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ORIGINAL ARTICLE **ÖZGÜN ARAŞTIRMA**

THE ETIOLOGICAL RELATIONSHIP OF OBSTRUCTIVE SLEEP APNEA-OBESITY AND CAROTID ATHEROSCLEROSIS

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ABSTRACT

INTRODUCTION: Obstructive sleep apnea syndrome (OSAS) is an independent risk factor for the presence of cerebral small-vessel disease, stroke and burden of cerebral atherosclerosis and it is frequently associated with comorbidities such as obesity, metabolic disorders, hypertension, coronary artery disease, circulatory insufficiency, and carotid atherosclerosis, all of which contribute to adverse effects on the cerebrovascular system. This study aimed to analyze the relationship between carotid atherosclerosis (CA) and obesity, vascular risk factors in patients with OSAS.
METHODS: In this retrospective study, we reviewed patients admitted to neurology and chest diseases outpatient clinic with sleep apnea syndrome and hospitalized during nightime for Polysomnography between September 2020-June 2023. Patients with moderate or severe OSAS who underwent bilateral carotid artery Doppler USG for any reason within six months were included in the study group. The control group included the patients meeting criteria who admitted to neurology outpatient clinic. Body Mass Index (BMI) of all patients was calculated as weight (kg) /height (m2). Patients with acute or chronic upper airway or lung infection, severe obstructive or restrictive lung diseases, acute-subacute stroke and myocardial infarction, heart or renal failure, patients with a history of major surgery within six months were also excluded.
RESULTS: According to the inclusion and exclusion criteria, 47 patients with moderate or severe OSAS and 40 patients without OSAS were recruited. Male sex and patients with obesity (BMI>30 kg/m2) were more prevalent in the study group and the difference was statistically significant (P<0,05). However, the incidence of CA was similar between all groups, there was no significant difference.
DISCUSSION AND CONCLUSION: Although obesity was more common in the OSAS group, the number of patients with CA or stenosis was similar in both groups. The findings suggest that well-known vascular risk factors and comorbidities may have a primary effect on atherogenesis, rather than a direct effect of OSAS.
Keywords: OSAS, carotid atherosclerosis, obesity, stroke, comorbidities.

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OBSTRUKTİF UYKU APNE SENDROMU, OBEZİTE VE KAROTİS ATEROSKLEROZU ARASINDAKİ ETYOLOJİK İLİŞKİ

ÖZ

GİRİŞ ve AMAÇ: Obstrüktif uyku apne sendromu (OSAS), serebral küçük damar hastalığı, inme ve serebral ateroskleroz yükü açısından bağımsız bir risk faktörüdür ve sıklıkla obezite, metabolik bozukluklar, hipertansiyon, koroner arter hastalığı, dolaşım yetmezliği ve karotis aterosklerozu gibi eşlik eden hastalıklarla ilişkilidir, bunların hepsi serebrovasküler sistem üzerinde olumsuz etkilere katkıda bulunur. Bu çalışmada OSAS'lı hastalarda karotis aterosklerozu (KA) ile obezite ve vasküler risk faktörleri arasındaki ilişkinin incelenmesini amaçladık.

YÖNTEM ve GEREÇLER: Bu retrospektif çalışmada, Eylül 2020-Haziran 2023 tarihleri arasında nöroloji ve göğüs hastalıkları polikliniğine uyku apne sendromu nedeniyle başvuran ve polisomnografi için gece yatırılan hastaları inceledik. Çalışma grubuna orta ve ağır OSAS saptanan ve son altı ay içinde karotis doppler USG çekilen hastalar dahil edildi. Kontrol grubu nöroloji polikliniğine başvuran kriterleri karşılayan hastalardan oluşturuldu. Tüm hastaların Beden Kitle İndeksi (BKİ) ağırlık (kg)/boy (m2) olarak hesaplandı. Akut veya kronik üst solunum yolu veya akciğer enfeksiyonu, ciddi obstrüktif veya restriktif akciğer hastalıkları, akut-subakut inme ve miyokard enfarktüsü, kalp veya böbrek yetmezliği olan hastalar ve son altı ay içinde majör cerrahi öyküsü olan hastalar çalışma dışı bırakıldı.

