Relationship between Cardio-Ankle Vascular Index (CAVI) and Obstructive Sleep Apnea (OSA)

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Background and Objective: *A non-invasive test called Cardio-Ankle Vascular Index (CAVI) measures aortic stiffness, which is an early sign of atherosclerosis. Obstructive sleep apnea (OSA) has a close association with cardiovascular mortality and morbidity. We sought to assess the relationship between OSA and arterial stiffness.*
Material and Method: *Seventy-one patients with OSA (apnea-hypopnea index AHI ≥5, mean age 51.5±14.1 years, 27 females) and 11 controls (AHI <5, mean age 56.8±11.8 years, 5 females) were enrolled in the study. In all subjects, arterial stiffness (CAVI) was performed and recorded along with blood pressure, pulse pressure of brachial arteries, and ankle arteries.*
Results: *The demographic data of the patients with OSA and controls were not significantly different. Subjects with OSA demonstrated higher values of mean ankle artery pulse pressure than the controls (73.1±14.6 vs. 59.6±6.1 mmHg, respectively), but arterial stiffness and CAVI had no statistically significant difference (7.47±1.68 vs. 7.25±1.61, respectively).*
Conclusion: *There was no relationship between arterial stiffness CAVI, and the presence of OSA. However, there was a significant association between ankle artery pulse pressure and the presence of OSA.*

Keywords: *CAVI, OSA, Pulse pressure*

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Several techniques, such as flow-mediated dilatation, pulse wave velocity (PWV), and carotid intima media thickness (IMT), and a noninvasive and automatic method of measuring the brachial-ankle PWV (baPWV) were applied as an indicator for local and systemic arterial stiffness parameter in several studies[1,2].

This new index was named as the cardio-ankle vascular index (CAVI), which reflects the overall stiffness of aorta, femoral artery and tibial arteries, thus reflecting the stiffness of a considerable length of the artery[3], It is not affected by blood pressure at the time of measurement. Many previous studies have demonstrated the significance of arterial stiffness as a surrogate marker for determining the prognosis of the cardiovascular disease[4].

Obstructive sleep apnea (OSA) is associated with increased cardiovascular morbidity and mortality[5,6]. Several comorbidities of OSA, such as obesity, hypertension, and dysglycemia, play a significant indirect role in the relationship between OSA and the development of cardiovascular diseases[7,8]. And it was recently reported that several pathogenetic mechanisms linked to OSA, such as inflammation, increased the production of reactive oxygen species, and endothelial dysfunction, might directly contribute to the progression of vascular damage, which is strongly associated with future cardiovascular events[9].

A recent study[1] study in a University of Medicine in Turkey has concluded that a patient with OSA exhibited increased aortic stiffness without the coexistence of classic vascular risk factors and correlated this with the severity of OSA (according to RDI and/or the severity of nocturnal arterial desaturation).

However, data from patients with OSA in Sleep Center of The Faculty of Medicine Siriraj Hospital indicated, the patients with OSA have hypertension, diabetes mellitus, hyperlipidemia, coronary heart disease and cerebrovascular disease, that are often found together. Therefore, in this research we study the relationship between the severity OSA

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and cardio-ankle vascular index (CAVI) to determine whether, it can be used or not.

Material and Method

Study protocol

The authors selected 89 patients referred for evaluation of snoring and possible sleep apnea between January 2012 and March 2013. All patients were hospitalized in the sleep laboratory for a diagnostic overnight study. Before the sleep study, patients completed a questionnaire regarding their medical and sleep history and current medications. This information was subsequently reviewed by a physician during a follow-up visit to the sleep clinic. Additional data that were collected during the sleep study included patient demographics and anthropomorphic measurements. The study was approved by our Local Ethics Committee, and written informed consent was obtained from each participant.

Criteria for exclusion

Exclusion criteria were impaired cardiorespiratory function, defined as the occurrence of respiratory failure, bronchopulmonary infection, or congestive heart failure in the previous two months.

