Research Article

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Nocturnal sweating in obstructive sleep apnoea and its association with sleep parameters

Güray Koç[1], Kübra Mehel Metin[2], Bülent Devrim Akçay[3], Ömer Karadaş[1], Refah Sayın [4], Sinan Yetkin[3]

[1]Clinic of Neurology, Gülhane Training and Research Hospital, Ankara, Turkey
[2]Clinic of Neurology, Ankara Training and Research Hospital, Ankara, Turkey
[3]Clinic of Psychiatry, Gülhane Training and Research Hospital, Ankara, Turkey
[4]Department of Neurology, Ufuk University, School of Medicine, Ankara, Turkey

Abstract

Objective: Nocturnal sweating is a condition frequently encountered in obstructive sleep apnoea (OSA) patients. This study aimed to examine the relationship between nocturnal sweating and sleep parameters in patients with and without OSA.

Methods: A total of 355 patients were included in this study. The patients were separated into two groups: group 1, consisting of patients with OSA and group 2, the control group, which contained patients without OSA. The presence of nocturnal sweating and its effect on sleep parameters were obtained by polysomnography and examined.

Results: In patients from group 1 that had nocturnal sweating, the rapid eye movement (REM) sleep percentage (12.80%, min–max: 0.40–29.60 vs 14.00%, min–max: 1.10–29.40; p=0.034), and REM episode (3, min–max: 1–6 vs 4, min–max: 1–6; p=0.002) were significantly lower, and the apnoea-hypopnoea index (AHI) (26.70, min-max:0-107.80 vs 17.40, min-max: 0-108.40; p=0.04) during the REM period was significantly higher compared to the patients from group 2 without nocturnal sweating. There was no difference in nocturnal sweating between group 1 and group 2.

Conclusion: This study indicates that the REM sleep period may be suppressed due to high sympathetic activity in relation to nocturnal sweating. Moreover, the high AHI detected during the REM sleep period also indicates the association between increased respiratory events and nocturnal sweating during this period.

Keywords: Apnoea-hypopnoea index, obstructive sleep apnoea, rapid eye movement, nocturnal sweating, sympathetic activity

INTRODUCTION

Nocturnal sweating is a frequently encountered condition and intense nocturnal sweating is frequently observed in patients with obstructive sleep apnoea (OSA). Studies have shown there is a subjective decrease in nocturnal sweating after positive airway pressure (PAP) therapy (1-4). Nocturnal sweating may cause problems for the patient and his/her bed partner and impair the patient's quality of life (1). However, a small number of patients have reported nocturnal sweating to their doctors. There are several possible causes for nocturnal sweating: malign conditions, infections, endocrine and neurologic disorders, menopause, panic attacks, room temperature, and thick sleeping clothes, as well as sleep disorders such as OSA (5, 6). Nocturnal sweating negatively impacts daily life activities, especially since they cause increased daytime fatigue and sleep problems (5). There are several unknown factors related to the causes, evaluation, and treatment of nocturnal sweating.

Sweating is controlled by the sympathetic nervous system in the skin. The main function of sweating is to increase heat loss and ensure thermoregulation. Body temperature has a considerable effect on sleep architecture; while a slight decrease in temperature increases deep sleep, a slight increase in temperature causes wakefulness and changes in sleep stages (7). Thermoregulation changes during sleep stages, and it is less effective during the rapid eye movement (REM) sleep period. However, the sweating frequency in healthy individuals is less during the REM period when compared to the non-rapid eye movement (NREM) period, and sweating occurs most frequently in slow-wave sleep (8).

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Corresponding Author: Güray Koç **E-mail:** gurayerhan@gmail.com **Submitted:** 10 September 2018 **Accepted:** 30 January 2019

Obstructive sleep apnoea is characterized by repetitive closing of the pharyngeal airway during sleep, and it is related to oxygen desaturation and arousal. Disturbed sleep and increased daytime sleepiness are frequently reported in patients with OSA (9). Nocturnal sweating generally occurs in the neck and the upper part of the body in patients with OSA. While the rate of nocturnal sweating was 34% in patients with OSA, it decreased to 12% with PAP therapy. The patients in which nocturnal sweating did not subside, were younger and had more severe OSA (10). Furthermore, in another study, no relationship was found between the severity of OSA and nocturnal sweating (11).

