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The serum leptin level in non-obese patients with obstructive sleep apnea

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ABSTRACT

Objective: This study aimed to determine the association between the severity of obstructive sleep apnea (OSA) and the serum leptin level in non-obese OSA patients.
Methods: This prospective case-control study included non-obese OSA patients that presented with sleep-related disturbances and underwent polysomnography (PSG) between April 2015 and June 2016. The serum leptin level was measured and its relationship to PSG parameters was investigated.
Results: The study included 73 OSA patients (20 female and 53 male) with a mean age of 41.1 ± 11.5 years and mean body-mass index (BMI) of 26.4 ± 2.7 kg m^{-2}. The serum leptin level in 44 patients with moderate/severe OSA (AHI ≥15) was 3.4 ± 2.6 ng mL^{-1}, versus 4.5 ± 3.8 ng mL^{-1} in 29 patients with snoring/mild OSA (AHI <15) (P = 0.20). There were not any correlations between any of the PSG parameters and the serum leptin level, but there was a significant correlation between the leptin level and BMI (r = 0.345, P < 0.01).
Conclusion: The serum leptin level does not differ significantly between non-obese OSA patients with moderate/severe and snoring/mild OSA. Obesity is the primary factor associated with the serum leptin level.

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1. Introduction

Obstructive sleep apnea (OSA) is characterized by repeated sleep-related upper airway obstruction and cessation of airflow, leading to intermittent hypoxia and hypercapnia. OSA patients have an increased risk of cardiovascular (CV) disease and coagulation abnormalities [1,2]. The underlying mechanism of OSA is not precisely known. It has been hypothesized that chronic intermittent hypoxia – leading to pro-inflammatory cytokine expression, oxidative stress, and endothelial dysfunction – plays an important role in the pathogenesis of OSA, and the association between OSA and CV disease [3].

Leptin is a protein hormone that is structurally similar to cytokines [4]. It is primarily produced by white adipose tissue and plays a role in increased energy expenditure and loss of appetite, thusly inducing weight loss [5]; however, the serum leptin level was reported to be high in obese patients and was attributed to a leptin-resistant state [6]. Moreover, leptin has a pro-inflammatory regulator effect on cytokine expression [7].

Although elevated leptin in OSA patients has been reported [5,8], the precise nature of the relationship between OSA and the leptin level remains unclear because of the confounding role of obesity. Possible causes of a high leptin level in OSA patients include the following: 1. Leptin resistance in obese individuals leading to hyperleptinemia (obesity is an essential feature of

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OSA patients); 2. Repeated airflow cessation leading to chronic intermittent hypoxia and inflammation, which induce leptin production by adipose tissue via inflammatory cytokine action; 3. Repeated arousals leading to sympathetic nervous system activation, which might be related to a high leptin level in OSA patients.

As both obesity and OSA are systemic inflammatory processes, and given the fact that most OSA patients are obese, it is not clear which (obesity or OSA) is the primary inducer of a high leptin level in OSA patients. As such, the present study aimed determine the relationship between the severity of OSA and the serum leptin level in non-obese OSA patients (so as to eliminate the effect of obesity).

2. Materials and methods

2.1. Participants

The study included 73 patients who were prospectively recruited from those who presented to Hacettepe University, School of Medicine, Department of Otolaryngology, Ankara, Turkey, with sleep-related disturbances between April 2015 and June 2016. The Epworth sleepiness scale was used to assess symptoms of daytime sleepiness. After complete physical examination and ENT examination, including fiberoptic nasopharyngoscopy and Müller's maneuver, all patients underwent standard overnight polysomnography (PSG). Inclusion criteria were a BMI <30 kg m^{-2} (non-obese), age 18–60 years, and an Epworth score >10. According to PSG findings, patients with an apnea-hypopnea index (AHI) ≥ 15 were included in the moderate/severe OSA group and patients with an AHI <15 were included in the snoring/mild OSA group.