BULGULAR: Dahil etme ve dışlanma kriterlerine göre, orta veya ağır OSAS'ı olan 47 hasta ve OSAS'ı olmayan 40 hasta çalışmaya dahil edildi. Çalışma grubunda erkek cinsiyet ve obezitesi olan (BMI>30 kg/m2) hastaların oranı daha fazlaydı ve aradaki fark istatistiksel olarak anlamlıydı (P< 0,05). Ancak KA görülme sıklığı tüm gruplar arasında benzerdi, anlamlı bir fark yoktu.

TARTIŞMA ve SONUÇ: Obezite OSAS grubunda daha sık görülmesine rağmen KA veya stenozu olan hasta sayısı her iki grupta da benzerdi. Bulgular, OSAS'ın doğrudan etkisinden ziyade, bilinen vasküler risk faktörlerinin ve komorbiditelerin aterogenez üzerinde birincil bir etkiye sahip olabileceğini düşündürmektedir.

Anahtar Sözcükler: İnme, akut dönem, hastane yatışı, kaygı.

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a prevalent sleep disorder presented with reduced pharyngeal muscle tone during sleep, which causes temporary airway collapse, oxygen desaturation, frequent arousals, and sleep fragmentation (1,2). OSAS is observed in 5–15% of the adult population in developed countries and has been associated with increased cardiovascular and cerebrovascular morbidity (3). The main risk factors for OSAS are upper body obesity, increased neck perimeter, and male sex (1,4).

OSAS is an independent risk factor for cerebral small vessel disease, stroke and the burden of cerebral atherosclerosis (1,2,5). The mechanisms regarding the interaction between OSAS and cerebrovascular (CV) complications are multifactorial and controversial. This is mainly because OSAS is commonly associated with comorbidities such as obesity, metabolic disorders, hypertension, coronary artery disease, circulatory insufficiency and carotid atherosclerosis, all of which contribute to adverse effects on the CV system (1,6).

Several studies on animal models and clinical data suggests the direct and indirect effects of OSAS on the development of atherosclerosis, and OSAS is considered as a recent cardiovascular risk factor. OSAS and its main mechanisms; intermittent hypoxia, carbon dioxide (CO_2) retention, and increased sympathetic excitability, may contribute to atherogenesis by triggering endothelial dysfunction and inflammation, oxidative stress and also evolving lipid peroxidation, dyslipidemia, and insulin resistance (5,7,8).

In addition, some of epidemiological studies have indicated obesity as an independent risk factor for carotid disease; identified as carotid plaques (CP) or carotid intima-media thickness (c-IMT), with increased risk of mortality, cardiovascular and CV diseases (9,10). It is suggested that obesity increases early atherosclerotic changes and is a well known risk factor for CVD (11,12).

Obesity, described by the World Health Organization as an excessive fat accumulation, with a Body Mass Index (BMI) greater than or equal to 30 kg/m^2, is a major modifiable risk factor for CVD that significantly contributes to the progression of atherosclerosis and it can influence

the CV risk both directly and indirectly. In addition, the relation between atherosclerosis and obesity is complicated, with the inflammatory process as the major relation (12).

In this study, we aimed to analyze the interrelation between carotid atherosclerosis (CA) and obesity in patients with OSAS. There is an association between CA and obesity and also OSAS. Several studies have suggested that OSAS is an independent risk factor for CA, however, there is also a major link between obesity and atherosclerosis. Most of the patients with OSAS are obese (BMI >30 kg/m^2), so there is a complex and controversial relationship between obesity, OSAS and CA that needs to be investigated.

METHODS

Subjects: In the present retrospective study, we reviewed patients admitted to the neurology and chest diseases outpatient clinic with a complaint of sleep apnea syndrome and who were hospitalized during nighttime for the recording of Polysomnography (PSG) between September 2020 and June 2023. Patients who had moderate or severe OSAS according to our PSG results and underwent bilateral carotid arter doppler USG (CA-DUSG) for any reason within six months were included in study group. The control group was selected from the patients admitted to the neurology outpatient clinic who underwent CA-DUSG and met the exclusion criteria. The body mass index (BMI) of all patients was calculated as weight (kg) / height (m^2). Patients with BMI>30 kg/m2 were considered obese as defined by World Health Organization (WHO) criteria.

Patients who had acute or chronic upper airway or lung infection, severe obstructive or restrictive lung diseases, with acute-subacute stroke-myocardial infarction (MI) or major chronic stroke or MI, heart or renal failure and patients with a history of major surgery, such as cerebrovascular, lung and cardiovascular or ENT system were also excluded.