Untreated OSA patients have increased sympathetic activity both while asleep and awake, and this condition is indicated by the direct record of muscle sympathetic nerve activity and the catecholamine level in plasma and urine (12). Depending on sympathetic activity, reports have shown that nocturnal sweating is greater in patients with OSA (13). However, to the best of our knowledge there is no data about the relationship between nocturnal sweating and stages of sleep, especially in the REM stage.

This study aimed to examine the relationship between nocturnal sweating and sleep parameters in patients with OSA and in patients without OSA and with simple snoring.

METHODS

The data of the patients who had undergone polysomnography, with a pre-diagnosis of OSA at Gülhane Sleep Research Centre between January 2013 and December 2015, were retrospectively examined. According to the polysomnography results, the patients were separated into two groups: group 1, which contained patients with OSA and group 2, the control group, which consisted of patients without OSA. The patients from group 1 (with OSA) were further separated into severe, moderate, and mild groups. The data of the patients 18 years of age and older were included in the study. The patients answered a health history questionnaire about endocrinopathy, hypertension, diabetes, cardiovascular disease and coronary artery disease. The weight and height of the patients was received, and their body mass index (BMI) was calculated. The results of the Epworth sleepiness scale were evaluated. Information about the presence of nocturnal sweating as well as frequency (the number of days with nocturnal sweating in a week) was recorded in the patients' data records and used for this study. Frequent nocturnal sweating was defined as reporting nocturnal sweating ≥ 3 times a week (13). The patients were also divided into groups with regards to the presence of frequent nocturnal sweating. Patients using medication affecting the central nervous system such as antidepressant, antipsychotic and antiepileptic drugs were excluded from the study due to drugs' potential to affect sleep parameters. Also, patients with diseases that can interfere with autonomic functions such as parkinsonism, stroke, dementia, brain injury, multiple sclerosis and epilepsy were excluded from the study. The relationship between nocturnal sweating in patients and the presence and severity of OSA was examined. This retrospective study was approved by the Ankara Numune Training and Research Hospital Ethical Committee (22.11.2017/1544). It was conducted in accordance with the Declaration of Helsinki. As this is a retrospective study, there is no need for patient consent.

The polysomnography records were taken by a Grass Comet Plus AS40 branded polysomnograph device (Natus Neurology Incorporated, Middleton, WI, USA). Electroencephalography (F3-M2, C3-M2, O1-M2, F4-M1, C4-M1, O2-M1), electrooculography, submental electromyography (EMG), bilateral anterior tibialis EMG, and electrocardiography recordings were performed in accordance with the American Academy of Sleep Medicine (AASM) criteria. Respiratory inductive plethysmography belts recorded chest and abdominal movements. Airway flow was evaluated with a nasal airway and thermistor. Pulse and oxygen desaturation were measured by a finger probe oximeter. The body position was ensured by a sensor attached to the chest belt. The records were obtained by experienced technicians. Sleep scoring (in 30-second periods) and respiratory events, during sleep, were conducted according to the AASM criteria version 2.2, which was the latest version at the time of scoring (14). Sleep and respiratory events were scored manually. A respiratory event was scored as an apnoea when both of the following criteria were met: 1) There was a drop in the peak signal excursion by ≥ 90% of pre-event baseline using an oronasal thermal sensor, 2) The duration of the ≥ 90% drop in sensor signal was ≥ 10 seconds. A respiratory event was scored as a hypopnoea if each of the following criteria were met: 1) The peak signal excursions dropped by ≥ 30% of pre-event baseline using nasal pressure; 2) The duration of the ≥ 30% drop in signal excursion was ≥ 10 seconds; 3) There was a ≥ 3% oxygen desaturation from pre-event baseline or the event was associated with an arousal. The apnoea-hypopnoea index (AHI) was determined by counting the number of apnoeas and hypopnoeas per hour during sleep. Patients with an AHI value ≥ 5 were determined to have OSA. Obstructive sleep apnoea severity was determined using four AHI cut-off points: AHI <5/h, AHI 5 to <15/h, AHI 15 to <30/h, and AHI 30/h (15). All sleep records were evaluated by a certificated sleep doctor.

Statistical Analysis

Statistical analyses were performed with the Statistical Package for the Social Sciences for Windows, version 23.0 (SPSS IBM Corp.; Armonk, NY, USA). The Kolmogorov-Smirnov test was used to assess whether the data exhibited a normal distribution. The data that exhibited a normal distribution were presented using the mean and standard deviation. The data that did not exhibit a normal distribution were presented using the median and minimum-maximum values. Univariate analyses to identify variables associated with nocturnal sweating in OSA patients was investigated using Chi-squared,

Student's T and Mann-Whitney U tests, where appropriate. For multivariate analysis, the possible factors identified with univariate analyses were further entered into logistic regression analysis to determine independent factors of nocturnal sweating. Hosmer-Lemeshow goodness of fit statistic was used to assess the model fit. A 5% type-I error level was used to infer statistical significance. Graphics were obtained using MedCalc trial version (MedCalc Software, Ostend, Belgium).

