**The Relation of Polysomnographic Parameters to Carotid and Brachial Artery Intima-media Thickness in Patients with Severe Obstructive Sleep Apnea Syndrome**

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**ABSTRACT**

**Objective:** To evaluate carotid (C) and brachial (BA) artery intima-media thickness (IMT) in patients with severe obstructive sleep apnea syndrome (OSAS) along with factors predicting CIMT and BA-IMT increase.

**Method:** A total of 81 patients with severe OSAS (Mean (SD) age: 48.0(10.5) years, 70.4% were males) and 49 healthy controls (Mean (SD) age: 47.9(9.7) years, 73.5% were males) were included. Data on demographics, cardiovascular risk factors and CIMT and BIMT values were evaluated in each subject along with factors predicting CIMT and BIMT increase in the patient group.

**Result:** Mean (SD) left and right CIMT and BIMT were significantly higher in OSAS than control group ($p<0.001$ for each). There was no significant association of polysomnographic parameters with CIMT. Average apnea duration was associated with increase in right and left BA-IMT (B=0.011, p=0.011 for the right and B=0.011, p=0.002 for the left). A significant negative association was noted between average oxygen saturation and left BA-IMT (B=-0.012, p=0.005).

**Conclusion:** Our findings revealed significant increase in of CIMT and BA-IMT in patients with severe OSAS compared to controls. None of the polysomnographic parameters, but age and smoking, was determinants of increased CIMT, while apnea duration and arousal index were significant predictors of BA-IMT.

**Keywords:** Brachial artery, cardiovascular risk factors, carotid artery, intima-media thickness, obstructive sleep apnea syndrome, polysomnography.

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## INTRODUCTION

Obstructive Sleep Apnea Syndrome (OSAS) is a highly prevalent airways disease (1) characterized by recurrent episodes of cessation of breathing during sleep leading to hypoxia–reoxygenation sequences (2, 3) and associated with significant cardiovascular morbidity and mortality (2, 4) mostly related to atherosclerosis (5).

Hypoxia, hypercapnia, micro-arousals, sympathetic hyperactivity, oxidative stress, systemic inflammation and hyper-coagulability are considered amongst the pathophysiological consequences of the disease and implicated in the development of hypertension, endothelial dysfunction and higher intima-media thickness (IMT) values that predispose to development of atherosclerosis (1).

Hence, while the precise mechanism underlying the link between OSAS and cardiovascular diseases has not been fully explained (1), the role of early atherosclerosis has been proposed among the intermediary mechanisms that could explain the link between OSAS and cardiovascular morbidity (4).

Early stages of atherosclerosis consist of a functional impairment of endothelial surface with a consequent impairment of arterial vasodilation capacity and the thickening of intima-media space (6, 7). Based on its significant relation to presence as well as severity of coronary atherosclerosis, measurement of carotid IMT (CIMT) has been a validated parameter for detecting subclinical atherosclerosis (6, 7). It has recently been shown that brachial artery IMT (BA-IMT) was also correlated to CIMT and thereby might serve as a marker of cardiovascular risk (8).

Given the importance of determination of subclinical atherosclerosis in patients with OSAS to prevent the adverse cardiovascular events, to investigate its relation with endothelial dysfunction, an important early physiological event in atherosclerosis, is reasonable. The present study was designed to evaluate CIMT and BA-IMT in patients with OSAS in

comparison to healthy controls and to investigate polysomnographic parameters predicting the CIMT and BA-IMT increase and thereby the early atherosclerosis.

## METHODS

*Study population*

A total of 81 patients diagnosed with severe OSAS (mean (SD) age: 48.0(10.5) years, 70.4% were males) and 49 healthy control subjects (mean (SD) age: 47.9(9.7) years, 73.5% were males) were included in the present study. Patients with the diagnosis of severe OSA as confirmed by polysomnography, without known cardiovascular disease, and any vasoactive treatment were included. Having a disease potentially affecting BP regulation (Parkinson disease, renal or cardiac transplantation, cardiac heart failure, dm ve hipertansiyon), atrial fibrillation or frequent premature beats (10/min), previous treatment of OSA with continuous positive airway pressure (CPAP), oral appliances, or maxillofacial surgery were the exclusion criteria. Control group was composed of patients who underwent polysomnography due to the complaint of snoring, while polysomnography revealed normal findings with AR index of <10 and AHI of <5.

