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Evaluation of the effect of polysomnography sleep variables on the development of morning headaches in obstructive sleep apnea syndrome

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Abstract

Background & Objective: This study aimed to investigate the relationship between sleep variables in patients with obstructive sleep apnea syndrome (OSAS) with associated morning headaches and to determine the characteristic features of these headaches. *Methods:* Among the 546 patients evaluated in the sleep laboratory, 154 individuals who were not diagnosed as having OSAS were excluded from the study. Sleep variables of 160 patients with morning headache and 232 patients without morning headache were compared. *Results:* The mean Epworth Sleepiness Scale (ESS) scores (12.17±5.71) ($p<0.001$), mean Apnea-Hypnea Index (AHI) (36.48±24.47) ($p=0.019$), mean AHI during rapid eye movement (REM) sleep (AHI-REM) (37.45±28.83), and mean Oxygen Desaturation Index (ODI) (52.36±30.96) ($p=0.015$) were significantly higher in patients with morning headaches. The mean oxygen saturation (SpO_2) of patients with headaches was (89.83±4.62) ($p=0.038$), and their mean lowest SpO_2 (70.37±14.87) was significantly lower ($p=0.049$). It was observed that 87.5% of the patients with morning headaches had bilateral pain, 60% had mild pain, 65% had pain lasting 2 to 3 hours, and 72% had oppressive-compressive pain, 68% had a frequency of attacks of 5 to 15 per month, and 70% had no additional neurological symptom.
Conclusions: Our results suggest that the AHI is significantly increased during REM sleep in patients with OSAS with morning headaches. This suggests that the pathogenesis of headaches is due to the effects of apnea during REM sleep. Moreover, the fact that the ODI was higher, and oxygen saturations were lower in the headache group emphasizes that hypoxia should be considered in the pathophysiologic process.

Keywords: Morning headache, obstructive sleep apnea syndrome, polysomnography, apnea-hypopnea index; AHI-REM

INTRODUCTION

Recurrent episodes of upper airway obstruction during sleep that restrict breathing are an indicator of obstructive sleep apnea syndrome (OSAS). It is usually associated with symptoms such as daytime sleepiness, snoring, and morning headaches. These headaches are usually caused by repeated oxygen desaturation and fragmented sleep. The diagnosis of OSAS is usually determined using the Apnea-Hypopnea Index (AHI), which measures the number of apneas and hypopneas per hour of sleep. An AHI of at least 5 is indicative of OSAS, along with symptoms such as excessive daytime sleepiness.[1]

Morning headaches are frequently reported by people with OSAS and are recognized as a type of headache associated with sleep apnea in the International Classification of Headache Disorders (ICHD-3).[2] This classification stresses the significance of addressing OSAS both in terms of its respiratory effects and its potential impact on headache disorders. The frequency of headaches in individuals with OSAS is not low. Studies have shown that 12 to 47% of patients presenting with headaches during the night or exclusively in the morning had OSAS.[3]

Sleep fragmentation can cause several physiologic alterations which can exacerbate

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headaches on awakening. Recognizing morning headaches as a potential indicator of OSAS is important for the timely diagnosis and management of the condition. In addition to reducing sleep-related symptoms, addressing OSAS can lessen the frequency and intensity of morning headaches.[3,4]

There is considerable clinical, anatomic, biochemical, and physiologic evidence supporting a relationship between the normal physiology of sleep and the occurrence of headaches in biologically predisposed individuals. Nevertheless, although the relationship between headache and sleep is well defined, the physiologic mechanism is not fully understood. Several hypotheses have been suggested to explain the relationship between OSAS and the occurrence of headaches, especially immediately after awakening. Hypercapnia, vasodilator pulsation, and fluctuation of oxygen saturation (SaO2) with an increase in intracranial pressure during the night are considered to be contributing factors.[5,6]

This study aimed to investigate the relationship between sleep variables in patients with OSAS who experienced headaches and determine the characteristic features of these headaches.

