipose tissue is functions as an endocrine organ. Adipose tissue is able to secret numerous types of adipokine, including apelin, chemerin, leptin, resistin, vaspin. Hence, these adipokines affecting obesity can be used for diagnostic purposes and may play a role in the physiopathology of OSA. In the present study, there were two groups, consisting OSA and control groups. The average age of the groups was not significantly different. The mean BMI, glucose and HOMA-IR in the OSA groups were significantly higher than the control. Therefore, OSA

**Table 3** Correlation of Apelin, Chemerin, Vaspin with other parameters

| | Apelin | | Chemerin | | Vaspin | |
|---|---|---|---|---|---|---|
| | *r* | *p* | *r* | *p* | *r* | *p* |
| Age | 0.043 | 0.690 | 0.033 | 0.761 | 0.157 | 0.145 |
| BMI | −0.008 | 0.943 | 0.209 | 0.051 | **0.221** | **0.038** |
| AHI | −0.173 | 0.212 | **0.304** | **0.025** | 0.020 | 0.886 |
| ODI | −0.,195 | 0.158 | **0.278** | **0.042** | 0.046 | 0.740 |
| Glucose | −0.032 | 0.767 | 0.181 | 0.092 | 0.006 | 0.955 |
| HbA1c | 0.015 | 0.887 | 0.039 | 0.716 | 0.042 | 0.699 |
| Insulin | 0.005 | 0.962 | 0.002 | 0.985 | 0.001 | 0.995 |
| Cholesterol | 0.025 | 0.816 | 0.089 | 0.409 | 0.202 | 0.059 |
| LDL | 0.002 | 0.982 | 0.178 | 0.098 | 0.163 | 0.129 |
| Hemoglobin | 0.014 | 0.897 | **0.228** | **0.032** | 0.006 | 0.958 |
| Iron | −0.046 | 0.668 | **0.339** | **0.001** | 0.005 | 0.964 |
| CRP | **0.226** | **0.034** | −0.073 | 0.497 | 0.047 | 0.665 |

**Table 4** Correlation of Leptin, Resistin, ages with other parameters

| | Leptin | | Resistin | | AGEs | |
|---|---|---|---|---|---|---|
| | *r* | *p* | *r* | *p* | *r* | *p* |
| Age | 0.209 | 0.051 | 0.047 | 0.664 | −0.192 | 0.076 |
| BMI | **0.376** | **0.000** | 0.093 | 0.387 | 0.048 | 0.663 |
| AHI | −0.004 | 0.976 | 0.134 | 0.335 | −0.031 | 0.825 |
| ODI | −0.017 | 0.905 | 0.117 | 0.399 | 0.068 | 0.633 |
| Glucose | **0.246** | **0.021** | 0.044 | 0.684 | −0.055 | 0.615 |
| HbA1c | **0.228** | **0.032** | 0.011 | 0.919 | 0.029 | 0.791 |
| Insulin | **0.422** | **0.000** | 0.015 | 0.888 | 0.183 | 0.091 |
| Cholesterol | 0.135 | 0.211 | **−0.302** | **0.004** | 0.127 | 0.243 |
| LDL | 0.065 | 0.549 | **0.406** | **0.000** | **0.227** | **0.035** |
| Hemoglobin | **−0.528** | **0.000** | −0.163 | 0.130 | −0.078 | 0.473 |
| Iron | **−0.382** | **0.000** | 0.142 | **0.**186 | −0.118 | 0.278 |
| CRP | 0.190 | 0.076 | 0.100 | 0.354 | 0.055 | 0.618 |