This study was approved by Health Sciences University Tepecik Training and Research Hospital Non-Interventional Clinical Researches Ethical Committee (Date: 20.11.2023, No: 2023/10-51), and the written consents were taken from all subjects. The study was conducted in accordance with the ethical standards of the Declaration of Helsinki.

Laboratory tests and clinical data: Laboratory tests; total cholesterol, LDL, blood urea, hemoglobin, platelet and leucocyte levels were obtained from medical records within 3 months. Demographic features including age, sex, and atherosclerotic CVD risk factors; BMI, smoking history, hyperlipidemia and comorbid vascular diseases (hypertension, diabetes mellitus, previous stroke, chronic obstructive lung disease) were recorded.

Polysomnography: Patients underwent polysomnographic investigation with either Alice 6 LDE PSG device. Recordings were conducted during nighttime, in accordance with the patient's circadian rhythm. Electrodes were placed in line compatible with the guidelines determined by the American Academy of Sleep Medicine (AASM). Audio and video recordings along with electrocardiographic, electroencephalographic, electrooculographic and electromyographic recordings were included. To detect saturation (SpO_2%), a pulse oximeter was used. The respiratory and sleep parameters were analyzed and classified compliant with the standard AASM criteria as follows: non-REM 1 sleep stage (N1), non-REM 2 sleep stage (N2), non-REM 3 sleep stage (N3), REM sleep stage, arousal index, sleep latency (SL), wake after sleep onset (WASO), sleep efficiency (SE), apnea–hypopnea index (AHI), oxygen desaturation index (ODI) and snoring.

AASM standard criteria were utilized for interpretation of PSG recordings. A 90% interruption of oronasal airflow for at least 10 s was accepted as obstructive apnea. A ≥30% decrease in airflow associated with a ≥3% reduction in oxygen saturation or arousal was considered to be hypopnea. The total number of apneas and hypopneas per hour of sleep were obtained to calculate the AHI. PSG findings were evaluated for the diagnosis of OSA, according to the guidance of the International Classification of Sleep Disorders 2. The severity of OSAS was classified as mild, moderate, or severe (5≤AHI<15, 15≤AHI<30, AHI≥30) respectively.

Doppler Ultrasonography: All patients were diagnosed by two independent radiologists blinded to research data with high-resolution B-mode colored Doppler ultrasound. The presence and the location of plaques were recorded. According to the American Society of Ecography Task Force (ASETF) recommendations, increased

carotid intima-media thickness (c-IMT) is considered early atherosclerosis (≥1.1 mm), carotid plaques and stenosis were stated as advanced stages of the atherosclerotic process. The presence of plaque is defined by focal structures protruding into the arterial lumen by at least 1.5 mm or focal narrowing of the vessel wall. The rates of carotid stenosis (normal: 0% stenosis; mild: <50% stenosis; moderate: 50–69% stenosis; severe: 70–99% stenosis) were calculated. Carotid atherosclerosis was defined as a c-IMT increase, carotid plaque and carotid stenosis in our study as per the ASETF recommendations.

Statistical analysis: All data were analyzed utilizing SPSS 24.0 software version (SPSS, Chicago, IL, USA). The results of measurements were expressed as mean±standard deviation. Student T and Chi-square tests were used to compare numeric and string variables of two groups, respectively. Binary logistic regression analysis was performed for study and control groups, correlation study was performed with Pearson's correlation analysis; p values less than 0.05 were considered to indicate statistical significance.

RESULTS

According to the inclusion and exclusion criteria, 47 patients with moderate or severe OSAS and 40 patients without OSAS who underwent CA-DUSG were recruited. The mean age of the study group was 55.63±11.39 and that of the control group was 60.67±12.87 (p=0.059).

Study group's (n=47) gender was following; male (M)/female (F)= 38/9, control group (n=40) gender: M/F= 24/16. A significant difference between the groups in terms of gender was determined (p=0.032).

The comparison of demographic and clinical data of the groups is shown in Table 1. Male sex and patients with obesity (BMI>30 kg/m^2) were more common in the study group and the difference was significant (P<0.05). As indicated, there was no difference in terms of CA among groups (p=0.597).

The comparison of laboratory tests is stated in Table 2. There was not any significant difference between the two groups in terms of LDL, total cholesterol, whole blood count parameters.

