Is Sleep Apnea Worse in the Winter?

Mustafa Yilmaz (1)
Nigar Yilmaz (2)
Dilek Aslan Ozturk (1)
Ercan Baldemir (3)
Gülser Karadaban Emir (1)
Murat Sahan (4)
Yasemin Unal (1)
Ayse Sözen (1)
Gülnihal Kutlu (1)

- (1) Mugla Sitki Kocman University, Faculty of Medicine, Department of Neurology, Mugla, Turkey
- (2) Mugla Sitki Kocman University, Faculty of Medicine, Department of Biochemistry,

Mugla, Turkey

- (3) Mugla Sitki Kocman University, Faculty of Medicine, Department of Biostatistics, Mugla, Turkey
- (4) Mugla Sitki Kocman University, Faculty of Medicine, Department of Otolaryngology, Mugla, Turkey

Correspondence:

Mustafa Yilmaz Mugla Sitki Kocman University, Faculty of Medicine, Department of Neurology, Mugla, Turkey **Email:** mustafayilmaz@mu.edu.tr

Abstract

Objectives: The present study aimed to investigate the seasonal variability of AHI values among patients with sleep apnea.

Patients and Methods: In order to conduct the retrospective study, we accepted 304 patients (223 male and 81 female) between May 2014 to May 2015 into our study at Mugla Sitki Koçman University Medical faculty of sleep disorders clinic. Patients were divided into four groups according to the timing of the PSG: winter, spring, summer, and autumn.

Results: We reviewed the records of patients, including their PSGs, and found that their AHI values were comparable across seasons. The average AHI value for males was 34.95± 29.95 and 21.88±2.76 for females; we observed no significant statistical difference among the four groups (both males and females) in terms of AHI.

Conclusion: Although patient complaints increased due to exacerbation of diseases such as asthma and allergic rhinitis in the winter, this study did not reflect a significant change in AHI values. Therefore, PSG examinations do not need to be repeated in different seasons.

Keywords: sleep apnea, seasonal variation

Introduction

Obstructive sleep apnea syndrome (OSAS) is the most widely suffered sleep disorder after insomnia and is increasingly common due to the prevalence of obesity. It describes a situation in which breathing is briefly and repeatedly interrupted for at least ten seconds, resulting in a reduction of blood oxygen levels [1,2]. This occurs when the muscles in the back of the throat fail to keep the airway open, despite efforts to breathe [3]. Obstructive sleep apnea can be caused by many factors, such as adenotonsillar hypertrophy, allergies and viral respiratory infections [4]. Furthermore, the prevalence of sleep apnea increases due to allergic rhinitis and asthma [5]. As the seasons change, sleep patterns change, and allergic rhinitis and asthma are more common in the spring [6].

Polysomnography (PSG), a type of sleep study, is a multiparametric test used to examine eye movements, muscle-brain-heart activity, oxygen saturation, position and nasal flow during sleep [7]. The apnea-hypopnea index (AHI) is the most common means to measure sleep apnea, recording the number of apneas or hypopneas per hour of sleep. A person's AHI is classified as mild-moderate or severe [8]. In this study, we aimed to determine and compare patients' AHI values, based on PSG tests, over four seasons, independent of age, sex, body mass index (BMI) and chronic obstructive pulmonary disease (COPD) status.

Materials and Methods

Study Population

In order to conduct the retrospective examination, we accepted 304 (223 male and 81 female) patients between May 2014 to May 2015 into our study at Mugla Sitki Kocman University Medical faculty of sleep disorders clinic. We reviewed these patients' records, including PSG results, and participants were divided into four groups according to the timing of the PSG: winter, spring, summer, and autumn. We ensured that the groups were similar in regards to age, sex and body mass index (BMI). In our study, the exclusion criteria included patients with chronic obstructive pulmonary disease (COPD). The patients' AHI values were compared across seasons. The study protocol has been approved by the Ethics Committee of the University.

Polysomnography

All patients underwent technician-attended whole-night polysomnography with EMBLA S4500 equipment in the sleep laboratory of our hospital. Polysomnography recordings were obtained between 10:00 pm and 06:00 am (8 hours). Six-channel electroencephalography (two each: occipital, central, and frontal), right and left electrooculography, electrocardiography, chin and right and left tibialis muscle electromyography, oronasal pressure, thoracal and abdominal respiratory efforts, pulse oximetry, position, and snoring sound were recorded. The polysomnographic data were scored manually by a

certified and experienced physician in accordance with the American Academy of Sleep Medicine Manual for Scoring Sleep and Associated Events, Version 2 [9]. Apnea was scored when there was a drop in the peak signal excursion by > 90% of pre-event baseline and the duration of the > 90% drop in sensor signal was > 10 s. Hypopnea was scored when the peak signal excursions dropped by > 30% of pre-event baseline for > 10 s in association with either > 3% arterial oxygen desaturation or an arousal. The AHI was calculated by dividing the number of apnea/ hypopnea events by the number of hours of sleep. Oxygen desaturation index 3 (ODI3) was calculated by dividing the number of 3% drops in oxygen saturation by the number of hours of sleep. The minimum oxygen saturation was noted.

