Uploaded to VFC Website

November 2012

This Document has been provided to you courtesy of Veterans-For-Change!

Feel free to pass to any veteran who might be able to use this information!

For thousands more files like this and hundreds of links to useful information, and hundreds of "Frequently Asked Questions, please go to:

Veterans-For-Change

Veterans-For-Change is a 501(c)(3) Non-Profit Corporation
Tax ID #27-3820181

If Veteran's don't help Veteran's, who will?

We appreciate all donations to continue to provide information and services to Veterans and their families.

https://www.paypal.com/cgi-bin/webscr?cmd=_s-xclick&hosted_button_id=WGT2M5UTB9A78

Note: VFC is not liable for source information in this document, it is merely provided as a courtesy to our members.

Clinical Profiles of Obstructive Sleep Apnea Syndrome

Obstrüktif Uyku Apne Sendromunun Klinik Özellikleri

Meral Uyar[1], Osman Elbek[1], Neriman Aydın[2], Nilüfer Çifçi[1], Nazan Bayram[1], Füsun Fakılı[1], Nevhiz Gündoğdu[1], Öner Dikensoy[1]

[1]Gaziantep University, Pulmonary Diseases, Gaziantep, Turkey
[2]Gaziantep University, Public Health, Gaziantep, Turkey

ABSTRACT

Introduction: To evaluate the clinical profiles of obstructive sleep apnea syndrome.

Material and Method: Sixty two consecutive patients who were diagnosed as having obstructive sleep apnea syndrome were included in the study.

Results: The male/female ratio of patients with an apnea-hypopnea index of over 5 was 2.64. A family history of the syndrome was present in 43.5% of the patients. Of the 62 patients, 35% were diagnosed as having an overlap syndrome due to the coexistence of chronic obstructive pulmonary disease. The mean Epworth Sleepiness Scale score in mild, moderate and severe obstructive sleep apnea syndrome patients were 8.81±6.46, 10.38±5.14, 15.34±5.44, respectively (p=0.001). Hypertension, diabetes mellitus, coronary artery disease, subclinical hypothyroidism and cerebrovascular accidents were detected in 35.5%, 11.3%, 16.1%, 5.9%, and 1.6% of the cases, respectively. No correlation was identified between groups with respect to smoking habits, fasting blood glucose, triglyceride, LDL, HDL and total cholesterol levels. We detected a significant difference in neck and waist circumferences and hematocrit values in those having severe obstructive sleep apnea syndrome (p<0.05). Although the Epworth Sleepiness Scale score, smoking habit, gender distribution, neck circumference, fasting blood glucose, triglyceride, LDL, HDL and total cholesterol levels did not differ significantly between those having obstructive sleep apnea syndrome and overlap syndrome, body mass index, waist circumference and hematocrit values were significantly higher in overlap syndrome patients (p<0.01).

Conclusion: Study showed that many OSAS patients have comorbid diseases, and taking a family history of patients suspected as having OSAS is essential. Also, severe obstructive sleep apnea syndrome patients have a higher Epworth Sleepiness Scale score, and neck and waist circumference.

(Tur Toraks Der 2008;9:113-6)

Key words: Family history, neck circumference, sleep apnea syndrome, waist circumference

Received: 02. 11. 2007 *Accepted: 15. 05. 2008*

ÖZET

Giriş: Çalışma obstrüktif uyku apne sendromunun klinik özelliklerini değerlendirmek için yapıldı.

Gereç ve Yöntem: Obstrüktif uyku apne sendromu tanısı alan 62 ardışık hasta çalışmaya dahil edildi.