Written informed consent was obtained from each subject following a detailed explanation of the objectives and protocol of the study, which was conducted in accordance with the ethical principles stated in the "Declaration of Helsinki" and approved by the institutional ethics committee.

*Assessments*

Control and patient groups were compared in terms of demographic characteristics (age, gender), biochemical (low-density lipoprotein (mg/dL), triglyceride (mg/dL), high-density lipoprotein (mg/dL), total cholesterol (mg/dL), C-Reactive protein (CRP, mg/dL)) and

hematological (hematocrit (%) and hemoglobin (g/dL) ) findings along with mean (SD) CIMT and BA-IMT measurements. Data on body mass index ($kg/m^2$), smoking and lipid profile were evaluated as cardiovascular risk factors in each patient with polysomnographically confirmed OSAS. Additionally, linear regression of polysomnographic parameters, patient demographics and cardiovascular risk factors to CIMT and BA-IMT were evaluated in the patient group to determine the factors predicting IMT increase via regression models.

*IMT measurement*

IMT measurement was performed via B-mode ultrasonography (Applio Ultrasound, Toshiba Medical Systems, Tokyo, Japan) with a sectorial probe of 7.5 MHz with axial and lateral resolution of 0.15 mm and in accordance with the methodology previously described (9,10). Examinations and readings were performed by trained and certified blinded sonographers and readers. The values of IMT for left and right common carotid and brachial arteries were recorded in any subject.

*Polysomnography*

The sleep analysis included overnight polysomnography, which documented the sleep disturbances and severity of the OSAS according standard criteria (11). The investigation was performed with EMBLA 4500 (Cogent Technologies, USA) monitoring system. According to the known diagnostic standards, the minimal time for examination was 6 h. For the documentation of the sleep, we used standard 14 channel polysomnography, including electroencephalogram (C3-A2, C4-A1, O1-A2, O2-A1), electro-oculograms, electromyograms (EMG) of the left/right extremity, electrocardiogram (ECG), heart rate, nasal and oral air flow, thoracic and abdominal movements, registration of snoring, position of the body, pulse oxymetry monitored oxygen saturation (SaO2) and a polysomnography with video-watching. The sleep phases and arousals were analyzed in conformity with

Rechtschaffen and Kales' criteria (12). All the results were analyzed manually. The breathing was registered by nasal pressure cannulas and combined respiratory inductive plethysmography, which uses composed signal and a thermistor. Apneas and hypopneas were evaluated in accordance with the accepted international criteria (11). The apnea index (AI) was defined as the number of apneas per hour sleep while hypopnea index (HI) — the number of hypopneas per 1 h sleep. The apnea/hypopnea index (AHI), combined the number of apnea and hypopnea per 1 h sleep. The OSAS diagnosis was made on the basis of an apnea/hypopnea index (AHI) $\geq$ 5 with consideration of severe OSAS for AHI values >30 (1). The oxygen desaturation index was defined as the number of events in which oxygen saturation falls below 90% per hour of sleep (11). The oxygen desaturation index, basal oxygen saturation and average oxygen saturation values were also recorded. Arousals were identified as abrupt shifts of at least 3-s duration in electroencephalogram frequency. An arousal index was defined as the number of arousals per hour of sleep.

*Statistical analysis*

Statistical analysis was made using computer software (SPSS version 20.0.0.1, SPSS Inc. Chicago, IL, USA). Chi-square ($\chi^2$) test was used for the comparison of qualitative data, Student's t or Mann Whitney U tests for quantitative variables. Linear regression was used entering each covariate in a distinct model and the results of mutually adjusted models were presented. Significance level was set at 0.05.

**RESULTS**

Comparison of OSAS and control groups in terms of demographic and clinical characteristics CIMT and BA-IMT OSAS and control groups were homogenous in terms of mean(SD) age

and gender (Table 1). Values for right and left CIMT and BA-IMT were significantly higher in OSAS than control group ($p<0.001$ for each) (Table 1).