We also analyzed the study group separately and divided the group into two groups: moderate and severe OSAS. Eighteen patients had moderate OSAS and 29 patients had severe OSAS according to our PSG results. There was no significant difference between the two groups. However only patients with HT were more common in the severe OSAS group (19 versus 7), however this difference was not significant (p=0.074) (Table 3). There was also no difference in terms of CA among the groups (p=0.122).

Table 1. Comparison of demographic and clinical data.

	Study group (n=47)	Control group (n=40)	P value
Age	55.63±11.39	60.67±12.87	0.059
Gender (M/F)	38/9	24/16	0.032
HT (n)	26	26	0.359
DM (n)	11	11	0.661
HU (n)	3	2	0.782
Obesity	17	6	0.026
CA	25	19	0.597
Smoking	26	16	0.154
PS	3	6	0.188
COPD	3	5	0.325

M/F: male/female HT: hypertension DM: diabetes mellitus HU: hyperuricemia CA: carotid ather-osclerosis PS: previous stroke COPD: chronic obstructive pulmonary disease.

Table 2. Comparison of laboratory tests.

	Study group (n=47)	Control group (n=40)	P value
Hgb	13.97±1.60	14.07±1.82	0.805
Wbc	7.35±1.99	7.25±1.88	0.800
LDL	131.23-31.46	140.83-43.21	0.235
Plt	251.38-62.53	239.80-52.72	0.358
HL(n)	24	24	0.404

Hgb: hemoglobin WBC:leucocyte LDL: low density lipoprotein Plt: platelet HL: Hyperlipidemia.

Table 3. Comparison of demographic and clinical data of moderate and severe OSAS groups.

	Moderate OSAS (n=18)	Severe OSAS (n=29)	P value
Age	54.50-9.97	56.34-12.30	0.595
Gender (M/F)	13/5	24/4	0.236
HT (n)	7	19	0.074
DM (n)	3	8	0.390
HU (n)	0	3	0.158
Obesity	6	11	0.750
CA	7	18	0.122
Smoking	10	16	0.980
PS	0	3	0.158
COPD	0	3	0.158

M/F: male/female HT: hypertension DM: diabetes mellitus HU: hyperuricemia CA: carotid atherosclerosis PS: previous stroke COPD: chronic obstructive pulmonary disease.

The differences between the moderate and severe OSAS groups in terms of laboratory tests were not significant (Table 4). Only a high levels of WBC count in the severe OSAS group was

determined, which was not a significant difference (p=0.067).

Table 4. Comparison of laboratory tests of the moderate and severe OSAS groups.

	Moderate OSAS (n=18)	Severe OSAS (n=29)	P value
Hgb	14.08-1.24	13.91-1.81	0.730
Wbc	6.68-1.38	7.77-2.21	0.067
LDL	139.07-33.15	126.36-29.92	0.181
Plt	259.77-59.54	246.17-64.79	0.466
HL(n)	8	16	0.474

Hgb: hemoglobin WBC:leucocyte LDL: low density lipoprotein Plt: platelet HL: Hyperlipidemia.

DISCUSSION AND CONCLUSION

The relationship between OSAS, obesity and atherosclerosis has been shown in several studies. They suggested that OSAS has an unfavorable effect on the cardiovascular system, commonly by the elevation of arterial blood pressure, autonomic dysfunction, and alteration in lipid and glucose metabolism. Chronic intermittent hypoxia due to OSAS might be an independent risk factor for atherosclerosis leading to a proinflammatory propensity. In addition, OSAS severity is related to c-IMT and calcification of carotid arteries, has a relation with CV disease, and has been suggested as a risk factor for CA (1,4,13,14).

A series of current studies have suggested a relation between OSAS and subclinical atherosclerosis, however the definite mechanism and clinical data on OSAS and CA is still controversial. OSAS and atherosclerosis have many common risk factors, however it is debate, if their association is specific or because of comorbidities. Moreover, the inflammatory biomarkers that could clarify the association between OSAS, inflammation and atherosclerosis were not investigated in most of current studies (5).