METHOD

Patients aged 18 to 70 years who underwent polysomnography (PSG) in our hospital's sleep laboratory from February 2022 to December 2023 and who had one or more of the symptoms of snoring, excessive daytime sleepiness, and witnessed apnea while sleeping were included. The patients' habits, chronic diseases, usual headaches (patients were asked if they had migraines, tension-type headaches, or autonomic cephalalgias), demographic data, and body mass index (BMI) were recorded. The Epworth Sleepiness Scale (ESS) was applied to the patients before undergoing PSG.

Sleep and physiologic variables were monitored using a Philips Respironics Alice 5 Diagnostic Sleep System. Electroencephalography (EEG) with 10 channels (C3, C4, O1, O2, Fp1, Fp2, F3, F4, P3, P4), submental electromyography (EMG), right and left eye electrooculography, electrocardiography, oronasal airflow (thermal sensor and nasal pressure transducer), body position, thoracic and abdominal motion (inductance plethysmograph), arterial blood oxygen saturation measurements (finger pulse oximetry), left and right leg movement sensors (EMG), and a tracheal sounds were recorded. Apnea was defined as a decrease of more than 90% in the airflow signal measured using the thermal sensor for at least 10 seconds. Hypopnea was defined as a decrease in the nasal pressure signal of more than 30% compared with baseline and desaturation of more than 3% compared with baseline for at least 10 seconds or resulting in arousal. An AHI <5 with snoring was considered as simple snoring, 5-14.9 was taken as mild OSAS, 15-29.9 as moderate OSAS, and ≥30 as severe OSAS. PSG was performed using the same protocol in all cases, and the recordings were made by an experienced sleep physician who was blinded as to whether the patients had headaches.

In the study, patients diagnosed with OSAS were divided into two groups as patients with concomitant morning headaches and patients without headaches. The questions in the questionnaire for the diagnosis of patients with morning headaches were based on the ICHD-3 criteria. Patients were questioned about the location of pain (bilateral/unilateral), the severity of pain (mild, moderate, severe), duration, pain character (throbbing, oppressive-compressive), frequency of attacks, and accompanying symptoms. The primary criteria were headache on waking up in the morning and improvement within 4 hours. Neurologic and physical examinations, routine blood tests, and brain imaging were performed to exclude possible space occupying lesions and other pathologies.

Age, BMI, the ESS, presence of comorbidity, REM latency, AHI, AHI-REM, AHI during non-rapid eye movement (AHI-NREM), Oxygen Desaturation Index (ODI), mean oxygen saturation (SpO_2), and lowest SpO_2, and other sleep and respiratory variables were compared between the groups.

Ethical approval for the study was received from Karatay University Local Ethics Committee (Number: 2023/020).

Statistical analysis

The data obtained as a result of the study were analyzed using the Statistical Package for the Social Sciences (SPSS) 27.0 package program. In descriptive analyses, frequency data are presented as number (n) and percentage (%), and numerical data are presented as mean±standard deviation, minimum-maximum. A t-test for independent samples was used to compare the numerical data between two groups. The homogeneity of variance was evaluated using Levene's test. Logistic

regression analysis was used. The Chi-square (χ^2) test and Fisher's exact Chi-square test was used to compare categorical data. Significance was accepted as $p<0.05$.

RESULTS

In our study, 546 patients hospitalized in our sleep laboratory were evaluated. One hundred fifty-four patients were excluded from the study because they did not have OSAS. Sleep variables of 160 patients with morning headaches and 232 patients without morning headaches were evaluated. The majority (62.6%) of the patients were male, and 70.0% had obesity. ESS was found to be normal in 61.9% of the patients, and ESS was found to be normal in 37.7%. OSAS was present in 71.8% of the patients, and 33.7% were defined as having severe OSAS (Table 1).

The mean age of patients with headaches (48.86±11.31 years) was significantly higher than those without (42.96±12.32 years) (p=0.001). The mean ESS scores of patients with headaches (12.17±5.71) were significantly higher than those without (8.06±6.14) (p<0.001). The mean AHI of patients with headaches (36.48±24.47) was significantly higher than those without (28.77±21.03) (p=0.019). The mean AHI-REM of patients with headaches (37.45±28.83) was significantly higher than those without (28.82±28.57) (p=0.040). The mean ODI was significantly higher in patients with headaches (52.36±30.96) than in those without (42.20±26.44) (p=0.015). The mean SpO_2 of patients with headaches (89.83±4.62) was significantly lower than those without (90.97±3.00) (p=0.038). The lowest mean SpO_2 of patients with headaches (70.37±14.87) was significantly lower than those without (74.14±11.75) (p=0.049). No significant difference was found between BMI, sleep efficiency, sleep latency onset time, total sleep time, N1, N2, N3, AHI-NREM, and REM latency values of patients with and without headaches (p>0.05) (Table 2).