Hemoglobin ($p<0.001$), hematocrit ($p<0.001$), triglyceride ($p<0.001$), HDL ($p=0.022$), total cholesterol ($p=0.001$) and C-reactive protein ($p<0.001$) levels were significantly higher in OSAS than the control group (Table 1).

*Polysomnographic parameters*

Polysomnographic findings in OSAS group are presented in Table 2. The mean apnea hypoapnea index (AHI) of the OSAS group was 64.7(24.6) per hour sleep, the mean oxygen saturation $SaO_2$% was 86.8(4.4), the minimum oxygen saturation was 72.3(9.9) and the arousal index was 15.3(11.2).

*The predictive role of polysomnographic parameters, patient demographics and cardiovascular risk factors on CIMT*

Based on results of mutually adjusted model; no significant association of polysomnographic parameters with CIMT is observed. Whereas age was associated with increase in right and left CIMT ($B=0.004$, $p=0.034$ for right and $B=0.005$, $p=0.036$ for left) and being smoker was associated with increase in right CIMT ($B=0.084$; $p=0.034$) (Table 3).

*The predictive role of polysomnographic parameters, patient demographics and cardiovascular risk factors on BA-IMT*

Based on results of mutually adjusted model; average apnea duration was associated with increase in right and left BA-IMT ($B=0.011$, $p=0.011$ for the right and $B=0.011$, $p=0.002$ for the left). Average oxygen saturation was negatively associated with left BA-IMT ($B=-0.012$, $p=0.005$), respectively. Age was associated with increase in right BA-IMT ($B=0.003$, $p=0.016$) (Table 4).

## DISCUSSION

Our findings concerning the relation of polysomnographic parameters, demographics and cardiovascular risk factors to CIMT and BA-IMT in a large group of patients with severe OSAS revealed the presence of carotid and brachial arterial wall hypertrophy in patients compared with controls. None of the polysomnographic parameters predicted the increase in CIMT, whereas average apnea duration and arousal index were the best polysomnographic predictors of an increase and decrease in BA-IMT, respectively. Age was a common predictor for the increase in both CIMT and BA-IMT, whereas being active smoker for CIMT was the significant predictor of arterial wall hypertrophy.

Thickening of the intima-media complex of carotid as well as brachial arteries among severe OSAS patients in our study population is in agreement with the past studies suggesting a connection between OSAS and the progression of the atherosclerotic cerebrovascular disease (13, 14). However, although CIMT values were significantly increased in our patients with severe OSAS predicting the increased risk of early atherosclerosis, polysomnographic parameters were not the significant determinants of this association. Apneic episodes have been reported to trigger cardiovascular, hemodynamic and hemorrhagic changes as potential promoters for stroke incidence in patients with cardiovascular risk factors (15), while the oxygen desaturation that accompanies the apneic episodes has been shown to lead to generative changes of the artery wall (13). Hence, given the association of BA-IMT to average apnea duration and arousal index, our findings indicate the predictive role of longer apnea duration and lesser sleep fragmentation on brachial but not carotid artery wall hypertrophy, while oxygen saturation index had no influence on either CIMT or BA-IMT. Similar to our findings, increased atherosclerosis burden was reported to occur in the absence of significant oxygen desaturation, suggesting a significant role of sleep fragmentation in coronary atherosclerosis (16).

Dyslipidemia, BMI, smoking and the cigarettes smoked per day have been considered amongst the parameters with possible influence on the IMT (1). Based on the epidemiological data, patients with OSAS are often associated with obesity, diabetes and dyslipidemia, arterial hypertension and active smoking (1, 13). OSAS patients compared with BMI-matched control group that were selected among healty volunteeers with no disease and absance of any patients with diabetus and hypertension in our study.

Dyslipidemia and obesity were the outstanding co-morbidities in our patients with severe OSAS, whereas only active smoking were determined as the significant predictors of an increase in left BA-IMT and right CIMT, respectively.