Possible pathogenesis of CA in patients with OSAS has been asserted. First of all, an OSAS-mediated physiologic cascade may contribute to endothelial dysfunction and promote atherosclerosis and then vascular calcification by aggravating atherogenic cardiometabolic disorders. Furthermore, the vibration of snoring is conveyed through the surrounding tissues to the carotid artery wall, initiating an inflammatory pathway and thickening the arterial wall which leads to atherosclerosis and the formation of atheroma and plaque (2,13,15). In addition, intermittent hypoxia accelerates the production of reactive oxygen species in the vascular wall, elevates levels of serum inflammatory markers (such as interleukin-6 (IL-6), interleukin-8 (IL-8) and C-reactive protein (CRP)) and increases lipid uptake of macrophages, which leads to endothelial dysfunction, chronic inflammation, and atherosclerosis (2,8,5,14). Suzuki et al. demonstrated that arousals related with respiratory and non-respiratory disorders are indicated as one of the most important factors for acceleration of atherosclerotic processes in patients with moderate-to-severe OSAS. Therefore, intermittent hypoxemia seems to be the most significant factor influencing CA (14). Moreover, Ji et al. asserted that high CRP levels might play a role in the process of atherosclerosis in OSAS patients (5). The indirect mechanisms are also mediated through hypertension, insulin resistance, diabetes, and dyslipidemia, which are well-known risk factors for atherosclerosis (2,5).

In the present study, the male sex was more prevalent in the study group as expected and there was no difference between the moderate-severe OSAS and control groups in terms of carotid atherosclerosis and other CVD risk factors. This finding is different from the literature, which may be because of the data of the control group included elderly patients with known vascular risk factors such as HT, HL and DM.

On the other hand, obesity is the most commonly mentioned risk factor for the development of OSAS. In patients with a BMI >40 kg/m^2, the prevalence of OSAS has been reported to be between 40% and 90 (7). Similar to the general population, in our study obesity (BMI>30 kg/m^2) was significantly more prevalent in patients with moderate-severe OSAS than in the control group (p=0.026), but we could not analyze other values of anthropometric measurements, since the study was retrospective. Obesity could be associated with carotid plaque instability and progression, but its relation with carotid symptomatology has not been proven and should be researched in future studies (10). The Emerging Risk Factors Collaboration analyzed 221.934 patients from 58 cohorts and suggested that BMI, waist circumference (WC), and waist-to-hip ratio (WHR) were all associated with CVD risk, and the researchers found that their impact on CVD risk were similar. However, in the Rotterdam and the INTERSTROKE and INTERHEART Study, it was suggested that WHR and WC had a stronger

association with the progression of CA and also the occurrence of stroke and myocardial infarction cases than general obesity respectively (16,10). According to our study, obesity is a risk factor for OSAS, but we cannot suggest that it may be related to the progression or propensity of CA.

CA-DUSG was utilized in our study because it is non-invasive and easy to apply. Arterial atherosclerosis has traditionally been evaluated using Doppler USG; c-IMT and carotid plaque consistently predict future CVD events and in current literature there are several studies using these measurements in OSAS patients (13).

In this study, we did not determine a significant difference in terms of CA in either group, and the frequency was quite similar. Additionally, we did not find any difference when comparing the groups separately, such as severe and moderate OSAS.

Although it was not statistically significant, blood WBC levels were higher and HT was more prevalent in the severe OSAS group than in the moderate OSAS group (p=0.067 and p=0.074 respectively). This result might be significant if the number of cases was larger.

As known, OSAS is an independent risk factor for stroke, and continuous positive airway pressure (CPAP) treatment decreases the risk and improves recovery after stroke. In addition, CPAP treatment may also reduces central blood pressure and arterial stiffness. OSAS and atherosclerosis coexist in most of patients. Therefore OSAS is more common in patients with carotid artery stenosis which is often related with elderly patients, obesity and vascular risk factors (1). The mechanism by which OSAS is an independent risk factor for generalized atherosclerosis is through studies demonstrating that CPAP treatment can significantly reduce affected carotid vascular morphology; namely c-IMT, a risk factor of CVD (4,5,16). However, the definite relationship between OSAS and atherosclerosis is still controversial and inflammation is stated as a common feature of OSAS and atherosclerosis (5).

There are some limitations to our study. First, it is retrospective and the case number is small. Second, serum levels of inflammatory markers, especially CRP, were not analyzed. Third, the findings of c-IMT increase, carotid plaque and stenosis could be analyzed and divided into groups for further statistical analysis.