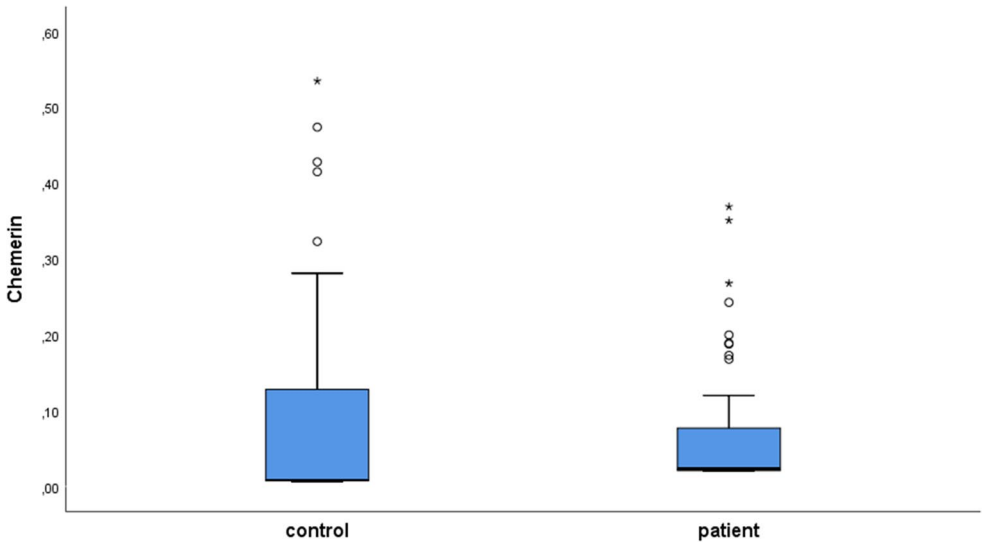


**Fig. 1** Please provide caption for Fig. 1

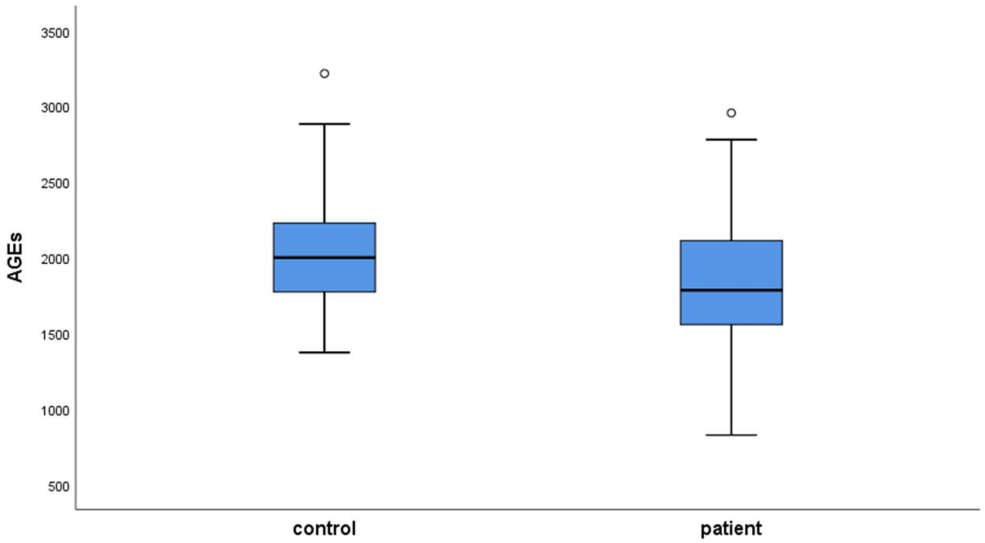


**Fig. 2** Please provide caption for Fig. 2

is likely to be closely associated with obesity related biomarkers. However, obese patients do not necessarily suffer from OSA while some patients with OSA have normal BMI. Similarly, serum levels of cholesterol and triglycerides significantly increased in OSA groups when compared with the control group. Increased fat in OSA group indicated that these were deposited around the upper airway. Therefore, it could be useful to reduce weight in order to relieve and treat OSA. In the present study, OSA group had significantly higher BMI than the control group [14].

Furthermore, in the present of this study, apelin, chemerin, leptin, resistin and vaspin adipokines were investigated for relationship with OSA. Many factors contribute to the pathogenesis of OSA. These factors have been classified as anatomical and nonanatomical causes [11]. A deterioration in the function of the pharyngeal dilator muscle leads to early awakening due to mild airway obstruction?.

Apelin is synthesised from adipose tissue by insulin action, and high plasma levels have been found in cases with obesity-related hyperinsulinism and insulin resistance [12]. It has been shown that intravenously administration of apelin increased glucose utilisation in skeletal tissue and significantly reduced blood glucose in mice. With this feature, apelin is a promising target in the management of insulin resistance [13]. In our study, apelin levels of the patients were higher than control groups but were not significantly

**Table 5** Multivariate regression analysis of variables

| | *p* value | OR (Odds Ratio) | % 95 Confidence Interval | |
|---|---|---|---|---|
| BMI | <0.001 | 1.706 | 1.286 | 2.264 |
| LDL | 0.023 | 1.035 | 1.005 | 1.067 |
| AGEs | 0.009 | 0.997 | 0.994 | 0.999 |
| HbA1c | 0.049 | 21.44 | 1.020 | 450 |

different (Table 2). This may be due to the small sample size and that only one research centre participated in the study.