Sleep study

Overnight polysomnography was performed on all patients. The equipment consisted of a Compumedics, which recorded the following channels: central electroencephalogram, electrooculogram, chin electromyogram, pulse oximeter, chest and abdominal excursion, airflow (by oronasal thermistry), single bipolar electrocardiogram and body position. The respiratory disturbance index (RDI) was defined as the total number of apneas and hypopneas per hour during sleep time. Apnea was defined as a reduction in airflow to <25% of baseline for >10 seconds, and hypopnea was defined as a decrease in airflow or thoracoabdominal excursion to <70% of baseline for >10 seconds, associated with a 4% fall in oxyhemoglobin saturation. Sleep staging was performed according to the criteria of Rechtschaffen and Kales[10]. OSA was defined in patients with an apnea-hypopnea index (AHI) ≥5, and controls as an AHI <5.

Blood pressure

All patients had blood pressure measured in the supine position with a mercury sphygmomanometer, to determine SBP, DBP, MAP, and pulse pressure of brachial and ankle artery.

Arterial stiffness

CAVI reflects the stiffness of the whole arterial segment composted of the aorta, femoral artery, and tibial artery. With the patient lying supine, an electrocardiogram is and heart sounds are monitored. Pulse wave velocity (PWV) from the heart to the ankle is obtained by measuring the length from the origin of the aorta to the ankle. CAVI can be calculated from PWV at the origin of the aorta to the ankle portion of the tibial artery, systolic and diastolic blood pressure is measured at the upper brachial artery.

Blood sampling

Blood tests during this year including total cholesterol, Triglyceride, HDL cholesterol, LDL cholesterol, fasting glucose, HbA_1C blood urea nitrogen, and creatinine were recorded.

Statistics

All analyses were performed by the computerized SPSS 16 package program (Statistical Package for Social Sciences, SPSS). Results are given as means ± SD. Student's t test categorical data were compound using Chi-square test or fisher's exact test, as appropriate, was used to compare continuous variables.

Logistic regression analysis to assess the strength of association between variables. Multivariate regression analysis was used to identify determinants of CAVI and to evaluate the interaction between OSA-group and controls-group. The strength of these relationships was expressed using the β-coefficient and the *p*-value. A $p<0.05$ was considered statistically significant.

Results

Basic characteristics

Overall, 89 patients were enrolled into study. From Sleep study, seven patients who had been diagnosed with upper airway resistant syndrome (increase respiratory effort without apnea or hypopnea) were excluded. The remaining 82 patients were analyzed.

The characteristics of the participants are summarized in Table 1. There was no statistically significant difference between the patients with OSA and controls ($p>0.05$) with respect to age, gender, height, weight, body mass index (BMI), underlying disease (HTN, DM, hyperlipidemia, CAD, and Ischemic stroke), or plasma sampling (total cholesterol, triglyceride, LDL, FBS, BUN, and creatinine).

Table 1. Clinical parameters between patients with OSA and control

Characteristics	Control (AHI <5) (n = 11)	OSA (AHI ≥5) (n = 71)	*p*-value
Female, n (%)	5 (45.5)	27 (38.0)	0.743
Age (years), mean ± SD	51.5±14.1	56.8±11.8	0.185
Height (cm), mean ± SD	163.1±8.2	163.2±9.0	0.980
Weight (kg), mean ± SD	71.1±17.4	77.6±20.3	0.322
BMI (kg/m^2), mean ± SD	26.5±5.2	28.9±6.2	0.234
HTN, n (%)	3 (27.3)	35 (49.3)	0.173
DM, n (%)	2 (18.2)	13 (18.3)	1.000
Hyperlipidemia, n (%)	3 (27.3)	30 (42.3)	0.512
CAD, n (%)	1 (9.1)	5 (7.0)	1.000
Ischemic stroke, n (%)	0 (0)	3 (4.2)	1.000
BUN (mg/dl), mean ± SD	15.2±7.0	15.4±6.8	0.939
Cr (mg/dl), mean ± SD	0.98±0.30	0.95±0.27	0.790
FBS (mg/dl), mean ± SD	131.5±57.3	105.4±22.5	0.318
TC (mg/dl), mean ± SD	170.0±36.0	182.3±44.7	0.490
TG (mg/dl), mean ± SD	105.8±40.4	128.7±63.5	0.359
HDL (mg/dl), mean ± SD	62.1±14.9	51.5±14.7	0.082
LDL (mg/dl), mean ± SD	86.6±27.6	101.5±34.3	0.276

