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Relation of Obstructive Sleep Apnea to Coronary Artery Calcium in Non-obese versus Obese Men and Women Aged 45 – 75 Years

Faith S. Luyster, PhD[1], **Kevin E. Kip, PhD**[2], **Aryan N. Aiyer, MD**[3], **Steven E. Reis, MD**[4], and **Patrick J. Strollo Jr., MD**[5]

[1]School of Nursing, University of Pittsburgh, Pittsburgh, PA

[2]College of Nursing, University of South Florida, Tampa, FL

[3]Division of Cardiology, University of Pittsburgh Medical Center, Pittsburgh, PA

[4]Department of Medicine, University of Pittsburgh, Pittsburgh, PA

[5]Division of Pulmonary, Allergy, and Critical Care, University of Pittsburgh, PA

Abstract

Sleep apnea and obesity are strongly associated and both increase the risk for coronary artery disease. Several cross-sectional studies have reported discrepant results regarding the role that obesity plays in the relation between sleep apnea and coronary artery calcium (CAC), a marker of subclinical coronary disease. The present study investigated the association between sleep apnea and presence of CAC in a community cohort of middle-aged men and women without preexisting cardiovascular disease, stratified by body mass index (BMI) (BMI < 30 versus ≥ BMI 30). Participants underwent electron beam computed tomography to measure CAC and underwent home sleep testing for sleep apnea. The presence of CAC was defined as an Agatston score > 0. Sleep apnea was analyzed categorically using apnea hypopnea index (AHI). The sample was comprised of primarily males (61%) and Caucasians (56%), with a mean age of 61 years. The prevalence of CAC was 76%. Among participants with a BMI < 30 (n = 139), AHI ≥ 15 (compared to AHI < 5) was associated with a 2.7-fold odds of having CAC, but the effect only approached significance. Conversely, in participants with a BMI ≥ 30, sleep apnea was not independently associated CAC. In conclusion, sleep apnea is independently associated with early atherosclerotic plaque burden in non-obese individuals.

Keywords

sleep apnea; coronary artery calcium; obesity

Corresponding Author: Faith S. Luyster, PhD, **Office:** 412-624-7910, **Fax:** 412-383- 7293, luysterfs@upmc.edu, **Address:** University of Pittsburgh School of Nursing, 3500 Victoria Street, Room 415, Pittsburgh, PA 15261.

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The present study investigated the association of sleep apnea and subclinical coronary atherosclerosis as defined by coronary artery calcium (CAC) scoring in a community cohort of middle-aged men and women without preexisting cardiovascular disease. We anticipate that the association between sleep apnea and CAC will be masked or minimized by the presence of obesity based on the known effects of obesity on coronary atherosclerotic burden[1,2]. Data were stratified by body mass index (BMI) (BMI < 30 versus BMI ≥ 30). We hypothesized that the effect of sleep apnea on CAC presence would be stronger among non-obese relative to obese participants.

METHODS

Participants were recruited from a prospective, community-based cohort study, Heart Strategies Concentrating On Risk Evaluation (Heart SCORE), designed to investigate racial disparities in cardiovascular risk in 2000 participants[3]. Heart SCORE eligibility criteria included age 45–75 years, residence in the greater Pittsburgh metropolitan area, ability to undergo baseline and annual follow-up visits, and absence of a known comorbidity expected to limit life expectancy to < 5 years. Participants were classified into 1 of 3 groups: preexisting cardiovascular disease (prior myocardial infarction, coronary revascularization, or stroke); moderate/high (>10%) probability of cardiovascular disease event in next 10 years; or low probability of cardiovascular disease events, based on Framingham risk score profiles[4]. For the present analysis, we examined baseline data and excluded participants with preexisting cardiovascular disease and/or missing data on cardiovascular risk factors. Electron beam computed tomography (EBCT) scans and home sleep testing for sleep apnea were not performed on all participants, thus CAC and sleep apnea data were available for a total of 324 participants. We included participants who underwent EBCT and home sleep testing within 24 months of each other (n = 276) and excluded participants who had a change in BMI ± 2 within the period between EBCT and home sleep testing assessments (n = 24), in order to account for potentially altered CAC profiles. The final sample for the present analysis included 252 participants. Participants included in the present analysis had a greater proportion of males and those with diabetes and/or hypertension as compared to the Heart SCORE population. The Heart SCORE protocol was approved by the institutional review board at the University of Pittsburgh Medical Center, and all participants provided written informed consent.