Notably, age was the common significant predictor of both BA-IMT and CIMT in our study population, which supports the statement that the severity of OSAS was related to subclinical atherosclerosis irrespective of the presence of other cardiovascular risk factors except for age. Accordingly, the presence of OSAS has been suggested to be a marker of subclinical atherosclerosis, dependent on increasing age and the severity of subclinical atherosclerosis that correlates with the severity of OSAS (17). However, being entirely composed of severe OSAS patients, mean carotid IMT values measured in our study population were lower than past reports (4, 13, 18-20), while similar to some reports conducted with patients devoid of important risk factors such as obesity, diabetes, or hypertension (2) and patients during the early course of the disease who were relatively free of confounding factors (5).

A significant relationship between carotid IMT and oxygen desaturation severity in OSAS was reported in study populations (18-20). The absance of any patients with diabetus and hypertension in our study population supports that carotid remodeling was also seen in the absence of these comorbidities (2), given the lack of significant association between CIMT increase and polysomnographic variables, our findings are not in line with the

suggestion that significant effect of apnea-induced intermittent hypoxia rather than co-morbidities was evident in carotid remodeling (1, 2).

The increased risk for developing adverse outcomes attributable to OSAS was reported to be independent of age, obesity, cholesterol and blood pressure levels in past cohort studies (21, 22). However, despite they were suffering from severe OSAS, CIMT values in our patients were lower than reported in past studies and predictive role of age and smoking but not sleep parameters was noted on CIMT increase. Accordingly, our findings emphasize the need for further investigation of factors responsible in exacerbation and the progression of existent carotid remodeling and support the lack of evidence considering the independent association of mild to-moderate sleep disorders with subclinical atherosclerosis (23).

Likewise, the results of a past study in a large sample of community dwelling subjects showed loss of meaningful associations of sleep-disordered breathing (SDB) indices with carotid plaques or carotid IMT evidence after adjusting for CVD risk factors, implying confounded crude associations (23). Indeed, the inclusion of patients with varying degrees of sleep disorder ranging from mild to moderate SDB to severe OSAS in those studies supporting a positive association (20, 24, 25) has been suggested to explain the report of discrepant results (25). However, no association was documented between CIMT and sleep parameters in our patient population even it was composed entirely of patients with severe OSAS who had moderate rates of concomitant degree of obesity and dyslipidemia.

Being able to be measured simultaneously to flow-mediated vasodilation (FMD) assessed in the same brachial artery, BA-IMT was shown to significantly correlate to a cumulative cardiovascular risk index for heart attack as well as to carotid IMT (8). Our findings support that brachial IMT may be a marker of the grade of atherosclerosis and

vascular function, providing additive information for stratifying subjects with cardiovascular risk factors and of value in the assessment of atherosclerosis (8, 26).

Based on the predictive role of apnea duration and arousal index on BA-IMT rather than CIMT, endothelial function may be assumed not to be uniformly affected by exposure to intermittent hypoxia or apnea events in patient with OSA (27). In this regard, given that brachial not carotid IMT was associated with sleep parameters in our patients with severe OSAS, our findings indicate the divergent vascular responses to obstructive apneas and intermittent hypoxia in OSA population (28) and are in agreement with the statement that the thickening process of the intima-media of the carotid artery might be different from the atherosclerotic process that occurs in the coronary artery, whereas the mechanism responsible for endothelial dysfunction of the brachial artery parallels the atherosclerotic process of the coronary artery (29).

BA-IMT was demonstrated to be associated with cardiovascular risk factors and coronary artery disease (CAD) and the prognostic value of BA-IMT was shown in terms of late cardiovascular events in patients admitted for stable angina on multivariate Cox regression analysis, BA-IMT >0.37mm (odds ratio=1.96; p=0.03), remained significantly associated with cardiovascular events (30).Accordingly, our findings related to predictive role of apnea duration and arousal index on BA-IMT values should be cautiously interpreted given that average 0.26 (left) and 0.27 (right) mm of BA-IMT in in our patients with severe OSAS.

Nevertheless, given the predictive role of polysomnographic parameters on BA-IMT but smoking on CIMT in our patients, our findings emphasize the predictive role of IMT in early atherosclerosis commonly in arteries at different levels of conduit system in OSAS patients but with the likelihood of different mechanism underlying endothelial dysfunction of

the brachial and carotid arteries with respect to atherosclerotic process of the coronary artery (29).