OSA = obstructive sleep apnea; AHI = apnea-hypopnea index; BMI = body mass index; HTN = hypertension; DM = diabetes mellitus; CAD = coronary artery disease; BUN = blood urea nitrogen; Cr = creatinine; FBS = fasting blood sugar; TC = total cholesterol; TG = triglyceride; HDL = high-density lipoprotein; LDL = low-density lipoprotein

Table 2 provides information about the values of parameters when compared between the OSA group (AHI ≥5) (OSA patient) and Controls group (AHI <5).

From Table 3, a multivariate analysis found that the mean ankle arterial pulse pressure of OSA-group was higher than the controls-group with statistical significance. The mean ankle arterial MAP and mean CAVI of the two groups did not differ statistically.

Discussion

Recent research[1] study in a University of Medicine in Turkey has concluded that the patient with OSA exhibited increased aortic stiffness without coexistence of classic vascular risk factors and correlated with the severity of OSA. That can be used in Thailand or not.

This paper showed that CAVI were not significantly different between the OSA-group and controls-group, but are not the same as the research above. This difference might be explained by the following reasons.

First: The patients in control-group were not normal. They had other diseases with symptoms suspicious for obstructive airway diseases (such as snoring and drowsiness during the day) that were reasons for doing a sleep study. After that, if the result indicated an AHI <5, they were placed in the control group. Therefore, the control group was not composed of normal persons.

Second: Many patients had co-morbid diseases such as hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, and cerebrovascular disease. The modification of these risk factors (weight reduction[11], blood glucose control[12] (ARB[13], CCB[14]), lipid-lowering agent[14,15], or stopping smoking[16]) can reduce arterial stiffness or CAVI. This can explain why the CAVI of the two groups are not different.

Third: Recent research[1] study in a University of Medicine in Turkey has concluded that the patient with OSA exhibited increased aortic stiffness without coexistence of classic vascular risk factors. Consequently, CAVI help to detect subclinical atherosclerotic in OSA. However, in the present study[17-19], many patients have co-morbid disease as mentionned above. In several studies, it is shown that these co-morbid diseases worsen vascular elasticity

Table 2. Parameters between patients with OSA and controls

Characteristics	Controls (AHI <5) n = 11	OSA (AHI ≥5) n = 71	*p*-value	OR	95% CI
R_B-sBP (mmHg), mean ± SD	120.6±15.8	130.1±16.5	0.081	1.040	0.995-1.087
L_B-sBP (mmHg), mean ± SD	120.9±14.3	129.0±13.9	0.081	1.047	0.994-1.102
Mean_B-sBP (mmHg), mean ± SD	120.7±14.6	129.5±14.7	0.071	1.046	0.996-1.099
R_B-PP (mmHg), mean ± SD	48.0±11.2	54.0±11.6	0.109	1.058	0.988-1.134
L_B-PP (mmHg), mean ± SD	46.6±11.2	53.2±11.4	0.078	1.074	0.992-1.162
Mean_B-PP (mmHg), mean ± SD	48.2±11.2	53.6±10.8	0.124	1.062	0.984-1.146
R_B-MAP (mmHg), mean ± SD	89.5±13.4	96.7±13.4	0.104	1.046	0.991-1.104
L_B-MAP (mmHg), mean ± SD	92.8±10.2	95.0±11.5	0.545	1.018	0.960-1.080
Mean_B-MAP (mmHg), mean ± SD	91.1±11.1	95.8±11.9	0.224	1.038	0.977-1.103
R_A-sBP (mmHg), mean ± SD	129.8±15.1	147.1±19.7	0.011	1.061	1.013-1.110
L_A-sBP (mmHg), mean ± SD	133.9±19.2	146.6±18.3	0.043	1.045	1.001-1.091
Mean_A-sBP (mmHg), mean ± SD	131.8±16.6	146.9±18.7	0.020	1.056	1.009-1.105
R_A-PP (mmHg), mean ± SD	59.4±7.4	73.7±15.0	0.006	1.099	1.027-1.176
L_A-PP (mmHg), mean ± SD	59.8±7.9	72.5±15.2	0.014	1.074	1.015-1.136
Mean_A-PP (mmHg), mean ± SD	59.6±6.1	73.1±14.6	0.008	1.096	1.024-1.172
R_A-MAP (mmHg), mean ± SD	93.2±9.1	103.6±13.8	0.024	1.071	1.009-1.137
L_A-MAP (mmHg), mean ± SD	96.5±15.5	103.9±13.4	0.108	1.047	0.990-1.108
Mean_A-MAP (mmHg), mean ± SD	94.9±11.8	103.7±13.0	0.042	1.066	1.002-1.133
Mean CAVI, mean ± SD	7.25±1.61	7.47±1.68	0.689	1.080	0.740-1.578