During the baseline visit, detailed demographic and medical histories were collected. Height and weight were measured to calculate BMI. The medical history inquired about a history of previously diagnosed hypertension, hyperlipidemia, and diabetes mellitus, as well as current medications. Resting blood pressure measurement was based on the average of 2 seated blood pressures by trained nurses. Laboratory assessment of lipoprotein levels were performed on venous blood draw in the fasting state. Age, gender, race/ethnicity, and smoking status (current/past smoker: yes/no) were self-reported.

Dyslipidemia was defined as having blood lipid concentrations within the following parameters, HDL cholesterol ≤ 40 mg/dL, total cholesterol ≥ 200 mg/dL, or self-reported treatment for dyslipidemia. Hypertension was defined as diastolic blood pressure (DBP) ≥ 90 mm Hg and/or systolic blood pressure (SBP) ≥ 140 mm Hg, or self-reported usage of

antihypertensive medication. Diabetes was defined as a self-reported diagnosis, current use of anti-diabetic medication, or fasting glucose ≥ 110 mg/dL.

Sleep apnea was assessed with a previously validated portable monitor that measures airflow and snoring via a nasal pressure signal (ApneaLink, ResMed Corp)[5]. An apnea was defined as a decrease in airflow of ≥ 80% from baseline for ≥ 10 seconds. A hypopnea was defined as a decrease in airflow between > 30% and < 80% from baseline for ≥ 10 seconds. Sleep apnea was analyzed categorically using the apnea-hypopnea index (AHI): 0–4, 5–14, and ≥ 15 events/hour.

Electron beam computed tomography image acquisition was obtained with an Imatron C150 scanner (GE Imatron Inc, South San Francisco, CA). To evaluate the coronary arteries, 30–40 contiguous 3-mm thick transverse images were obtained from the level of the aortic root to the apex of the heart during maximal breath holding. Images were acquired by using electrocardiogram triggering (80% of the RR interval) of 100 millisecond exposure during the same phase of the cardiac cycle. Calcium scores were calculated by the Agatston method, based on the detection of ≥ 3 contiguous pixels > 130 Hounsfield units[6]. Presence of CAC was defined as an Agatston score > 0.

Statistical Analysis

Differences in demographic characteristics were compared between the 3 sleep apnea groups (AHI < 5, AHI 5–14, AHI ≥ 15) using χ^2 test for categorical variables and analysis of variance with *post hoc* comparison for continuous variable. The non-parametric Kruskall-Wallis test was used to compare the median value of CAC scores among the sleep apnea groups. Frequencies of the presence of CAC were calculated among the 3 groups in the overall population and within non-obese and obese (BMI < 30 versus BMI ≥ 30). Multivariate logistic regression controlling for age, gender, race/ethnicity (Caucasian and other versus African American), smoking status, diabetes, hypertension, dyslipidemia, and BMI was used to calculate the odds ratio of the presence of CAC among individuals with no sleep apnea (AHI <5: reference group), mild sleep apnea (AHI 5–14), and moderate to severe sleep apnea (AHI ≥ 15). SPSS statistical software (SPSS Inc, Chicago, IL) for Windows 20 was used for all statistical analyses.

RESULTS

Demographic and clinical characteristics of the sample are presented in Table 1. The prevalence of CAC in the total sample was 75%. The frequency of CAC presence increased with severity of sleep apnea ($\chi^2 = 6.54$, $p < .05$) (Figure 1). In age, gender, race/ethnicity, smoking status, diabetes, hypertension, and dyslipidemia adjusted analysis, the odds ratio (95% confidence interval) for the presence of CAC was 2.33 (1.01 – 5.38) for participants with AHI ≥ 15 compared to those with AHI < 5 ($p < .05$) (Table 2). The association was no longer significant after adjusting for BMI. No significant difference in presence of CAC was found between those with AHI 5–14 and AHI < 5.

The % of participants with CAC increased with sleep apnea severity among those with a BMI < 30 ($\chi^2 = 6.77$, $p < .05$) (Figure 1). Specifically, among those with a BMI < 30, 53%

of participants with AHI < 5, 66% with AHI 5–14, and 79% with AHI ≥ 30 had evidence of CAC. Among those with a BMI ≥ 30, there was no significant difference in % of participants with CAC among the 3 AHI groups (< 5, 5–14, ≥ 30) (Figure 1). There were similar trends among males and females (data not shown). Consistent results were observed in logistic regression analyses adjusted for age, gender, race/ethnicity, smoking status, diabetes, hypertension, and dyslipidemia. Among 113 participants with a BMI ≥ 30, AHI was not independently associated with CAC (Table 2). Conversely, among 139 participants with a BMI < 30, AHI ≥ 15 (compared to AHI < 5) was associated with a 2.7-fold odds of having CAC (adjusted odds ratio = 2.70, 95% confidence interval: 0.88–8.33, p =.08), but the effect only approached conventional level of statistical significance. The formal test of interaction between BMI (dichotomous) × AHI ≥ 15 in relation to CAC presence was not statistically significant ($p = 0.13$).

