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KLİNİK ÇALIŞMA
RESEARCH ARTICLE

The association of body mass index values with severity and phenotype of sleep-disordered breathing

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ABSTRACT

The association of body mass index values with severity and phenotype of sleep-disordered breathing

Introduction: *To investigate the relationship between body mass index (BMI) and the severity of obstructive sleep apnea (OSA) and to determine the BMI cut-off values for sleep-disordered breathing among adult population.*

Materials and Methods: *Data from 515 patients were evaluated retrospectively. These included demographic data, BMI, apnea-hypopnea index (AHI), oxygen saturation (SaO_2) and oxygen desaturation index (ODI). The BMI cut-off value for sleep-disordered breathing was determined and comparisons were made between two groups of patients (BMI ≤ 33 and BMI > 33). Descriptive and comparative analyses were performed using SPSS, version 24.*

Results: *Higher BMI values were found to be correlated with diagnosis and severity of OSA and reduced sleep efficiency. Patients in the BMI > 33 group had significantly higher rates of co-morbid diseases than patients in the BMI ≤ 33 group. Patients with BMI ≤ 33 had significantly lower ODI values than patients with BMI > 33. In patients with BMI > 33, arousal index was significantly higher and SaO_2 values were lower than those with BMI ≤ 33. In rapid eye movement (REM) sleep-related OSA, BMI values were higher than positional/classical OSA.*

Conclusion: *Patients with higher BMI experienced frequent nocturnal oxygen desaturation periods resulting in higher arousal indexes and decreased sleep*

efficiency. REM sleep-related OSA and high BMI values together may lead to increased nocturnal oxygen demand. We recommend the threshold values of BMI > 33 to be considered for screening OSA among adult population.

Key words: *Body mass index; obstructive sleep apnea; sleep quality; rapid eye movement (REM) sleep; ROC curve*

ÖZ

Beden kitle indeks değerlerinin uykuda solunum bozukluğunun ağırlığı ve fenotipi ile ilişkisi

Giriş: *Çalışmamızda amaç, beden kitle indeksi (BKİ) değerlerinin obstrüktif uyku apnesi (OSA)'nde ağırlık derecesi ve fenotipleri ile ilişkisini incelemek ve erişkin hasta grubunda uykuda solunum bozukluğunu işaret eden BKİ eşik değerini öngörmekti.*

Materyal ve Metod: *Beş yüz on beş hastanın verileri retrospektif olarak incelendi. Demografik veriler, BKİ, apne-hipopne indeksi (AHİ), oksijen satürasyon değerleri (SaO_2), oksijen desatürasyon indeksi (ODİ) ve arousal indeks verileri kaydedildi. Uykuda solunum bozukluğunu işaret eden BKİ eşik değeri (eşik değer: 33) saptandıktan sonra, çalışma hastaları iki ayrı gruba ayrılarak (BKİ ≤ 33 ve BKİ > 33) karşılaştırmalı analizler yapıldı.*

Bulgular: *Yüksek BKİ değerlerinin hem uyku apnesi tanısında hem hastalık ağırlığı hem de azalmış uyku etkinliği ile korele olduğu görüldü. BKİ > 33 olan grupta diğer gruba göre istatistiksel olarak anlamlı derecede komorbid hastalık varlığı görüldü. ODİ değerleri BKİ ≤ 33 olan grupta diğer gruba oranla daha düşük olarak izlendi. BKİ > 33 olan grupta arousal indeksin istatistiksel olarak anlamlı derecede yüksek olduğu ve SaO_2 değerinin diğer gruba oranla daha düşük olduğu izlendi. Ayrıca REM-ilişkili uyku apnesi hastalarında, BKİ değerlerinin pozisyonel/klasik OSA'ya göre BKİ değerlerinin daha da yüksek olduğu gözlendi.*

Sonuç: *BKİ değerleri yüksek olan hastalar, daha fazla sıklıkla yüksek arousal indeks ve azalmış uyku etkinliği ile sonuçlanan noktürnal oksijen desatürasyonu deneyimlemektedir. REM uykusu ilişkili OSA ve yüksek BKİ değerleri birlikteliği artmış noktürnal oksijen ihtiyacına yol açmaktadır. Ayrıca çalışmamızın bir diğer sonucu da OSA tarama çalışmalarında BKİ > 33 değerinin eşik değer olarak göz önüne alınabileceğini ortaya koymuştur.*