R_B = right brachial artery; L_B = left brachial artery; Mean_B = mean brachial artery; R-A = right ankle artery; L_A = left ankle artery; Mean_A = mean ankle artery; sBP = systolic blood pressure; MAP = mean arterial pressure; PP = pulse pressure; CAVI = cardio-ankle vascular index

Table 3. Multivariate analysis between patients with OSA and controls

Parameter	*p*-value	OR	95% CI
Mean_A-PP	0.029	1.099	1.010-1.196
Mean_A-MAP	0.598	1.021	0.944-1.105
Mean CAVI	0.339	0.807	0.519-1.253

or increased arterial stiffness. Therefore, we may not be able to see the relationship of CAVI with OSA patients.

Fourth: The sample size was too small.

Pulse pressure in the OSA-group is greater than the controls-group and is statistically significant. Based on earlier research, that pulse pressure is a valuable surrogate marker for arterial stiffness[20]. In conclusion, the patients with OSA have higher arterial stiffness than normal persons, so pulse pressure will be higher too.

Both pulse pressure and CAVI are used to measure arterial stiffness as well, but on the pulse pressure with CAVI to match with OSA patients, so that the relationship was not the same.

Normally, both systolic and diastolic blood pressures tend to increase with age[21]. However, beyond the age of 50 or 60, there is no further increase in diastolic blood pressure and, in many cases it actually declines. Thus, with increasing age, the pulse pressure widens. In our research, although the average age of the two groups did not differ significantly statistically the OSA-group tends to be older than average. Therefore, the pulse pressure may not be described by arterial stiffness because of a single OSA. However, it may be explained by age parameters that are more likely to occur than in OSA-group. Therefore, that can cause the pulse pressure to be higher than in the controls-group.

The pulse pressure may be higher than the actual measurement because the problems include the normal amplification of the pressure wave as it travels from the aorta to the periphery, although this effect becomes less pronounced with increasing age. As can

be seen from the present study, the pulse pressure of the ankle artery is higher than pulse pressure of the brachial artery. A number of studies have shown that pulse pressure is better predictor of coronary heart disease risk than either systolic or diastolic pressure alone, in the over-50 seconds[22]. But central pressure is the more accurate predictor of risk than peripheral blood pressure[23]. Therefore, pulse pressure alone is inadequate to assess arterial stiffness accurately, especially when using ankle artery alone.

Limitation

The limitations of the present study are: First, many patients have co-morbid diseases such as hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, and cerebrovascular disease that were confounding factors interfering with the interpretation of the data. Second: the sample size is too small. Therefore, a further longitudinal study in subjects diagnosed with OSA without co-morbid disease should be done with true normal subjects for comparison.

What is already known on this topic?