White adipose tissue is a source and target for chemerin signalling. Chemerin is a secreted as protein and is thought to have a regulatory role in adipogenesis and adipocyte function. It shows different effects from cell to cell [14]. It increases insulin-dependent glucose uptake in fat cells [15], whereas it causes insulin resistance in muscle cells [16]. Xu et al. had found that chemerin levels were significantly higher in patients with severe OSA after PSG compared to the control group [17]. In addition, they found that plasma chemerin was higher after sleep than before sleep. They also showed that these levels were associated with anthropometric measurements, including BMI and AHI values. As a result, they emphasised that chemerin levels increased with OSA and were also associated with obesity. Another study performed by Feng et al. [18] found that serum chemerin levels were significantly higher in patients diagnosed with severe OSA than in patients with mild-to-moderate OSA and healthy controls. In our study, the chemerin levels of the patient were significantly increased compared to the control groups therefore, the results of our study are in parallel with these studies. In addition, OSA group had signifinat higher BMI value than control group (Table 2). Additionally, regression analysis showed that high BMI increased the risk of disease by 1.7 times.

Some studies have shown that serum vaspin levels contribute to the development of IR and type 2 diabetes mellitus [8]. At the same time, it has been found to cause changes in insulin resistance-related genes and levels of leptin, glucose transporter, adiponectin, and resistin. The present study supports this observation that serum glucose, HbA1c and HOMA-IR levels were statistically increased the OSA group when compared to the control group.

Another adipokine is leptin has which has important biological function as suppressing appetite, is involved in oxidative stress, inflammations, thrombosis and arteriosclerosis [13] Leptin increases ventilation response by the nervous system and leptin resistance in obesity could be diminished the ventilatory response and may play an important role in the apnea-hypopnea index (AHI). Studies have estimated that leptin plays an important role in severe OSA. On the other hands, it is unclear whether leptin could be a biomarker for OSA. A study conducted by Li et al. which reported that individuals diagnosed with OSA had significantly increased level of serum leptin when compared to the healthy participants. In additions, a positive correlation was reported between leptin and the AHI [2]. Patial et al. reported that serum leptin is associated with the severity of OSA. Moreover, it has also been reported that serum leptin levels exert a strong predictive value for the occurrence of OSA. The results of this study have concluded that no significant changes in serum leptin were present between the control and OSA groups. However, a statistically significant positive moderate correlation was observed between leptin and BMI [19].

It has been reported that resistin is a marker of inflammation and increases in paralell with increased OSA severity. It may be possible be that intermittent hypoxia causes epigenetic changes in resistin gene that decreases its expression [9]. In the present study, no significant differences were found between the control and OSA individuals in the level of serum resistin. However, a moderate negative correlation was observed between resistin and cholesterol. Similarly, a moderate negative relationship was seen between resistin and LDL (Table 4).

Some studies have reported that AGEs displays a positive correlation with abnormal glucose metabolism. These products have been reported to be associated with a various diabetic complications [20]. It has been reported that AGEs increased in chronic obstructive pulmonary disease [21]. A study performed by Tan et al. revealed that serum level of AGEs increased in patients with OSA when compared with the control individuals. Also, this study revealed that serum AGEs values are associated with a biochemical marker of oxidative stress in the night-time desaturation interval in patients with OSA, but not with fasting glucose levels. However, it has been reported that the higher serum AGEs levels in patients with OSA when compared tohealth indiviuals remains controversial [5]. In the present study, serum AGEs levels significantly decreased in OSA individuals when compared with the control group. In additions, a weak positive correlation was found between AGEs and LDL (Table 4).

In conclusion, according to the results of the present study, significant differences were observed in chemerin and AGEs parameters in OSA individuals when compared with control groups. These adipokines may be applicable biomarkers for the diagnosis and efficacy of the OSA treatment. However, the relationship between obesity and OSA is very complex and more studies are needed to further validate the results reported in this study.

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**Data availability** The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

## Declarations

**Ethical approval** All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional an/or national research committee and with the 1964 Helsinki declaration ans its later amendments or comparable ethical standards. Local ethics committee approved the study protocol.

**Informed consent** Informed consent was obtained from all participants included in the study.

**Conflict of interest** All authors (ERO, MAY, NS, AAH) certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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