Anahtar kelimeler: *Beden kitle indeksi; obstrüktif uyku apnesi; uyku kalitesi; REM uykusu; ROC eğrisi*

INTRODUCTION

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder affecting approximately 20-30% of men and 10-15% of women in the general population (1,2). It is associated with collapse of upper airways, intermittent hypoxemia, resulting in arousals during the night and excessive daytime sleepiness (EDS) (3). Increased risk of cardiovascular diseases including stroke, myocardial infarction, cardiac arrhythmias and hypertension are the major pathological consequences of sleep-related breathing disorders (1,4,5). Sleep apnea and its health-related consequences can greatly impact the quality of life of patients suffering from this condition. Although the exact mechanisms underlying the consequences of OSA are unknown, increased oxidative stress, activation of sympathetic nervous system, tissue hypoxia and sleep fragmentation have been suggested as causative factors (2).

Symptoms of OSA usually progress within years; therefore, underestimation and delay of diagnosis is a common problem among patients with OSA. Polysomnography (PSG), including the use of electroencephalogram, electrooculography, and electromyography is the gold standard for diagnosis of OSA. Risk factors for OSA include male gender, obesity, advanced age, alcohol and cigarette consumption, morphological variations in upper air ways leading to narrowing in the diameters (3). A previous prevalence study among adult Turkish population indicated that sleep disorders were significantly more common in the subjects who have lower education status, lower average income, smoking habit and obesity (6). Another study examined the relationship between anthropometric obesity indexes such as waist (WC) and neck circumference index (NC), body mass index (BMI) and OSA in Turkish adult population and found that BMI, WC, and NC enlargement were significant risk factors for OSA development (7).

OSA had increased incidence over the last few decades in developed countries due to the changes in lifestyle along with an increase in the prevalence of obesity (8,9). A relationship between OSA and obesity has been reported in earlier studies suggesting that two conditions closely interact with each other (10-12). In recent years, several human population studies have suggested that obesity may be fostered by metabolic, inflammatory, cardiovascular, and neurologic abnormalities (13). The enlargement of adipocytes in obesity may exceed the normal oxygen diffusion distance, thus compromising the effective oxygen supply from the vasculature and leading to localized hypoxia. Therefore, OSA-associated chron-

ic intermittent hypoxia in obesity may exacerbate adipose tissue hypoxia and produce further adipose tissue inflammation and dysfunction (14,15). The aim of this study was to investigate the relationship between BMI and the severity of sleep apnea using polysomnographic parameters and to determine the cut-off values of BMI for sleep disordered breathing (SDB) among adult population.

MATERIALS and METHODS

Study Design and Subjects

This study was a retrospective, non-interventional investigation conducted in a single center. The study group consisted of 515 consecutive patients. Data of patients over one year (2017) with complaints of sleep symptoms who underwent polysomnographic examination were evaluated retrospectively. Routine laboratory questionnaires, including Epworth Sleepiness Scale (ESS) for determining EDS and Sleep Quality Questionnaire for detailed questioning of sleep habits and complaints were employed (16,17). Demographic data, sleep complaints, history with accompanying illnesses, history of smoking and alcohol consumption, duration of symptoms, and body measurements were recorded prior to conduction of the polysomnographic test. Apnea-hypopnea index (AHI), OSA fenotype, sleep efficiency (%), sleep latency (minutes), oxygen saturation (SaO_2) with average and Nadir values and oxygen desaturation index (ODI) were also recorded.

Polysomnography and BMI Calculation

All patients underwent video-assisted full-night polysomnography (Neuron- Spectrum-5, Neurosoft Sleep Systems, Ivanovo, Russia). All of the PSG's were scored according to the scoring guidelines of American Academy of Sleep Medicine (AASM) (18). For all patients, a minimum of 6 hour PSG data were recorded. Subjects whose AHI was ≥ 5 were determined as having OSA. Oxygen saturation was detected by an oximeter. Respiratory movements were measured by chest and abdominal belts. Both oro-nasal termistor and nasal pressure sensor were used to detect respiratory events. Sleep stages were scored in 30-second epochs according to criteria of AASM (6). Body Mass Indexwas calculated as weight divided by the square of height (kg/m²) (19). (patients who are overweight were considered as: BMI between 25 and 29.9 kg/m², while people with obesity: BMI of 30 kg/m² or more) (19).