The ankle arterial pulse pressure had significant correlation with the presence of OSA. There was no correlation between CAVI and OSA.

What this study adds?

The presence of comorbidities and their modification may affect the CAVI result in patients with symptoms suspected OSA.

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Potential conflicts of interest

None.

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การศึกษาความสัมพันธ์ระหว่างดัชนีวัดความยืดหยุ่นของหลอดเลือดแดง (cardio-ankle vascular index; CAVI) กับภาวะทางเดินหายใจอุดกั้นขณะนอนหลับ

ศศิธร ร่มสายหยุด, วัฒนชัย โชตินัยวัตรกุล, ธนัญญา บุณยศิรินันท์

ภูมิหลังและวัตถุประสงค์: *ค่าพารามิเตอร์ตัวใหม่ที่ใช้วัดค่าดัชนีความยืดหยุ่นของหลอดเลือดแดง หรือ cardio-ankle vascular index (CAVI) สามารถตรวจพบความผิดปกติในผู้ป่วยที่มีความเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดโดยที่ยังไม่แสดงอาการ และพบว่าโรคทางเดินหายใจอุดกั้นขณะหลับ (Obstructive sleep apnea, OSA) นั้นเพิ่มอัตราการเจ็บป่วยและอัตราการตายที่เกิดจากโรคหัวใจและหลอดเลือด จึงเป็นที่มาของการศึกษานี้เพื่อดูความสัมพันธ์ของค่าดัชนีความยืดหยุ่นของหลอดเลือดแดง (CAVI) กับภาวะทางเดินหายใจอุดกั้นขณะนอนหลับ*

วัสดุและวิธีการ: *ผู้ป่วยที่เข้ามาตรวจการนอนหลับทั้งหมด 89 ราย โดยแบ่งเป็นคนที่มี AHI ≥5 จัดเป็นกลุ่มที่มีภาวะทางเดินหายใจอุดกั้น (OSA-group) (ผู้หญิง 27 ราย อายุเฉลี่ย 51.5±14.1 ปี) และคนที่มี AHI <5 จัดเป็นกลุ่มที่ไม่มีภาวะทางเดินหายใจอุดกั้น (controls-group) (ผู้หญิง 5 ราย อายุเฉลี่ย 56.8±11.8 ปี) โดยทุกรายไปรับการตรวจวัดความยืดหยุ่นของหลอดเลือด (cardio-ankle vascular index, CAVI) และความดันโลหิตขณะนั้น แล้วนำมาเปรียบเทียบกันระหว่างสองกลุ่ม*

ผลการศึกษา: *ข้อมูลด้านประชากรศาสตร์ระหว่างสองกลุ่มนั้นไม่แตกต่างกันอย่างมีนัยสำคัญ กลุ่มที่มีภาวะทางเดินหายใจอุดกั้นขณะหลับ (OSA-group) มีค่าเฉลี่ยของค่าความต่างระหว่างความดันโลหิตตัวบนและความดันโลหิตตัวล่างของหลอดเลือดแดงที่ข้อเท้า (mean pulse pressure of ankle artery) สูงกว่ากลุ่มที่ไม่มีภาวะทางเดินหายใจอุดกั้นขณะหลับอย่างมีนัยสำคัญ (7.31±14.6 มิลลิเมตรปรอท เทียบกับ 59.6±6.1 มิลลิเมตรปรอท, p = 0.008) ส่วนค่าความยืดหยุ่นของหลอดเลือด (CAVI) ของทั้งสองกลุ่มนั้นไม่มีความแตกต่างกันอย่างมีนัยสำคัญ (7.47±1.68 เทียบกับ 7.25±1.61, p = NS)*

สรุป: *ในกลุ่มที่มีภาวะทางเดินหายใจอุดกั้นขณะหลับไม่พบความสัมพันธ์กับความยืดหยุ่นของหลอดเลือด (CAVI) อย่างไรก็ตามพบความสัมพันธ์กับค่า ankle artery pulse pressure*