Statistical Analysis

The data were processed and analyzed using SPSS-18 for Windows, Fisher's Exact Test, Pearson Correlations, and Pearson's Chi-Square test; logistic regression was used for the comparison of categorical and scale variables, where p<0.05 was considered to be statistically significant. Among the groups, variables that were found to be statistically significant and variables that are not conceptually compatible were added to the logistic regression model.

Materials and Methods

A total population of 304 patients (223 male and 81 female) with mean ages of 47.72 ± 12.68 in males and 49.70 ± 12.15 in females was studied. Mean BMI was 30.26 ± 5.16 in males and 30.88 ± 6.17 in females (Table 1). Mean age, sex and BMI did not differ among the four groups Total sleep duration was 380.16 ± 67.37 in males and $397,28 \pm 58.0$ in females (p=0.0501). We found that one season did not differ from the others, although patient AHIs were lowest in the spring and highest in the winter (Table 2, Figure 1). AHIs were highest in male patients with sleep apnea than female patients with sleep apnea (Figure 2).

Table 1

Patients with OSAS				
Male (n=223)		Female (n=81)		
Age	47.72±12.68	49.70±12.15		
вмі	30.26 ± 5.16	30.88±6.17		
Sleep Duration	380.16±67.37	397,28 ± 58.0		

Table 2

Gender	Seasons	AHI Scores	Std. Deviation
		(Mean)	
male	winter	43,9289	30,92175
	spring	30,5435	29,29490
	summer	32,1681	27,14016
	autumn	36,5386	30,84256
	Totally	34,9520	29,49444
female	winter	17,5045	21,73024
	spring	17,3957	19,66176
	summer	29,9417	29,43520
	autumn	22,3917	28,05441
	Totally	21,8827	24,86293
Totally	winter	35,2522	30,72469
	spring	26,9859	27,54966
	summer	31,6115	27,59003
	autumn	33,5071	30,58487
	Totally	31,4697	28,88036

Figure 1

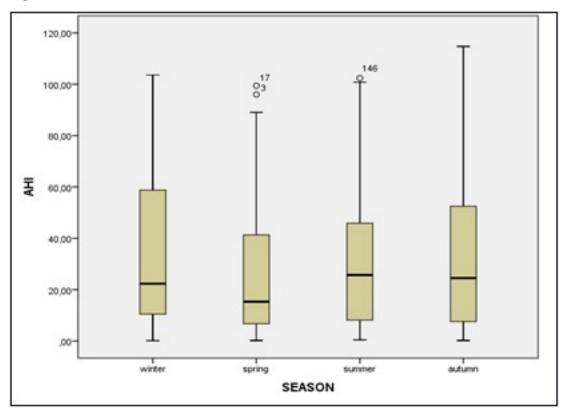
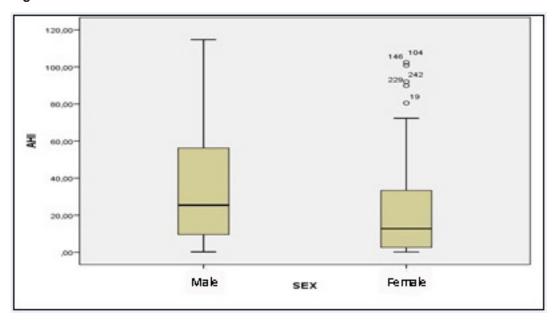


Figure 2



Discussion

In the present study, we have shown that patients with OSAS are not affected by the seasons. Although not statistically significant, we found that AHIs were lowest in the spring and highest in the winter. This runs contrary to some evidence in the literature indicating that patients with OSAS are affected by the seasons [10-12], although other sources have shown that patients with OSAS are not affected by the seasons. For instance, Dempsey demonstrated that airway infections and weather could have an effect on sleep apnea, but that the changes across the seasons had little effect on AHI values [13], while Cassol reported that more sleep disorder breathing events occurred in winter than in other seasons. They observed that people stopped breathing more than 30 times an hour in the colder months [14]. However, these differences may be associated with geographic location. The fact that many of these researchers are from Brazil and the USA could have affected their results. Our studies were conducted in Mu?la, but the same study may yield different results in another province.