Logistic regression analysis was applied to determine the relationship between OSAS severity and morning headaches. It was observed that morning headaches increased with the severity of OSAS (Table 3).

The comparison of the presence of headache according to sex and comorbidity in patients with OSAS is presented in Table 4. A significant difference was found when the presence of headache was compared between the sexes (p<0.001), with headaches found to be more common in females. Patients with hypertension were more likely to have headaches (p=0.027). No significant differences was found in headache frequency in patients with and without diabetes and CAD (p>0.05) (Table 4).

Analyzing the pain characteristics of patients with morning headache, it was observed that 87.5% had bilateral pain, 60% had mild pain, 65% had pain lasting 2 to 3 hours, 72% had

Table 1: Demographic features of the study population

Features	Group	n	%
Sex	Male	342	62.6
	Female	204	37.4
Obesity status	Normal	18	3.3
	Overweight	150	26.7
	Obese	382	70.0
ESS (severity of sleepiness)	Normal	106	37.7
	Increased	132	24.2
	Moderate	66	12.1
	Medium	32	5.9
	Severity	110	20.1
OSAS	Yes	392	71.8
	No	154	28.2
OSAS Severity Classification	Simple Snoring (AHI <4.9)	154	28.2
	Mild OSAS (AHI 5-14.9)	118	21.6
	Medium OSAS (AHI 15-29.9)	90	16.5
	Intense OSAS (AHI >30)	184	33.7

ESS: Epworth Sleepiness Scale, AHI: Apnea-Hypopnea Index

Table 2: Comparison of patients with OSAS with and without headaches

	Patients with OSAS with headaches (n=160)	Patients with OSAS without headaches (n=232)	*p*-value
Age	48.86±11.31	42.96±12.32	**0.001**
BMI	34.31±5.89	33.47±5.86	0.330
Epworth Sleepiness Score	12.17±5.71	8.06±6.14	**<0.001**
Sleep efficiency	81.13±11.97	81.74±10.37	0.702
Sleep latency	26.11±26.01	34.36±35.59	0.063
Total sleep time	381.37±68.63	383.42±55.24	0.818
REM latency	204.23±84.67	203.62±91.76	0.963
N1	2.06±2.52	2.99±4.63	0.073
N2	79.02±56.62	82.64±61.97	0.678
N3	37.92±38.74	39.89±30.64	0.692
AHI	36.48±24.47	28.77±21.03	**0.019**
AHI-REM	37.45±28.83	28.82±28.57	**0.040**
AHI-NREM	28.32±24.72	27.8±21.16	0.830
ODI	52.36±30.96	42.20±26.44	**0.015**
Mean SpO_2	89.83±4.62	90.97±3.00	**0.038**
Lowest SpO_2	70.37±14.87	74.14±11.75	**0.049**

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). OSAS: obstructive sleep apnea syndrome, F: female, M: male, BMI: body mass index, SEI: Sleep Efficiency Index, TST: Total Sleep Time AHI: apnea-hypnea index, REM: rapid eye movement sleep

oppressive-compressive pain, 68% had frequency of attacks 5 to 15 per month, and 70% had no accompanying symptoms (Table 5).

DISCUSSION

Our findings of sleep variables in patients with OSAS with and without morning headaches revealed that AHI, REM AHI, and ODI were significantly higher, and that mean SpO_2 was significantly lower in the group with headaches. Koç *et al*. evaluated the relationship between AHI-REM levels and morning headaches in individuals with OSAS, reporting that sleep variables in the REM period were the possible cause of OSAS headaches.[7] Our results support this theory.