Figure 1. Flowchart of patient selection

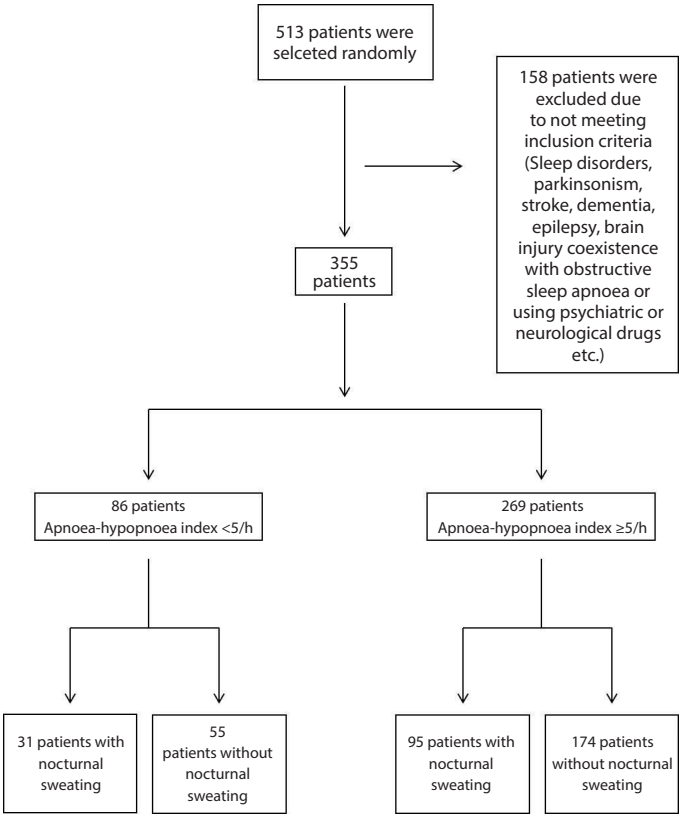


Table 1. Characteristics of the patients with and without OSA

	Patients with OSA (n=269)	Patients without OSA (n=86)	p
Gender (F/M)	83/186	24/62	0.604
Age (years)	45.57±11.24	36.81±11.22	<0.001
BMI (kg/m^2)	29.06 (18.67-60.41)	27.16 (19.10-58.27)	<0.001
Epworth sleepiness scale	10 (1-24)	10.5 (1-21)	0.754
Comorbid disease %	53.16%	38.37%	0.017
Nocturnal sweating	35.31%	36.04%	0.902

Significant findings (p<0.05) are shown in italics
Parametric data are shown as mean ± standard deviation, nonparametric data are shown as median (minimum-maximum)
OSA: obstructive sleep apnoea; F: female; M: male; BMI: body mass index; Comorbid disease: Presence of cardiovascular disease, defined as a doctor diagnosis of coronary artery occlusion (myocardial infarction or heart attack), heart failure. Hypertension and diabetes were defined as a doctor diagnosis and treatment with medication

RESULTS

In total, 355 patients were included in the study, where 158 patients, who met exclusion criteria as described above, were excluded from the study. Flowchart of patient selection for the study is shown in Figure 1. The age, gender, BMI, Epworth sleepiness scale, the presence of comorbid diseases in the patients included in the study and information on whether there was nocturnal sweating are indicated in Table 1. There was no difference in gender, the presence of nocturnal sweating and Epworth sleepiness scale values between group 1 and group 2. In group 1, the presence of a comorbid disease, BMI and age were found to be significantly higher compared to group 2 (Table 1).