The results of our study, unlike the literature, suggest that OSAS may not be an independent risk factor for CA and that its effect on atherosclerosis may be related to comorbidities and vascular risk factors triggered by obesity and OSAS. Although obesity was more common in the OSAS group, the number of patients with CA or stenosis was similar in both groups, suggesting that well-known vascular risk factors and comorbidities may have a primary effect on atherogenesis. To explain the main effect of OSAS in accelerating atherosclerosis, the association between CA and inflammatory factors, comorbidities and vascular risk factors (such as HT, DM, HL, obesity) should be investigated with prospective randomized large studies.

REFERENCES

1. Nahorecki A, Postrzech-Adamczyk K, Święcicka-Klama A, et al. Prevalence of sleep apnea in patients with carotid artery stenosis. Adv Exp Med Biol 2019; 1211: 69-75.
2. Woo HG, Song TJ, Jung JS, et al. Association between the high risk for obstructive sleep apnea and intracranial carotid artery calcification in patients with acute ischemic stroke. Sleep Breath 2021; 25(1): 299-307.
3. Deol R, Lee KA, Kanaya AM, et al. Obstructive sleep apnea risk and subclinical atherosclerosis in South Asians living in the United States. Sleep Health 2020; 6(1): 124-130.
4. Chang TI, Lee UK, Zeidler MR, et al. Severity of obstructive sleep apnea is positively associated with the presence of carotid artery atheromas. J Oral Maxillofac Surg 2019; 77(1): 93-99.
5. Ji P, Kou Q, Zhang J. Study on relationship between carotid intimamedia thickness and inflammatory factors in obstructive sleep apnea. Nat Sci Sleep 2022; 14: 2179-2187.
6. Cuspidi C, Tadic M, Gherbesi E, et al. Targeting subclinical organ damage in obstructive sleep apnea: A narrative review. J Hum Hypertens 2021; 35(1): 26-36.
7. Szymanski FM, Gorko D, Platek AE, et al. Prevalence of obstructive sleep apnea in patients with peripheral arterial diseases. Sleep Breath 2020; 24(3): 1035-1041.
8. Wang J, Yu W, Gao M, et al. Impact of obstructive sleep apnea syndrome on endothelial function, arterial stiffening, and serum inflammatory markers: An updated meta-analysis and metaregression of 18 studies. J Am Heart Assoc 2015; 4(11): e002454.
9. Vidal-Perez R, Franco-Gutiérrez R, Pérez-Pérez AJ, et al. Subclinical carotid atherosclerosis predicts all cause mortality and cardiovascular events in obese patients with negative exercise echocardiography. World J Cardiol 2019; 11(1): 24-37.
10. Ferreira J, Cunha P, Carneiro A, et al. Is obesity a risk factor for carotid atherosclerotic disease? - opportunistic review. J Cardiovasc Dev Dis 2022; 9(5): 162.
11. Seo DH, Cho Y, Seo S, et al. Association between metabolically healthy obesity and subclinical atherosclerosis in the Cardiovascular and Metabolic Diseases Etiology Research Center (CMERC) Cohort. J Clin

Med 2022; 11(9): 2440.
12. Rychter AM, Naskręt D, Zawada A, et al. What can we change in diet and behaviour in order to decrease carotid intima-media thickness in patients with obesity? J Pers Med 2021; 11(6): 505.
13. Koh TK, Kang EJ, Bae WY, et al. Quantitative analysis of carotid arterial calcification using airway CT in obstructive sleep apnea. Auris Nasus Larynx 2019; 46(4): 559-564.
14. Suzuki M, Shimamoto K, Sekiguchi H, et al. Arousal index as a marker of carotid artery ather-osclerosis in patients with obstructive sleep apnea syndrome. Sleep Breath 2019; 23(1): 87-94.
15. Chuang HH, Liu CH, Wang CY, et al. Snoring sound characteristics are associated with common carotid artery profiles in patients with obstructive sleep apnea. Nat Sci Sleep 2021; 13: 1243-1255.
16. Imahori Y, Mathiesen EB, Morgan KE, et al. The association between anthropometric measures of adiposity and the progression of carotid atherosclerosis. BMC Cardiovasc Disord 2020; 20(1): 138.

Ethics

Ethics Committee Approval: The study was approved by Health Sciences University Tepecik Training and Research Hospital Non-Interventional Clinical Researches Ethical Committee (Date: 20.11.2023, No: 2023/10-51),

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