The association of headache with OSAS is quite common. Although 18% of patients with OSAS had morning headaches, only 5-7% of the general population experiences this symptom.[8-10] Miksikastos *et al*. reported that the most common concomitant headache was medication overuse headache (MOH) in patients with OSAS.[8] Kristiansen *et al.* recently reported an increased frequency of morning headaches in patients with OSAS (11.8%) compared with those without OSAS (4.16%) in the general population.[11] OSAS was detected in 12-42% of people with nocturnal or morning headache using PSG.[3]

Our study showed that both AHI and AHI-REM levels were significantly higher in the group with OSAS headaches. However, we observed no statistically significant difference between

Table 3: Association between morning headache and OSAS severity

	Morning Headache	Mean Standard Deviation	*t*	*p*
Mild OSAS *(AHI 5-14.9)*	33/118	**0.058**	**3,528**	**<0.001***
Moderate OSAS *(AHI 15-29.9)*	38/90	**0.059**	**5,882**	**<0.001***
Severe OSAS *(AHI >30)*	89/184	**0.049**	**8,063**	**<0.001***

*p significance <0.001, OSAS: obstructive sleep apnoea syndrome, AHI: apnea-hypnea index

Table 4: Comparison of headache presence and various characteristics in patients with OSAS

	Headache			
	No (n=232) n (%)	Yes (n=160) n (%)	χ^2	p
Sex				
Male	176 (75.9)	80 (50.0)	13.977	**<0.001**
Female	56 (24.1)	80 (50.0)		
Hypertension				
No	198 (85.3)	116 (72.5)	4.901	0.027
Yes	34 (14.7)	44 (27.5)		
Diabetes				
No	210 (90.5)	134 (83.8)	2.018	0.155
Yes	22 (9.5)	26 (16.3)		
CAD				
No	214 (92.2)	136 (85.0)	2.595	0.107
Yes	18 (7.8)	24 (15.0)		

Significant findings (p<0.05) are shown. CAD: coronary artery disease

Table 5: Morning headache characteristics

	n=160	%
Location		
Unilateral	20	12.5
Bilateral	140	87.5
Headache severity		
Mild	96	60
Medium	44	27.5
Severity	20	12,5
Time (Hours)		
≤ 1	20	12.5
2-3	130	81.25
3-5	10	6.25
Pain character		
Oppressive/compressive	144	90
Throbbing	16	10
Frequency of attacks		
1-5/month	20	12.5
>5-15/month	136	85
>15/month	4	2.5
Concomitant symptom		
Migraine features	16	10
Autonomic symptoms	4	2.5
No concomitant symptoms	140	87.5

Significant findings (p<0.05) are shown. CAD: coronary artery disease

AHI-NREM and morning headaches in patients with OSAS. Although the increase in AHI values during the REM period in patients with morning headaches strengthens the view that it plays a role in the pathogenesis of headache in individuals with OSAS, the pathophysiology remains unclear. Dysfunction of central pain modulating circuits in brain regions such as the periaqueductal gray, dorsolateral pontine tegmentum, and rostral ventromedial medulla may be responsible for headaches. It has been argued that changes in brain regions that play an equal role in sleep regulation and nociception contribute to the dysfunction of central pain inhibitory activity, which leads to the occurrence of headaches after waking up in sensitive patients.[12-16]

In our study, we used the ESS to evaluate whether there was a significant relationship between morning headaches and increased daytime sleepiness. The mean ESS was significantly higher in the morning headache group (12.17±5.71) than in the group without morning headaches (8.06±6.14) (p<0.001). In the study of Seo et al., morning headaches were evaluated in 116 patients with OSAS and the ESS score was significantly higher in the morning headache group than in the group without morning headaches (10.90±5.45 vs. 8.13±4.27, p=0.003).[17] Furthermore, Kristoffersen *et al*. reported that increased daytime sleepiness was associated with increased headache frequency.[18] Besides, the ODI was observed to be higher than in the group without morning headaches, and statistically

significant decreases in mean SpO_2 and lowest SpO_2 values were also observed. Supporting our results, Koç *et al*. evaluated 361 patients OSAS and reported that the AHI was higher in patients with morning headaches and SpO_2 was lower during REM sleep compared with the control group.[7]