Not only intermittent hypoxia due to sleep apnea but also sleep fragmentation or respiratory efforts occurring during obstructed breaths has been accused for the complex pathophysiology underlying OSAS dependent atherosclerosis (4). Consistent with the determinant role of arousal index, marker of sleep fragmentation, on the decrease in BA-IMT in our study, dissecting these different mechanisms via better definition of the specific roles of sleep fragmentation vs. oxygen desaturation on coronary atherosclerosis (16) has been considered likely help in targeting specific treatments beyond the suppression of sleep disordered breathing (4).

Certain limitations to this study should be considered. First, due to the cross-sectional design, no conclusions can be made about causal relationship. Second, due to concomitant presence of OSAS related disorders which are known to be more important markers of atherosclerosis than OSAS itself among patients with OSAS, the possibility of these disorders to primarily underlie CIMT increase in OSAS patients seems quite likely and remains to be investigated in randomized trials. Nevertheless, based on a large population of patients with polysomnographically confirmed severe OSAS, our findings represent a valuable contribution to the literature in terms of detection of the early atherosclerosis in arteries at different levels of conduit system in OSAS patients and the determinant role of polysomnographic parameters.

In conclusion, our findings in a large population of patients with polysomnographically confirmed severe OSAS revealed significant thickening of IMT of the common carotid and brachial arteries compared to controls. None of the polysomnographic parameters were determinants of increased CIMT, whereas the apnea duration and arousal index were significant predictors of associated BA-IMT. Although both CIMT and BA-IMT

were significantly higher in OSAS patients than controls as predicted by the increased age, the two differed in terms of their relation to polysomnographic parameters and smoking . In this regard, our findings emphasize the consideration of the likelihood of different mechanism responsible for endothelial dysfunction of the brachial artery and carotid artery based on the atherosclerotic process of the coronary artery (29). Hence, since IMT seems to predict early atherosclerosis commonly in arteries at different levels of conduit system in OSAS patients, whether or not the determinant role of polysomnographic parameters was evident, to be able conclude the thesis of the role of OSAS as an independent risk factor for atherosclerosis, further studies are required to better define potential relationships between sleep disorders and atherosclerosis progression as well as the specific roles of sleep fragmentation vs. oxygen desaturation on coronary atherosclerosis.

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Table 1: Comparative demographic and clinical data of the studied population in three groups.

| | | **Group 1 (n=80) (OSAS)** | **Group 2 (n=80) (COPD)** | **Group 3 (n=37) (Healthy controls)** | ***p* Value** |
|---|---|---|---|---|---|
| Age (years) | | 48.1±10.4 | 58.3±8.9 | 47.6±9.7 | p1vs2<0.001<br>p1vs3=0.784<br>p2vs3<0.001 |
| Gender | Female | 24 (30%) | 27 (33.8%) | 27 (73%) | <0.001* |
| | Male | 56 (70%) | 53 (66.2%) | 10 (27%) | |
| PAP | <20 mmHg | 62 (77.5%) | 44 (55%) | 37 (100%) | <0.001* |
| | ≥20 mmHg | 18 (22.5%) | 36 (45%) | 0 | |
| DM | No | 73 (91.3%) | 71 (88.8%) | 37 (100%) | 0.113 |
| | Yes | 7 (8.7%) | 9 (11.2%) | 0 | |
| HT | No | 71 (88.8%) | 72 (90%) | 37 (100%) | 0.112 |
| | Yes | 9 (11.2%) | 8 (10%) | 0 | |
| Smoking | No | 52 (65%) | 27 (33.8%) | 30 (81.1%) | <0.001* |
| | Yes | 28 (35%) | 53 (66.2%) | 7 (18.9%) | |

Pearson Chi-Square Test

(**Abbreviations:** OSAS= Obstructive sleep apnea syndrome; COPD= Chronic obstructive pulmonary disease; PAP= Pulmonary artery pressure; DM= Diabetes mellitus; HT= Hypertension; *: statistically significant)