Bulgular: Apne hipopne indeksi 5'ten büyük olan hastalarda erkek/kadın oranı 2.64 idi. Hastaların %43.5' inde aile öyküsü mevcuttu. 62 hastanın %35'i kronik obstrüktif akciğer hastalığının birlikteliğinden dolayı overlap sendromu olarak değerlendirildi. Obstrüktif uyku apne sendromlu hastaların ortalama Epworth Uykululuk Skalası Skoru sırasıyla 8.81±6.46, 10.38±5.14, 15.34±5.44 idi (p=0.001). Hipertansiyon, diabetes mellitus, koroner arter hastalığı, subklinik hipotiroidi ve serebrovasküler olay varlığı sırasıyla hastaların %35.5, %11.3, %16.1, %5.9 ve %1.6'sında mevcuttu. Gruplar arasında sigara içme alışkanlığı, açlık kan şekeri, trigliserid, LDL, HDL ve total kolesterol düzeyleri açısından fark tespit edilmedi. Ağır dereceli obstrüktif uyku apne sendromu olan hastalarda boyun ve bel çevresi ve hematokrit düzeyleri arasında anlamlı fark tespit edildi (p<0.05). Epworth Uykululuk Skalası Skoru, sigara içme alışkanlığı, cinsiyet dağılımı, boyun çevresi, açlık kan şekeri, trigliserid, LDL, HDL ve total kolesterol düzeyleri obstrüktif uyku apne sendromu ve overlap sendromu olanlarda farklılık göstermemesine rağmen vücut kitle indeksi, bel çevresi ve hematokrit düzeyleri overlap sendromlu hastalarda daha yüksekti (p<0.01).

Sonuç: Çalışmada OSAS hastalarının çoğunda komorbid hastalıkların bulunduğu, OSAS şüphesi olan hastalardan aile öyküsünün alınmasının önemli olduğu gösterilmiştir. Ayrıca ciddi obstrüktif uyku apne sendromlu hastalarda daha yüksek Epworth uykululuk skalası skoru, boyun ve bel çevresi tespit edilmiştir.

(Tur Toraks Der 2008;9:113-6)

Anahtar sözcükler: Aile öyküsü, boyun çevresi, uyku apne sendromu, bel çevresi

Geliş Tarihi: 02. 11. 2007 *Kabul Tarihi: 15. 05. 2008*

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a disorder which can cause significant morbidity and mortality. Patients with OSAS are at increased risk of having accidents [1], therefore OSAS is an extremely important pub-lic health problem. Male sex, obesity, older age and family history are all known risk factors for OSAS [2]. In this paper we reported clinical profiles such as risk factors and comorbid diseases in OSAS.

Presented At: 10th Annual Congress of Turkish Thoracic Society

Address for Correspondence / Yazışma Adresi: Meral Uyar, Gaziantep University, Pulmonary Diseases, Gaziantep, Turkey
E-mail: meraluyar1@yahoo.com

MATERIAL AND METHOD

The study population consisted of patients admitted to the Gaziantep University Sleep Laboratory during the period between July 2006-March 2007, who were diagnosed as having OSAS. The study population included those over 18 years of age. Apnea was defined as cessation of airflow for at least 10 seconds via mouth or nose, whereas hypopnea was defined as at least 50% flow limitation with 3% desaturation. The apnea hypopnea index (AHI) was the total number of apnea or hypopnea incidents per hour. An AHI of ≥5 was necessary for OSAS criteria. Patients were classified as having mild, moderate and severe OSAS by AHI of 5-15, 15-30, ≥30, respectively. Patients having chronic obstructive pulmonary disease (COPD) were subgrouped as overlap syndrome. Sleep studies were performed using the guidelines of Rechtschaffen and Kales [3]. All patients underwent
evaluation including polysomnography, lung function testing, arterial blood gas analysis, echocardiography, total blood count, biochemical analysis including fasting blood glucose, blood urea nitrogen, triglyceride, LDL, HDL and total cholesterol levels and thyroid function tests (TSH, FT4). The Epworth sleepiness scale (ESS) score wascalculated for each patient and ≥10 was evaluated as excessive daytime sleepiness. Body mass index (BMI) (kg/m^2) was calculated for each patient. Neck and waist circumferences were measured and the family history obtained for each patient.