Various studies have highlighted that morning headaches may be related to a combination of the direct effect of lower SpO_2 and hypercapnia caused by apnea attacks, disturbances of cerebral blood flow auto regulation, and excessive neck movements during sleep.[3,19-21] Goksan *et al*. reported that various possible mechanisms including hypoxemia might cause morning headaches in individuals with OSAS, but in the same study, they found no particular factors to support this situation in logistic regression analysis. Patients with morning headaches were found to have significantly lower SpO_2 during REM and NREM sleep and lower mean SpO_2 values during total sleep time. Accordingly, the authors argued that hypoxemia had a pathophysiologic role in the mechanisms of morning headaches.[22]

In contrast to our results, in a study in which PSG sleep variables of 384 patients with sleep apnea and 294 patients with sleep apnea without headache were evaluated, mean SpO_2 in both REM and NREM sleep were lower in the OSAS without headache (WOSAH) group than in the OSAS with headache (WSAH) group. The mean SpO_2 level was not lower in the WSAH group, and the authors also argued that sleep apnea headache was not related to either the severity of OSAS or oxygen desaturation because no statistical difference was found between headaches according to AHI.[23] Similarly, in a study evaluating 75 patients with OSAS, it was reported that no statistically significant relationship was observed between headache and sleep variables.[24] Kristiansen *et al.* observed no significant differences in parameters such as total sleep time, mean SpO_2, higher mean oxygen desaturation levels, or lower means of lowest SpO_2 when comparing individuals with OSAS with or without sleep apnea headaches. This suggests that the pathophysiology of sleep apnea headache cannot be explained by oxygen desaturation alone.[11]

Göder *et al*. compared PSG recordings on the nights preceding morning headaches with those on the nights without morning headaches in patients with OSAS and found that the occurrence of morning headaches was associated with decreases in total sleep time, sleep efficiency, and the amount of REM sleep, and an increase in the duration of wakefulness on the previous night. Contrary to the results of our study, Göder *et al.* reported that oxygen desaturation and AHI did not differ when the nights with and without morning headaches were compared.[16] Considering the results in the literature, we see that there is still no consensus on the pathophysiologic process forming the relationship between OSAS and headache.

In our study, the pain characteristics of our patients with morning headache were compatible with the ICHD-3 beta criteria for sleep apnea headache.[2] It is very important to recognize sleep apnea headache by distinguishing it from other headaches according to the ICHD-3 beta criteria. In this way, CPAP treatment, which is known to be effective in sleep apnea headache, can be preferred in individuals whose headache is determined to be a sleep apnea headache.[25]

PSG in individuals with headaches revealed mild-to-severe OSAS in approximately one-third of the patients examined. As expected, older age, male sex, and higher BMI were independently associated with headaches. In patients with OSAS, the concomitant headache was most commonly MOH (half of all cases), followed by cluster headache (one-third of the cases) and migraine (one-quarter of the cases); hypnic headache was detected in one patient. Mild autonomic symptoms were present in almost half of the patients (not only in patients with CH). Headache attacks were of moderate intensity and usually lasted for several hours.[8] In our study, morning headache was observed more frequently in females with OSAS. Supporting our findings, Greenough *et al*. also reported that morning headaches were more common in female patients with OSAS than in male patients.[26] Göksan *et al*.'s findings support that morning headache is predominantly reported by female patients with OSAS, despite the higher number of male patients with more severe disease.[21]

The retrospective nature of our study, the small sample size, the inability to evaluate the pain response of patients after CPAP treatment in this context can be considered limitations of our study. Prospective studies including larger groups are required. The pathophysiologic features related to the occurrence of morning headaches in patients with OSAS, whose characteristics are still unclear, should be elucidated.

In conclusion, in our study, the effects of morning headache and sleep variables such as ODI, SpO_2, and AHI on headaches in patients with OSAS were evaluated. Our results suggest that the pathogenesis of headaches is related to the

pathophysiologic effects of apnea and consequent hypoxia in the REM sleep period. The results contribute to the characterization of morning headaches related to REM sleep physiology. The results may help in gaining a better understanding of headaches in the management of OSAS and improving treatment approaches.

DISCLOSURE

Ethics: Ethics committee approval was received Karatay University Local Ethics Committee approval was received. (2023/020) . This article does not contain any studies with human patients performed by any of the authors.

Data availability: The datasets generated during and/or analyzed dur ing the current study are available from the corresponding author on reasonable request.

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