Table 2. Sleep parameters in OSA group according to the presence of nocturnal sweating

	Nocturnal sweating (+) (n=95)	Nocturnal sweating (-) (n=174)	p
Gender (F/M)	35/60	48/126	0.116
Age (years)	47.77±10.98	44.37±11.24	0.017
Comorbid disease (%)	56.84%	51.15%	0.371
BMI (kg/m^2)	29.72 (21.46-60.41)	28.65 (18.67-48.83)	0.002
Epworth sleepiness scale	11 (1-22)	10 (1-24)	0.047
Sleep latency (minutes)	20.50 (2.50-247.50)	18.00 (0-237.50)	0.216
REM latency (minutes)	109.50 (10.00-408.50)	99.75 (4.00-380.00)	0.241
Sleep efficiency (%)	84.80 (28.70-96.00)	86.50 (39.90-98.80)	0.080
Sleep stage 1 (%)	21.80 (1.04-84.90)	20.65 (0.80-89.30)	0.253
Sleep stage 2 (%)	50.60 (2.00-89.30)	50.60 (3.60-89.80)	0.940
Sleep stage 3 (%)	10.30 (0-36.30)	11.70 (0-36.70)	0.166
REM sleep (%)	12.80 (0.40-29.60)	14.00 (1.10-29.40)	0.034
REM episode (N)	3 (1-6)	4 (1-6)	0.002
AHI TST	20.8 (5.00-100.80)	17.65 (5.00-128.80)	0.194
AHI REM	26.70 (0-107.80)	17.40 (0-108.40)	0.040
PLMI	4.60 (0-151.50)	4.70 (0-69.80)	0.941
Total sleep time under the 90% oxygen saturation (%)	22.65 (0-99.10)	11.20 (0-98.00)	0.013

Significant findings (p<0.05) are shown in italics
Parametric data are shown as mean ± standart deviation, nonparametric data are shown as median (minimum-maximum)
OSA: obstructive sleep apnoea; F: female; M: male; BMI: body mass index; REM: rapid eye movement; N: number; PLMI: periodic leg movement index; AHI: apnoea-hypopnoea index; TST: total sleep time

Group 1 was divided into two groups, with regards to the presence of nocturnal sweating and examined based on demographic information and sleep parameters. In the OSA group with nocturnal sweating, the age, BMI and Epworth sleepiness scale, the AHI during the REM period and the duration in which the oxygen saturation was lower than 90% during the total sleep time were found to be significantly higher, and the REM period and the number of REM episodes were found to be significantly lower compared to the OSA group without nocturnal sweating ($p<0.05$). The difference in the number of REM episodes is shown in Figure 2. No difference was observed in terms of gender, the presence of comorbid diseases, NREM sleep stages, sleep efficiency and total AHI between OSA patients with and without nocturnal sweating. The findings are indicated in Table 2.

Table 3. Independent risk factors on nocturnal sweating in OSA group

Risk Factors	OR (95% CI)	p
REM episode	0.769 (0.615-0.961)	0.021
BMI	1.051 (1.000-1.104)	0.049
Epworth sleepiness scale	1.049 (0.997-1.104)	0.067
F=17.381 p<0.001	Percentage correct 66.4%	

Significant findings ($p<0.05$) are shown in italics

OSA: obstructive sleep apnoea; BMI: body mass index; REM: rapid eye movement

To determine independent factors of nocturnal sweating in OSA patients, logistic regression analysis was used. Nocturnal sweating was found to be a dependent variable and the age, BMI, Epworth sleepiness scale, REM period, REM episode and AHI during the REM period were found to be independent variables. As a result, the effects of REM episode and BMI on nocturnal sweating were found to be statistically significant ($p<0.05$). The Odds ratio is presented in Table 3.

Table 4. Sleep parameters in group 2 according to the presence of nocturnal sweating

	Nocturnal sweating (+) (n=31)	Nocturnal sweating (-) (n=55)	p
Gender (F/M)	5/26	19/36	0.083
Age (years)	33.00±10.93	38.96±10.90	0.007
Comorbid disease (%)	29.03%	43.63%	0.249
BMI (kg/m^2)	28.07 (19.10-52.12)	26.53 (20.62-58.27)	0.950
Epworth sleepiness scale	10 (1-21)	12 (1-21)	0.695
Sleep latency (minutes)	15.50 (1.00-120.50)	20.50 (2.00-200.00)	0.197
REM latency (minutes)	84.50 (38.50-229.40)	100.50 (11.00-298.50)	0.087
Sleep efficiency (%)	92.40 (52.4-98.2)	86.8 (45.7-97.7)	0.089
Sleep stage 1 (%)	10.40 (4.50-30.50)	12.70 (4.40-41.70)	0.128
Sleep stage 2 (%)	55.90 (37.30-71.60)	54.10 (27.40-75.80)	0.173
Sleep stage 3 (%)	15.40 (6.90-29.50)	15.90 (0.50-30.60)	0.826
REM sleep (%)	15.20 (4.60-28.20)	15.60 (2.70-31.60)	0.836
REM episode (N)	4 (2-6)	3 (1-6)	0.086
AHI TST	2.00 (0-4.60)	1.70 (0-4.30)	0.870
AHI REM	2.30 (0-18.80)	2.80 (0-27.00)	0.622
PLMI	2.40 (0-24.60)	2.50 (0-63.30)	0.882