Patients with comorbid diseases, such as cardiopulmonary disease, chronic kidney disease, cancer, endocrinological disease, and neurological disease, congenital head and neck abnormalities, acute or chronic infections (including both systemic and local head and neck infection) were excluded from the study. In addition, smokers and alcohol abusers were excluded from the study. None of the patients included in the study were using steroids (inhaled, nasal, or oral) or non-steroidal anti-inflammatory drugs (NSAIDs). None of the patients had previously been diagnosed with OSA or had undergone surgery for OSA. Written informed consent were obtained from all patients and the study protocol was approved by the Hacettepe University Ethics Committee (GO 15/277).

2.2. Polysomnography

Computerized overnight PSG was performed using an Embla S4500 in our department's sleep laboratory. Electroencephalograms, electrocardiograms, electromyograms, and electrooculograms were recorded using surface electrodes. Nasal airflow was assessed via an oronasal thermistor. Thoracoabdominal movements were recorded using inductive plethysmography. Arterial oxyhemoglobin saturation was monitored using a finger probe.

Apnea was defined as total cessation of airflow for ≥ 10 s. Hypopnea was defined as a reduction in airflow signal of $\geq 30\%$ for ≥ 10 s, followed by a $\geq 4\%$ decrease in oxyhemoglobin saturation or arousal episodes. AHI was defined as the total number of apnea and hypopnea events (n) per hour. The apnea index (AI) was defined as the total number of apnea events (n) per hour and total apnea time was calculated by multiplying the number of apnea events during total sleep time by mean apnea event duration. The oxygen desaturation index (ODI) was defined as the number of times oxyhemoglobin saturation dropped to $<90\%$ per hour of sleep. Sleep stages and respiratory events were scored in accordance with American Academy of Sleep Medicine (AASM) 2013 scoring criteria [9].

2.3. Serum leptin measurement

Blood samples were obtained between 07:00 and 08:00 a.m. after overnight fasting and polysomnography. The samples were immediately sent to Hacettepe University, Department of Biochemistry, centrifuged at 3000 rpm for 10 min at 4 °C, and then stored at −80 °C until analysis. When the targeted number of patients was reached, all the samples were analyzed using a solid-phase EASIA immunoassay kit (DIAsource ImmunoAssays S.A., Belgium). The detection limit of the assay was 0.04 ng mL^{-1} and the intra-assay coefficient of variation was 3.5%.

2.4. Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows v.23.0 (IBM Corp., Armonk, NY) and Microsoft Excel (Microsoft Corp., Redmond, WA). The normality of the distribution of data was determined using the Kolmogorov–Smirnov test; all parameters were normally distributed. Descriptive analysis was performed and means were compared between groups using the t test. Correlation analysis was performed using Pearson's correlation test. The level of statistical significance was set at $P < 0.05$.

3. Results

Among the 73 patients, 53 (72.6%) were male and 20 (27.4%) were female. Mean age of the patients was 41.1 ± 11.5 years and mean BMI was 26.4 ± 2.7 kg m^{-2}. The mean serum leptin level was 3.9 ± 3.2 ng mL^{-1}. Mean polysomnographic parameter values are shown in Table 1.

According to AHI values and Epworth symptom scores, 44 patients (14 with an AHI ≥ 15 and <30, and 30 with an AHI ≥ 30) were in the moderate/severe OSA group and 29 (13 with an AHI <5, and 16 with an AHI ≥ 5 and <15) were in the

Table 1
Mean polysomnographic parameter values.

Patient (n = 73)	Range	Mean
AHI (n h^{-1})	0.6–107.2	24.5 ± 22.4
AI (n h^{-1})	0–100.6	13.8 ± 18
Total apneic time (min)	0–250	27.4 ± 45.4
Mean oxygen saturation (%)	90–97.9	95.1 ± 1.44
Mean time with $<90\%$ oxygen saturation (min)	0–130	5.1 ± 17.2
ODI (n h^{-1})	0–103	13.7 ± 17.8

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