Inclusion Criteria

This included adult patients > 18 years who had at least one major complaints of sleep-related breathing disorder. Full night polysomnogram with a total sleep time of > 4 hours.

Exclusion Criteria

Patients who suffer from mental retardation or psychiatric disorders leading to lack of cooperation with full night polysomnography and questionnaires were excluded.

Statistical Analysis

Descriptive statistics were performed for all data. Kolmogorov-Smirnov normality test was used to assess the normality of data distribution. Chi-square test was used to test whether the BMI correlates with gender, OSA, duration of symptoms, co-morbid diseases and ESS. Mann-Whitney U test was usedto test whether the BMI has a correlation with desaturation index, arousal index, and min SaO_2. Spearman's rho test was used in order to reveal the correlation between BMI values and sleep efficiency and latency. The receiver operating characteristic (ROC) curve analysis was applied to find the ideal cut-off value for BMI to determine SDB. The area under the ROC curve is a measure of how well a parameter can distinguish between two diagnostic groups (diseased/normal). All the statistical analysis were performed by using SPSS software version 24.0 (IBM, Chicago, USA). Statistical significance was accepted at $p< 0.05$.

Ethical Commitee Approval

Ethics committee approval was received for this study from the local ethics committee (dated July 2018 Number: 07/72). All subjects provided written informed consent. The study was conducted from the beginning of 2017 until the end of the year and it was conducted in accordance with the declaration of Helsinki latest update (2013).

RESULTS

Table 1 summarizes the demographics and descriptive data of the study population. Of the 515 patients, 170 (33%) were female and 345 (67%) were male. Mean age ± standard deviation was 47.8 ± 11.3. After polysomnographic evaluation, 172 (33.4%) subjects were found to be normal with AHI < 5 while the remaining 343 subjects (66.6%) were diagnosed to have SDB

Table 1. Descriptive statistical data of participants (n= 515)

	Mean	SD	Minimum	Maximum	p value*[a]
Age	47.8	11.3	19.0	77.0	0.01
BMI (kg/m²)	30.6	5.2	17.6	55.5	0.00
Height	169.5	10.1	11.5	197.0	0.00
Weight	87.6	15.0	50.0	170.0	0.01
Waist circumference (cm)	107.9	10.9	74.0	160.0	0.00
Neck circumference (cm)	40.5	2.9	10.0	54.0	0.00
AHI	11.7	13.6	0.2	102.7	0.00
Sleep latency	23.8	20.3	0.9	141.0	0.00
Sleep efficiency (%)	89.8	9.0	35.0	99.0	0.00
Arousal index	14.6	11.7	0.1	150.2	0.00
ODI	21.0	22.4	0.3	149.3	0.00
Mean SaO_2 (%)	92.0	0.03	74.0	99.0	0.00
Nadir SaO_2 (%)	79.0	0.1	50.0	96.0	0.00

[a] Calculated using Kolmogorov-Smirnov test.
* Significance value at p< 0.05.
SD: Standard deviation, ODI: Oxygen desaturation index, AHI: Apnea hypopnea index.

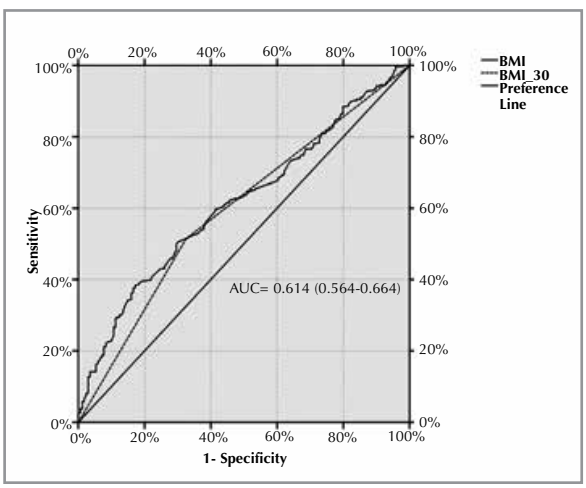

Figure 1. ROC curve presenting the cut-off value for BMI for the diagnosis of OSA.

with OSA distribution type of mild: 226 (65.9%), moderate: 66 (19.2%) and severe: 51 (14.9%).