Significant findings ($p<0.05$) are shown in italics

Parametric data are shown as mean ± standart deviation, nonparametric data are shown as median (minimum-maximum)

F: female; M: male; N: number; BMI: body mass index; REM: rapid eye movement; PLMI: periodic leg movement index; AHI: apnoea-hypopnoea index; TST: total sleep time

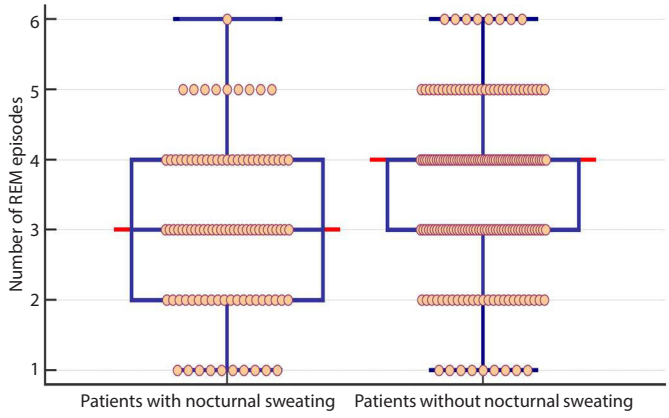
Group 2 was also divided into two groups based on the presence or absence of nocturnal sweating and examined with regards to demographic information and sleep parameters. Age was found to be significantly lower in the group with nocturnal sweating ($p=0.007$). There was no difference in regards to gender, comorbid disease, BMI, sleep activity, sleep stages, AHI in general and during the REM period ($p>0.05$). These findings are presented in Table 4.

DISCUSSION

The major findings of this study were the following: 1) AHI values during the REM period were significantly higher in OSA patients with nocturnal sweating compared to those without nocturnal sweating; 2) the duration of the percentage of REM sleep stage as well as the number of REM episodes was significantly lower in OSA patients with nocturnal sweating compared to those without nocturnal sweating. Also, patients with nocturnal sweating were older, overweight and sleepier compared to those without nocturnal sweating in the OSA group.

Figure 2. REM episodes of OSA patients with nocturnal sweating using combined box-and-whisker and dot plot graphic

REM: rapid eye movement; OSA: obstructive sleep apnoea



However, patients with nocturnal sweating were younger compared to those without nocturnal sweating in the control group. As expected patients were older, overweight and had more comorbid disease in the OSA group compared to the control group.

Arnardottir et al. indicated that individuals with nocturnal sweating among OSA patients had a lower REM sleep percentage and there was an increase in the REM sleep with PAP treatment (2). Supporting this finding, Arnardottir et al. found a negative relationship between nocturnal sweating and REM sleep, and also found a negative correlation between the electrodermal activity index measuring the activation of eccrine sweat glands, which can objectively evaluate sweating and the REM percentage (2). We also found that the REM sleep percentage was lower in OSA patients with nocturnal sweating. Additionally, we found that the REM sleep stage AHI was higher in OSA patients with nocturnal sweating in our sample. The REM sleep period may be suppressed by high sympathetic activity, and as a result, the REM sleep period may be lower due to an increase in the sympathetic activity in untreated OSA patients (2). It was demonstrated in animal models that the sympathetic activity plays a role in the regulation of the REM sleep period, and both noradrenaline agonists and reuptake inhibitors suppress REM sleep (16, 17). Supporting this, we also found that REM episode values were lower in nocturnal sweating patients. The negative relationship between hot flush in women during menopause and REM sleep period supports this finding as well (18). The findings of this study indicated that nocturnal sweating during sleep affects sleep architecture in patients with OSA. Additionally, OSA patients with nocturnal sweating have lower REM sleep period and REM episode values. On the other hand, it was determined that nocturnal sweating did not affect the sleep architecture in patients without OSA. Considering these two results together, nocturnal sweating, depending on an increase in sympathetic activity due to respiratory problems during sleep, affect the REM sleep periods.