Statistics: Data was recorded to SPSS 9 statistical software (Statistical Package for Social Science). Comparisons between groups were achieved by one way ANOVA test. $p<0.05$ was accepted as significant.

RESULTS

We evaluated results of 62 consecutive patients who were diagnosed as having OSAS. Of the 62 patients, 45 were male. The mean age of males was 49.02±11.79 (22-75) and 52.06±13.49 (19-74) for females (p=0.339). Hypertension was detected in 35.5% of patients, diabetes mellitus in 11.3%, coronary artery disease in 16.1%, hypothyroidism in 5.9% and cerebrovascular accident in 1.6% of patients. Distribution of patients evaluated as mild, moderate and severe OSAS with respect to AHI was established as 27%, 21%, and 52%, respectively. A family history compatible with OSAS was present in 43.5% of the study population. Symptoms were evaluated for each patient and 75% had snoring, 50% had witnessed apnea, 6% had headache, 69% had excessive daytime sleepiness, 42% had dyspnea, 3% fatigue and 3% had chest pain. Patients were asked about their smoking habits, and 53.2% of them were smokers and, when subsets of the patients were evaluated, 30.3% those having mild OSAS, 21.6% moderate OSAS and 48.5% of severe OSAS had a history of smoking (χ^2=0.35, p=0.84).

The ESS score was evaluated for each patient and the mean ESS for mild, moderate and severe OSAS groups were 8.81±6.46, 10.38±5.14 and 15.34±5.44, respectively. The ESS score was higher in severe OSAS patients (p=0.001) (Figure 1). Neck circumferences for each group were measured and the mean neck circumferences for mild and moderate OSAS were 41.00±3.94 and 42.80±2.53, respectively, whereas severe OSAS patients had a higher mean neck circumference of 45.50±3.86 (p=0.009). Waist circumferences for each group were 108.11±21.72, 116.67±17.66, 125.75±15.39 respectively (p=0.049) (Figure 2).

Hematocrit levels were 43.00±3.11 for mild OSAS, 46.25±3.52 for moderate OSAS and 47.21±6.20 for severe OSAS patients (p=0.038). Fasting blood glucose levels, triglyceride, LDL, HDL and total cholesterol levels between groups were not statistically significant (p>0.05) (Table 1).

When patients were separated according to having OSAS or additional COPD (overlap syndrome), 35% of the study population had overlap syndrome. No statistically significant difference was detected between OSAS and overlap syndrome in respect to comorbid diseases (p>0.05).

We detected statistically significant differences in BMI, waist circumference and hematocrit levels between OSAS and overlap syndrome patients (p<0.05) (Figure 3,

Table1. Characteristics of the study population

	Mild OSAS (n=17)	Moderate OSAS (n=13)	Severe OSAS (n=32)	p
Smoking history (%)	30.3	21.6	48.5	0.84
Fasting blood glucose	102.85±22.85	103.27±47.53	107.39±33.66	0.913
Triglyceride	212.87±153.31	142.27±87.73	106.0 ±25.60	0.285
LDL	109.0±27.27	103.45±36.31	106.0±25.60	0.893
HDL	41.50±11.58	45.18±9.65	47.33±12.17	0.316
Total cholesterol	183.60±27.79	172.33±47.36	192.22±29.49	0.239