Table 2: Results of IMT measurements, laboratory and respiratory data of three groups

| | **Group 1 (n=80) (OSAS)** | **Group 2 (n=80) (COPD)** | **Group 3 (n=37) (Healthy controls)** | ***p* Value** |
|---|---|---|---|---|
| Right C-IMT (mm) | 0.70±0.15 | 0.70±0.30 | 0.40±0.20 | p1vs2=0.706<br>p1vs3<0.001<br>p2vs3<0.001 |
| Left C-IMT (mm) | 0.60±0.10 | 0.70±0.20 | 0.40±0.10 | p1vs2=0.144<br>p1vs3<0.001<br>p2vs3<0.001 |
| Right B-IMT (mm) | 0.30±0.10 | 0.40±0.15 | 0.20±0.10 | p1vs2=0.724<br>p1vs3<0.001<br>p2vs3<0.001 |
| Left B-IMT (mm) | 0.30±0.10 | 0.3 ±0.1 | 0.10±0.10 | p1vs2=0.647<br>p1vs3<0.001<br>p2vs3<0.001 |
| Hb (g/dl) | 16.3±2.0 | 15.6±2.4 | 14.9±1.6 | p1vs2=0.049<br>p1vs3=0.001<br>p2vs3=0.082 |
| Hct | 48.5±6.7 | 47.9±7.7 | 44.9±5.0 | p1vs2=0.599<br>p1vs3=0.010<br>p2vs3=0.030 |
| LDL (mg/ml) | 124.3±36.8 | 105.8±37.6 | 111.5±32.0 | p1vs2=0.001<br>p1vs3=0.079<br>p2vs3=0.426 |
| HDL (mg/ml) | 39.7±10.3 | 43.2±41.9 | 44.3±11.8 | 0.620 |
| Cholesterol (mg/ml) | 204.0±47.4 | 174.5±48.4 | 180.0±39.6 | p1vs2<0.001<br>p1vs3=0.010<br>p2vs3=0.548 |
| TG (mg/ml) | 189.5±110.0 | 123.5±87.5 | 97.0±76.0 | p1vs2<0.001<br>p1vs3<0.001<br>p2vs3=0.014 |
| CRP (mg/ml) | 0.62±0.64 | 0.92±1.0 | 0.40±0.41 | p1vs2=0.001<br>p1vs3=0.001<br>p2vs3<0.001 |
| D-dimer (mg/ml) | 123.0±91.0 | 141.0±153.0 | 110.0±77.0 | p1vs2=0.008<br>p1vs3=0.559<br>p2vs3=0.086 |
| EF (%) | 62.50±5.00 | 60.00±5.50 | 65.00±8.0 | p1vs2<0.001<br>p1vs3=0.052<br>p2vs3<0.001 |
| Oxygen saturation (%) | 87.0±5.0 | 85.0±6.0 | 97.0±2.0 | p1vs2<0.001<br>p1vs3<0.001<br>p2vs3<0.001 |

Kruskal-Wallis Test (Post Hoc Test: Bonferroni corrected Mann-Whitney U test α=0,017)

(**Abbreviations:** OSAS= Obstructive sleep apnea syndrome; COPD= Chronic obstructive pulmonary disease; C-IMT= Carotid artery intima media thickness; B-IMT= Brachial artery intima media thickness; Hb= Hemoglobin; Hct= Hematocrit; LDL= Low density lipoproteins; HDL= High density lipoproteins; TG= Triglycerides; CRP= C-reactive protein; EF= Ejection fraction; min= Minutes

**Table 3** The predictive role of polysomnographic parameters, patient demographics and cardiovascular risk factors on carotid intima-media thickness