Figure 1 illustrates the ROC curve presenting the cut-off value for BMI. There was an impact power of BMI about foreseeing SDB patients with AHI > 5 [AUC: 0.614 (0.564-0.664), p< 0.05)]. According to this curve, ideal BMI cut-off value for the diagnosis of OSA was 33 kg/m² [AUC: 0.588 (0.538-0,639)/ p< 0.05)]. Therefore, further analysis was conducted to compare between the data of two groups: patients with BMI values of ≤ 33 and those with BMI > 33.

Table 2 shows the comparison of the two groups according to BMI values (BMI ≤ 33 and BMI > 33). Increased BMI values were significantly correlated with the severity of OSA. There was no significant difference in means of duration of symptoms and ESS values between patients in the two groups. However, BMI > 33 group had significantly higher rates of co-morbid diseases than BMI ≤ 33 group (p< 0.001). The ODI values in BMI ≤ 33 group were significantly lower compared to the BMI > 33 group (p< 0.001), indicating that participants with high BMI have more frequent desaturation of oxygen during sleep. This result also reflects the significantly lower arousal index in the BMI ≤ 33 group when compared to the BMI > 33 group (p < 0.001). In BMI > 33 group, Nadir SaO_2 values were found to be significantly lower than that of the BMI ≤ 33 group, with values of 77% and 84%, respectively (p< 0.001).

The PSG results revealed the sleep efficiency and latency of the individuals. There was a statistically significant negative correlation between BMI values and the sleep efficiency (p= 0.007) and (r= -0.119), i.e. an increase in BMI was associated with a decrease in sleep efficiency. Similarly, there was a positive correlation between sleep latency and BMI; however, this correlation was not statistically significant (p= 0.069) and (r= 0.80). In REM sleep-related OSA, BMI values (33.5 ± 5.5) were significantly higher than positional OSA (30.5 ± 5.2) and classical OSA (31.4 ± 5.7) kg/m² (p< 0.05).

Table 2. Comparison of two groups according to BMI values

		BMI ≤ 33		BMI > 33		
		N	%	N	%	p value*[b]
Gender	Female	106	26.9	64	52.9	0.000
	Male	288	73.1	57	47.1	
OSAS	Absent (habitual snorer)	153	38.8	19	15.7	0.000
	Mild	173	43.9	53	43.8	
	Moderate	42	10.7	24	10.8	
	Severe	26	6.6	25	20.7	
Duration of symptoms	< 1 year	59	15.0	12	9.9	0.155
	> 1 year	334	85.0	109	90.1	
Co-morbid diseases	Present	194	49.2	82	67.8	0.000
	Absent	200	50.8	39	32.2	
Epworth sleepiness scale (ESS)	< 10	311	78.9	90	74.4	0.291
	> 10	83	21.1	31	25.6	

[b] Calculated using Chi-square test (Pearson Chi-Square).

	BMI ≤ 33		BMI > 33		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	p value*[c]
ODI	18.38 ± 19.67	10.88 (0.31-149.34)	29.37 ± 28.12	17.14 (1.04-116.03)	0.000
Arousal index	13.58 ± 11.51	11.49 (0.10-150.2)	18.11 ± 11.53	15.45 (0.30-66.3)	0.000
Minimum O_2	0.81 ± 0.09	0.84 (0.50-0.96)	0.73 ± 0.12	0.77 (0.50-0.89)	0.000

[c] Calculated using Mann-Whitney U test.
* Significance value at p< 0.05.
ODI: Oxygen desaturation index, SD: Standard deviation.

DISCUSSION

In our study, there was a clear positive correlation between BMI and SDB in patients with AHI > 5. According to ROC curve, the ideal BMI cut-off value-indicating OSA diagnosis was found to be 33 kg/m². This value can be considered as Class 1 or 'mild obesity' (BMI between 30 and 34.9 kg/m²) rather than being 'overweight' according to an earlier study (20). In a study on Chinese population, authors recommended using a BMI with a cut-off value (28 kg/m^2) to allow the anesthetists to identify patients with high risk of OSA (21). It is important to note that BMI thresholds are defined differently for people of Asian population (obesity is BMI of 27.5 kg/m² or more) (20). In another study focusing on acute ischemic patients with OSA in New Zealand, the mean BMI was reported as 30 ± 7 kg/m² (22). This indicates that the BMI cut-off values for SDB varies among different patient population due to variations in the anthropometric obesity indexes.