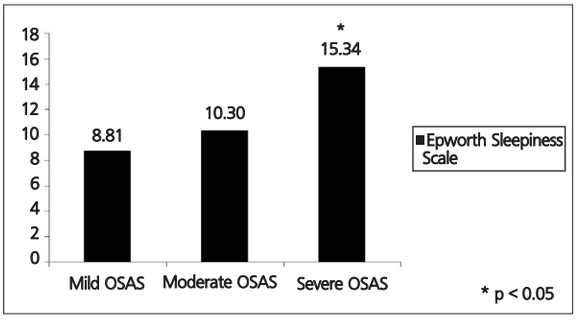

Figure 1. Epworth Sleepiness Scale

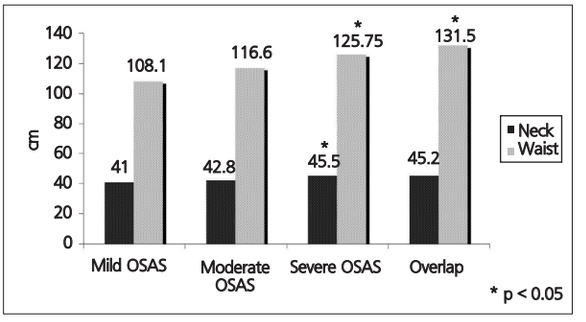

Figure 2. Neck and Waist Circumferences of OSA and Overlap Syndrome

4). We found no difference between sex, smoking habit, neck circumference, ESS, fasting blood glucose, triglyceride, LDL, HDL, total cholesterol levels of OSAS and overlap syndrome patients (p>0.05).

DISCUSSION

In this study we found that many OSAS patients have comorbid diseases, severe OSAS patients had higher ESS, neck and waist circumferences and patients with overlap syndrome had higher BMI, waist circumference and hematocrit values.

The prevalence of OSAS is reported to be 3-4% in some studies [4,5] and 4-6% of all middle aged men and 2% of all middle aged women [2]. We found a predominance in male subjects as a 2.64 fold increase. This result is compatible with the literature findings of OSAS being more prevalent in males [6,7]. The mean age distributions of our study population for male and female subjects were found to be 49.02±11.79 and 52.06±13.49, respectively and were similar to the literature and we found no difference between the sexes in mean ages [6].

We found the hypertension rate to be 35.5% in our study population as a comorbid disease. Kokturk et al have reported 38 of 94 OSAS patients (40.4%) as having cardiovascular diseases in their study population [8]. Sasanabe et al have shown that hypertension was more common in their patients with OSAS than in the controls, and another study found this ratio to be 45% in patients with OSAS [9,10]. Also, studies have revealed that of patients with essential hypertension, 34.8% had OSAS [4] and, in patients who have hypertension resistant to drug therapy, OSAS should have been sought [11]. There is increased risk of diabetes mellitus in OSAS patients, being compatible with our findings, as the incidence of diabetes mellitus was 11.3% in our study population [7]. We detected coronary artery disease in 16.1% of our OSAS patients, which is similar to a study by Peker et al [12]. We detected a cerebrovascular accident in one patient. Increasing medical evidence suggests that OSAS is an independent risk factor for cerebrovascular accidents, and conversely, those with cerebrovascular accidents are also at increased risk of having OSAS [13,14]. A study demonstrated that platelet activation, epinephrine, and high blood pressure play a role in the high prevalence of erebrovascular events in patients with OSAS [15]. Although a study by Resta et al have shown that subclinical hypothyroidism did not influence the prevalence and severity of OSAS [16], we detected subclinical hypothyroidism in 5.9% of our study population.

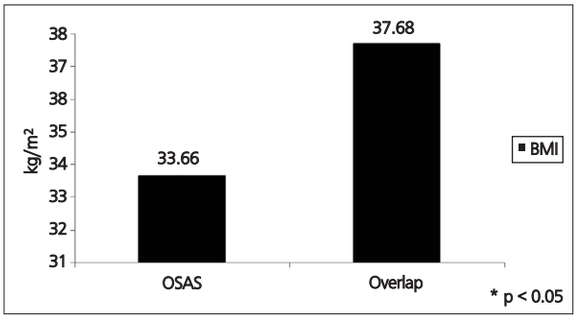

Figure 3. Body Mass Index of OSA and Overlap Syndrome

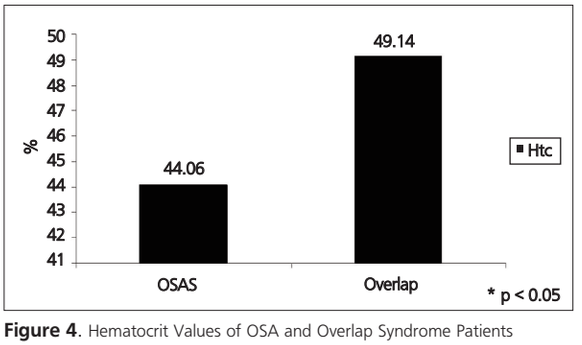

Figure 4. Hematocrit Values of OSA and Overlap Syndrome Patients

More than 2/3 of our study subjects had moderate or severe OSAS. Kjelsberg et al have found similar results in their study group [17]. This may be due to symptoms that cause advanced disease leading to patient admission.