Although inflammation and oxidative stress are important in the pathophysiology of OSAS, the relationship between inflammation remains poorly understood. Many factors affect the airway of a person with obstructive sleep apnea syndrome. For example, atmosphere and sun cycles can play a significant role in sleep quality [15]. Changes in seasons also affect the issue in different ways. Additionally, Kalra et al. found a high prevalence of snoring in young women with atopy and a significant association with asthma [11]. Similar studies have also shown that asthma is a frequent comorbidity in patients with OSAS (12). Similarly, Kalpaklioglu et al. reported that allergic rhinitis is a risk factor for a high apnea-hypopnea index, and after rhinitis treatment, a patient's AHI and Epworth Sleepiness Scale (ESS) scores will be reduced. They observed the most significant difference in a group treated with nasal steroid + antihistamine compared to the control group [16].

Several types of patients were included in this study, although ensuring similarity of age, sex and BMI may be considered one of its weaknesses. Here, using the same patients' PSG evaluation in different seasons would have been better. However, this was not attempted, as it was not covered by the study approval. In addition, patients who do not suffer from asthma and allergic rhinitis may be enrolled as a not statistical difference. At that time it could not be a generalization. Allergic rhinitis is a common disease in childhood [17], so if the study had been done in this age group, the AHI index would have been higher in the winter. Furthermore, we did not evaluate the complaints of the patients in this study. If we had evaluated their complaints by the subjective ESS test, results may have been more meaningful or useful.

In the light of the data obtained from this study, we found that seasons did not contribute to significant changes in AHI values. Therefore, examination of PSG does not need to be repeated in different seasons, especially among the elderly and those without asthma and allergic rhinitis.

References

- 1. Chokroverty S. Clinical Companion to Sleep Disorders Medicine. 2nd ed. Oxford, England: Butterworth-Heinemann Publishers; 2000
- 2. Crummy F, Piper AJ, Naughton MT. Obesity and the lung: Obesity and sleep disordered breathing. Thorax. 2008;63:738-46
- 3. Young T, Finn L, Kim H. Nasal obstruction as a risk factor for sleep disordered breathing. J Allergy Clin Immunol. 1997;99:757-762
- 4. Kramer MF, de la Chaux R, Fintelmann R, Rasp G. NARES: a risk factor for obstructive sleep apnea? Am J Otolaryngol. 2004;25:173-177
- 5. Staevska MT, Mandajieva MA, Dimitrov VD. Rhinitis and sleep apnea. Curr Allergy Asthma Rep. 2004;4(3):193-9
- 6. Skoner DP. Allergic rhinitis: Definition, epidemiology, pathophysiology, detection, and diagnosis. J Allergy Clin

Immunol. 2001; 108:2-8.

- 7. The American Academy of Sleep Medicine Inter-scorer Reliability Program: Sleep Stage Scoring, Richard S. Rosenburg, Steven van Hout, J Clin Sleep Med. 2013; 9(1): 81-87
- 8. Olson EJ, Moore WR, Morgenthaler TI, Gay PC, Staats BA. Obstructive sleep apnoea hypopnoea syndrome. Mayo Clin Proc. 2003;78:1545-52
- 9. Berry RB, Brooks R, Gamaldo CE, Harding SM, Lloyd RM, Marcus CL, Vaughn BV, for the American Academy of Sleep Medicine (2014) The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, Version 2.0.3. Accessed 1 July 2015
- 10. Kalra M, Biagini J, Bernstein D, Stanforth S, Burkle J, Cohen A, LeMasters G, Ann Allergy Asthma Immunol. Effect of asthma on the risk of obstructive sleep apnea syndrome in atopic women 2006 Aug; 97(2): 231-235.
- 11. Larsson LG, Lindberg A, Franklin KA, et al. Symptoms related to obstructive sleep apnoea are common in subjects with asthma, chronic bronchitis and rhinitis in a general population. Respir Med. 2001;95:423-429.
- 12. Dempsey JA, Veasey SC, Morgan BJ, O'Donnell CP. Pathophysiology of sleep apnea. Physiol Rev. 2010;90(1):47-112.
- 13. Cassol CM, Martinez D, da Silva FA, Fischer MK, Lenz Mdo C, Bós ÂJ. Is sleep
- apnea a winter disease?: meteorologic and sleep laboratory evidence collected over 1 decade. Chest. 2012;142(6):1499-507
- 14. Kent BD, Ryan S, McNicholas WT (2011) Obstructive sleep apnea and inflammation: relationship to cardiovascular co-morbidity. Respir Physiol Neurobiol 178(3):475-81.
- 15. Hatipoglu U, Rubinstein I. Inflammation and Obstructive Sleep Apnea Syndrome Pathogenesis: A Working Hypothesis, Respiration 2003;70:665-671
- 16. Kalpaklio?lu AF, Kavut AB, Ekici M. Allergic and nonallergic rhinitis: the threat for obstructive sleep apnea. Ann Allergy Asthma Immunol. 2009;103(1):20-5.
- 17. Wenzel S. Severe asthma in adults. Am J Respir Crit Care Med. 2005;172:149-60