When ODI values were compared, subjects with BMI ≤ 33 kg/m² had significantly lower ODI values than subjects in the BMI > 33 kg/m² group indicating that subjects with high BMI have more frequent desaturation of oxygen during sleep period. This result is also associated with low arousal index in the BMI ≤ 33 kg/m² group. In BMI > 33 kg/m² group, Nadir SaO_2 values were found to be lower than BMI ≤ 33 kg/m² group; with values of 77% and 84%, respectively. Similar to our findings, a recent study showed that higher BMI values were correlated with lower Nadir SaO_2 during overnight polysomnography (23). Nakano et al. also demonstrated that the diagnostic sensitivity and specificity of the ODI for OSA depends on BMI (24). An earlier study in Turkish adult population demonstrated that the average BMI, WC and NC of patients with OSA were statistically higher than those of normal subjects (7).

Our finding of an escalation in OSA risk with increased BMI value supports the results of earlier studies (7,23). In the adult population, the prevalence of OSA is estimated to be ~25%, and as high as 45% in subjects with obesity (25). In our study, we have

found that patients with BMI > 33 were diagnosed with OSA in 75.3% of cases (43.8% mild, 10.8% moderate and 20.7% severe OSA). Previous studies suggest that approximately 25% of adults with a BMI between 25 kg/m^2 and 28 kg/m^2 have at least mild OSA (AHI ≥ 5) (25). The prevalence of OSA in patients with obesity/severe obesity is nearly twice that of normal-weight adults (26). Furthermore, patients with mild OSA who gain 10% of their baseline weight are at a six fold-increased risk of progression of OSA, and an equivalent weight loss can result in a more than 20% improvement in the severity of OSA (26). It is known that obesity may worsen OSA due to fat deposition at specific areas of the body. Fat deposition in the tissues surrounding the upper airway appears to result in a smaller lumen and increased collapsibility of the upper airway, predisposing to apnea (27). Moreover, fat deposits around the thorax leading to truncal obesity reduce chest compliance and functional residual capacity, and may increase oxygen requirement (28). Although there is compelling evidence showing that obesity, as well as visceral obesity may predispose to OSA, and that losing weight results in the improvement of OSA, recent studies suggest that OSA may itself cause weight gain (29). Weight gain has been reported shortly after the diagnosis of OSA suggesting a reciprocal relationship. Factors such as increased appetite, reduced activity, diet refined carbohydrates, may conceivably contribute to weight gain in OSA patients (11).

Our findings revealed that in REM sleep-related OSA, BMI values were significantly higher than BMI values inpositional and classical OSA phenotype.This is in contrast to the results by Sakao et al. who found a lower mean BMI among REM-related OSA patients compared to those with NREM-related OSA in a Japanese population based study (30). The discrepancies between these studies may be due to difference in anthropometric measurements or genetic differences between Caucasians and Japanese populations.

In view of the established relationship between increased BMI and SDB, patients diagnosed with OSA and have high BMI values should be referred to dietary programs besides primary treatment modalities. Lifestyle modification especially focused in reduced-calorie diet together with physical activity and behavioral therapy to achieve weight loss is essential for management of these patients. In our patient population, we recommend the cut off value of BMI higher than 33 kg/m² to be considered for screening people for OSA in our population.

CONCLUSION

Obesity can lead to sleep-disordered breathing resulting in sleep fragmentation with decreased sleep efficiency rates. It is considered an important factor that causes worsening of OSA. We demonstrated close correlation between increased BMI values andthe diagnosis and severity of OSA, with a cut-off value of BMI > 33 as determinant for SDB. Patients with higher BMI experienced more frequent nocturnal oxygen desaturation periods resulting in higher arousal indexes and had decreased sleep efficiency than normal subjects. In REM sleep-related OSA, the BMI values were found to be higher than in case of positional and classical OSA phenotypes. REM sleep-related OSA and high BMI values together may increase nocturnal oxygen demand. Based on these results, we suggest that the threshold BMI values higher than 33 kg/m² tobe considered in screening people for OSA. These patients must be provided with counseling regarding appropriate life-style modifications and weight reduction strategies.

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CONFLICT of INTEREST

There is no conflict of interest related to this study.

AUTHORSHIP CONTRIBUTIONS

Concept/Design: ÇÖ, TÖ, MHT

Analysis/Interpretation: ÇÖ, TÖ, MHT, HYS

Data Acquisition: ÇÖ, TÖ

Written by: ÇÖ

Critical Revision: All of authors

Final Approval: ÇÖ, TÖ

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