We detected a family history of OSAS in 43.5% of the study population. A study by Guilleminault et al has shown disproportionate craniofacial anatomy as a risk factor for familial groups with OSAS [18]. Patients were asked about their smoking habits, and 53.2% were smokers and we did not detect any difference in the smoking habits of OSAS and overlap syndrome patients. Our result is higher than Kjelsberg et al., as they found the current smoker rate as %23 of their study population [17].

When we evaluated the symptoms of the study population, we found the most frequent symptom to be snoring, while excessive daytime sleepiness and witnessed apnea are the other common symptoms of our cases.The leading symptoms of Kjelsberg et al were similar [17].

The ESS score is a score that is widely used to evaluate sleepiness state. An ESS score greater than 10 suggests significant daytime sleepiness [19]. The scores of our study population of mild, moderate and severe OSAS patients were 8.81±6.46, 10.38±5.14, 15.34±5.44, respectively and we detected a statistically significant correlation between ESS scores and the severity of OSAS. This result shows that the sleepiness state may be increased by the severity of OSAS. Our results are compatible with a study showing that ESS scores were significantly correlated with AHI [20], although another study reported contradicting data that ESS is not influenced by AHI stratum [9].

Hora et al reported that OSAS patients had higher BMI, waist-to hip ratio and neck circumference as compared to controls [21]. We did not use a control group but we showed a significant difference in neck and waist circumferences and hematocrit values in those having severe OSAS. Similar results to those of our study were found by Ferguson et al concerning neck circumference, as AHI is influenced by the thickness of the neck [22].

We did not identify any difference among the study population regarding fasting blood glucose, triglyceride, LDL, HDL and total cholesterol levels. A study by Sasanabe [9] did not detect a significant difference in HDL le-

vels, whereas they detected differences in severe OSAS patients in their fasting plasma glucose, LDL and lipid profiles.

We detected overlap syndrome in 35% of patients referred to our clinic, although a recent study by Bednarek et al [23] declared that COPD in subjects with OSAS was as frequent as in the general population. This can be partially attributed to the patients who were referred from our chest clinic. We detected a statistically significant difference in BMI, waist circumference and hematocrit levels between OSAS and overlap syndrome patients. In contrast to our study, Radwan et al did not show any statistically significant difference between OSAS and overlap syndrome patients in BMI [24].

In conclusion, many OSAS patients have comorbid diseases, including hypertension, diabetes mellitus, and coronary artery disease at diagnosis. Taking the family history of patients suspected as having OSAS is essential because about half of them have a positive family history. We found that patients who had severe OSAS had higher ESS, neck and waist circumferences, and patients with overlap syndrome had higher BMI, waist circumference and hematocrit values. We suggest that we should be more aware in evaluating those patients with higher ESS, neck and waist circumference. If we consider the burden in sleep studies, those patients with a high probability of having OSAS, especially those having comorbid diseases, could be assessed earlier.