In the OSA group, AHI during the REM sleep period was found to be higher in patients with nocturnal sweating when compared to those without nocturnal sweating. The AHI during the total sleep time was not different between the OSA patients with and without nocturnal sweating. The REM sleep period was lower in patients with nocturnal sweating, but if the OSA patient with nocturnal sweating entered into the REM sleep period, there were more respiratory events in their record compared to those without nocturnal sweating, in the REM sleep period. On the other hand, this higher AHI during the REM sleep period in OSA patients with nocturnal sweating causes arousals and may result in disruption of REM sleep, which caused a shorter REM sleep period. However, a significantly lower REM episode in OSA patients with nocturnal sweating indicates that these patients have problems in not only sustaining the REM period, but also sustaining the internal sleep cycle with reduced REM episode. REM sleep latency was not different between the groups in our sample. Arnardottir et al. could not find a relationship between sweating and conventional indicators of OSA such as the AHI and desaturation index. However, they stated that the severity of OSA might affect sweating even if they did not determine it in their study (2). In support of this theory, it was determined in this study that the duration of oxygen saturation was lower than 90%, was significantly higher in the group with nocturnal sweating. This finding indicates that there is a relationship between the duration of hypoxia and sweating.

The patients with OSA and the control group were evaluated for the presence of nocturnal sweating and its effect on sleep parameters. There was no difference of nocturnal sweating between the groups. The individuals with nocturnal sweating in the OSA group were older than the individuals with nocturnal sweating in the control group. It was determined in previous studies that young age was related to nocturnal sweating in the group with apnoea and there was no such relationship in the general population (10, 11). In contrast, the findings of this study showed that individuals with sweating in the OSA group were older in age compared to individuals with sweating in the control group, who were associated with the young age.

This study found that comorbid diseases in apnoea patients were significantly higher compared to the group without apnoea, similar to findings from the literature (13). Moreover, the number of females was lower than males. This result was in accordance with the fact that the prevalence and diagnosis of OSA were less in females (19).

There was no difference in periodical leg movements during sleep between individuals with and without nocturnal sweating in the OSA group. While Mold et al. found a relationship between restless legs syndrome and nocturnal sweating, Arnardottir et al. could not find a relationship (11, 13). The Epworth sleepiness scale was found to be higher in individuals with nocturnal sweating in the group with apnoea. Nocturnal sweating did not affect the Epworth sleepiness scale in the control group. This condition indicates that the results on sleepiness are different in patients with and without apnoea. Contrary to the literature, there was no difference in Epworth sleepiness scale between the groups with and without apnoea (13). Our control group consisted of patients who were admitted to our sleep centre with complaints of snoring, sleepiness or observed breathing cessations during sleep and had polysomnography with a prediagnosis of OSA. But PSG test revealed that they did not have OSA. Also, they did not have any other sleep disorders. The Epworth sleepiness scale is a subjective test and one of the common complaints in the control group was sleepiness, which may be the reason there was no difference between the groups. While the BMI was found to be higher in individuals with nocturnal sweating in

the group with apnoea, there was no relationship between nocturnal sweating and BMI in the group without apnoea.

The following are limitations associated with this study: 1) nocturnal sweating was evaluated subjectively, they were not evaluated with objective methods; 2) the changes in the data after the PAP titration study were not evaluated. On the other hand, the fact that an examination between nocturnal sweating and sleep parameters was conducted and evaluated with the control group increased the importance of the study. To the best of our knowledge, this is the first study revealing increased respiratory events during the REM period in OSA patients with nocturnal sweating compared to those without nocturnal sweating, and nocturnal sweating is associated with REM episode. According to the results obtained from this study, there was a decrease in the REM period and the number of respiratory events increased in the OSA patients with nocturnal sweating.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Ankara Numune Training and Research Hospital (22.11.2017/1544).

Informed Consent: Due to the retrospective design of the study, informed consent was not taken.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – G.K., S.Y.; Design – G.K., S.Y.; Supervision – G.K., S.Y.; Resources – G.K., B.D.A., K.M.M., Ö.K., R.S.; Materials - G.K., B.D.A., K.M.M., Ö.K., R.S., S.Y.; Data Collection and/or Processing - G.K., B.D.A., K.M.M., Ö.K., R.S., S.Y.; Analysis and/or Interpretation – G.K., Ö.K., R.S., K.M.M.; Literature Search – G.K., B.D.A.; Writing Manuscript – G.K., S.Y.; Critical Review – B.D.A., K.M.M., Ö.K., R.S.

Conflict of Interest: The authors have no conflicts of interest to declare.

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