| | | **Carotid intima-media thickness** | | | |
|---|---|---|---|---|---|
| | | ***Right*** | | ***Left*** | |
| **Polysomnographic variables** | | **B** | ***p*** | **B** | ***p*** |
| Apnea-hypopnea index | | -0.001 | 0.212 | -0.002 | 0.173 |
| Night-time oxygen saturation index | | 0.0003 | 0.817 | 0.003 | 0.082 |
| Lowest oxygen saturation | | -0.003 | 0.424 | 0.002 | 0.729 |
| Average oxygen saturation | | 0.008 | 0.295 | 0.010 | 0.359 |
| Arousal index | | -0.002 | 0.245 | -0.002 | 0.434 |
| Average apnea duration | | -0.002 | 0.716 | 0.005 | 0.508 |
| Longest apnea duration | | 0.0004 | 0.638 | 0.001 | 0.329 |
| Duration of nocturnal oxygen saturation < 90% | | 0.00004 | 0.849 | 0.00032 | 0.271 |
| **Demographics** | Age (years) | 0.004 | **0.034** | 0.005 | **0.036** |
| | Gender | -0.043 | 0.365 | -0.047 | 0.471 |
| **Cardiovascular risk factors** | | | | | |
| Being smoker | | 0.084 | **0.034** | 0.056 | 0.288 |
| Hypertension | | -0.027 | 0.695 | -0.009 | 0.921 |
| Diabetes mellitus | | -0.045 | 0.530 | -0.128 | 0.187 |
| Body mass index (kg/m$^2$) | | 0.002 | 0.677 | -0.004 | 0.485 |
| Low-density lipoprotein (mg/dL) | | 0.0002 | 0.746 | -0.0004 | 0.574 |
| Triglyceride (mg/dL) | | -0.0001 | 0.353 | 0.00001 | 0.944 |
| High-density lipoprotein (mg/dL) | | 0.003 | 0.149 | 0.004 | 0.183 |
| Total cholesterol (mg/dL) | | -0.0002 | 0.700 | -0.0003 | 0.692 |

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|---|---|---|---|---|
| C-Reactive protein (mg/dL) | -0.001 | 0.961 | 0.020 | 0.613 |

Regression coefficients reported here were adjusted for other independent variables in the mutually adjusted model. B: adjusted regression coefficient

**Table 4** The predictive role of polysomnographic parameters, patient demographics and cardiovascular risk factors on brachial artery intima-media thickness

| | | Brachial artery intima-media thickness | | | |
|---|---|---|---|---|---|
| | | *Right* | | *Left* | |
| **Polysomnographic variables** | | **B** | ***p*** | **B** | ***p*** |
| Apnea-hypopnea index | | -0.001 | 0.385 | -0.001 | 0.307 |
| Night-time oxygen saturation index | | 0.002 | 0.103 | 0.001 | 0.429 |
| Lowest oxygen saturation | | 0.003 | 0.312 | 0.004 | 0.070 |
| Average oxygen saturation | | -0.001 | 0.846 | -0.012 | **0.016** |
| Arousal index | | -0.0002 | 0.889 | -0.002 | 0.132 |
| Average apnea duration | | 0.011 | **0.011** | 0.011 | **0.002** |
| Longest apnea duration | | 0.0003 | 0.660 | 0.0001 | 0.830 |
| Duration of nocturnal oxygen saturation < 90% | | 0.0001 | 0.677 | 0.00002 | 0.870 |
| **Demographics** | Age (years) | 0.003 | **0.041** | 0.002 | 0.138 |
| | Gender | -0.037 | 0.314 | -0.001 | 0.973 |
| **Cardiovascular risk factors** | | | | | |
| Being smoker | | -0.010 | 0.747 | 0.013 | 0.590 |
| Hypertension | | 0.061 | 0.239 | 0.079 | 0.062 |
| Diabetes mellitus | | 0.084 | 0.124 | 0.148 | **0.001** |
| Body mass index (kg/m$^2$) | | -0.001 | 0.714 | -0.003 | 0.228 |
| Low-density lipoprotein (mg/dL) | | -0.0004 | 0.330 | -0.0004 | 0.239 |
| Triglyceride (mg/dL) | | 0.0001 | 0.404 | -0.00002 | 0.751 |
| High-density lipoprotein (mg/dL) | | 0.0000 | 0.997 | -0.001 | 0.318 |
| Total cholesterol (mg/dL) | | 0.0001 | 0.722 | 0.001 | 0.108 |
| C-Reactive protein (mg/dL) | | 0.010 | 0.659 | 0.020 | 0.273 |

Regression coefficients reported here were adjusted for other independent variables in the mutually adjusted model. B: adjusted regression coefficient

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