REFERENCES

1. George CF, Smiley A. Sleep apnea & automobile crashes. Sleep 1999;22:790-5.
2. Coughlin S, Calverley P, Wilding J. Sleep disordered breathing-a new component of syndrome X. The International Association for the study of obesity. Obesity Rev 2001;2:735-41.
3. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects. Washington, DC: US Department of Health, Education, and Welfare, Public Health Service, NIH Publication; 1968.
4. Ip MSM, Lam B, Lauder I, et al. A community study of sleep disordered breathing in middle-aged Chinese men in Hong Kong. Chest 2001;119:62-9.
5. Ip M, Chung KF, Chan KN, et al. Previously unrecognized obstructive sleep apnea in Chinese subjects with essential hypertension. Lung 1999;177:391-400.
6. Shepertycky MR, Banno K, Kryger MH. Differences between men and women in the clinical presentation of patients diagnosed with obstructive sleep apnea syndrome. Sleep 2005;8:309-14.
7. AlamI, Lewis K, Stephens JW, et al. Obesity, metabolic syndrome and sleep apnoea: all pro-inflammatory states. Obesity Reviews 2006;8:119-27.
8. Kokturk O, Ciftci TU, Mollarecep E, et al. Elevated C-reactive protein levels and increased cardiovascular risk in patients with obstructive sleep apnea syndrome. Int Heart J 2005;5:801-9.
9. Sasanabe R, Banno K, Otake K, et al. Metabolic syndrome in Japanese patients with obstructive sleep apnea syndrome. Hypertens Res 2006;29:315-22.
10. Millman RP, Redline S, Carlisle CC, et al. Daytime hypertension in obstructive sleep apnea. Prevalence and contributing risk factors. Chest 1991;99:861-6.
11. Isaksson H, Svanborg E. Obstructive sleep apnea syndrome in male hypertensives, refractory to drug therapy. Nocturnal automatic blood pressure measurements-an aid to diagnosis? Clin Exp Hypertens A 1991;13:1195-212.
12. Peker Y, Carlson J, Hedner J. Increased incidence of coronary artery disease in sleep apnoea: long-term follow-up. Eur Respir J 2006;28:596-602.
13. Wierzbicka A, Rola R, Wichniak A, et al. The incidence of sleep apnea in patients with stroke or transient ischemic attack. J Physiol Pharmacol 2006;57:385-90.
14. Grigg-Damberger M. Why a polysomnogram should become part of the diagnostic evaluation of stroke and transient ischemic attack. J Clin Neurophysiol 2006;23:21-38.
15. Eisensehr I, Ehrenberg BL, Noachtar S, et al. Platelet activation, epinephrine, and blood pressure in obstructive sleep apnea syndrome. Neurology 1998;51:188-95.
16. Resta O, Carratu P, Carpagnano GE, et al. Influence of subclinical hypothyroidism and T4 treatment on the prevalence and severity of obstructive sleep apnoea syndrome (OSAS). J Endocrinol Invest 2005;28:893-8.
17. Kjelsberg FN, Ruud EA, Stavem K. Predictors of symptoms of anxiety and depression in obstructive sleep apnea. Sleep Medicine 2005;6:341-6.
18. Guilleminault C, Partinen M, Hollman K, et al. Familial aggregates in obstructive sleep apnea syndrome. Chest 1995; 107:1545-51.
19. Philips B, Kryger MH. Management of obstructive sleep apnea-hypopnea syndrome: overview. In: Kryger MH, Roth T, Dement WC, Eds. Principles and practice of sleep medicine. 4th Ed. Philadelphia: Elsevier Saunders, 2005:1109-21.
20. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14:540-5.
21. Hora F, Napolis LM, Daltro C, et al. Clinical, anthropometric and upper airway anatomic characteristics of obese patients with obstructive sleep apnea syndrome. Respiration 2007;74:517-24.
22. Ferguson AK, Ono T, Lowe AA, et al. The relationship between obesity and craniofacial structure in obstructive sleep apnea. Chest 1995;108:375-81.
23. Bednarek M, Plywaczewski R, Jonczak L, et al. There is no relationship between chronic obstructive pulmonary disease and obstructive sleep apnea syndrome:a population study. Respiration 2005;72:142-9.
24. Radwan L, Maszczyk Z, Koziorowski A, et al. Control of breathing in obstructive sleep apnoea and in patients with the overlap syndrome. Eur Respir J 1995;8:542-5.