Discussion

In this community cohort of middle aged adults without preexisting cardiovascular disease, we found an AHI ≥ 15 to be associated with a 2.3-fold odds of having CAC compared to an AHI < 5; however adjustment for cardiovascular risk factors and BMI rendered the association to non-significant levels. No difference in CAC presence was found between AHI 5–14 and AHI < 5. In analyses stratified by BMI, no significant association between AHI severity and presence of CAC was found among obese (BMI ≥ 30) individuals. Conversely, among non-obese adults, those with an AHI ≥ 15 had a 2.7-fold odds of having CAC compared to no sleep apnea after adjustment for cardiovascular risk factors and BMI, but the effect only approached statistical significance. Our sample was primarily comprised of participants with mild sleep apnea, thus a sample enriched with more severe sleep apnea may have revealed a stronger effect. Nevertheless, these findings suggest that sleep apnea, in particular moderate to severe sleep apnea (AHI ≥ 15), is independently associated with subclinical coronary atherosclerosis among non-obese individuals.

Sleep apnea and obesity often coexist[7,8] and each are strongly associated with cardiovascular disease including the development of coronary artery disease[9,10]. Obesity has been identified as an independent risk factor for atherosclerosis[1,11,12]. A randomized trial evaluating the effects of 4 months of continuous positive airway pressure (CPAP) therapy on early markers of atherosclerosis found a decrease in intima-media thickness which was associated with reductions in C-reactive protein and catecholamines suggesting that sleep apnea is an independent risk factor for atherosclerosis[13]. Given that both obesity and sleep apnea have similar cardiovascular consequences, it is important to explore whether the effect of sleep apnea on atherosclerotic burden may be masked or minimized by obesity. Results from previous community-based studies examining the association between sleep apnea and subclinical coronary atherosclerosis, as measured by CAC, are similar to our findings showing no association between AHI severity and CAC after adjustment for BMI[14,15]. In a population-based cohort of 258 Korean men and a community sample of 224 middle-aged men and women, higher AHI was associated with having any measureable CAC; however adjustment for BMI rendered the associations to non-significant levels[14,15]. Conversely, in patients with suspected sleep disorders, more severe sleep apnea was associated with CAC after controlling for BMI[16,17]. We extended prior investigations by stratifying our sample by

BMI (BMI < 30 vs BMI ≥ 30). Moderate to severe sleep apnea was independently associated with greater odds of having CAC but only among non-obese participants suggesting that obesity is a confounder of the association between sleep apnea and subclinical coronary atherosclerosis.

Pathogenic mechanisms linking sleep apnea to atherosclerosis have been proposed[18]. Inflammation is one possible mechanism underlying the pro-atherogenic effects of sleep apnea. More specifically, C-reactive protein, a serum maker of systemic inflammation, plays a direct role in the manifestation of atherosclerosis[19,20] and is elevated in those with sleep apnea independent of BMI[21] and visceral obesity[22]. Other possible mechanisms associated with sleep apnea that may contribute to the progression of atherosclerosis include increased oxidative stress[23], endothelial dysfunction[24,25], and sustained sympathetic nerve activity due to decreased baroreflex-mediated suppression of chemoreceptor-mediated sympathoexcitation[26]. Sleep apnea could also contribute to atherosclerosis indirectly, by causing insulin resistance due to elevated leptin levels[27] and dyslipidemia[28].

Study limitations include the cross-sectional study design, which precludes causal relationships between sleep apnea and subclinical coronary atherosclerosis and slight differences in gender distribution and rates of diabetes and hypertension between the current study population and the Heart SCORE cohort. AHI was assessed using a single-channel portable monitor (ApneaLink, ResMed Corp) instead of polysomnography. However, the portable monitor that we used has been previously validated with acceptable performance to identify obstructive sleep apnea[5,29,30]. There was a low prevalence of severe sleep apnea (AHI ≥ 30: n = 25) in our sample.

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- Greater sleep apnea severity is associated with coronary artery calcification.
- This association may exist primarily among non-obese individuals.
- Treatment of sleep apnea may be important for attenuating atherosclerotic progression.

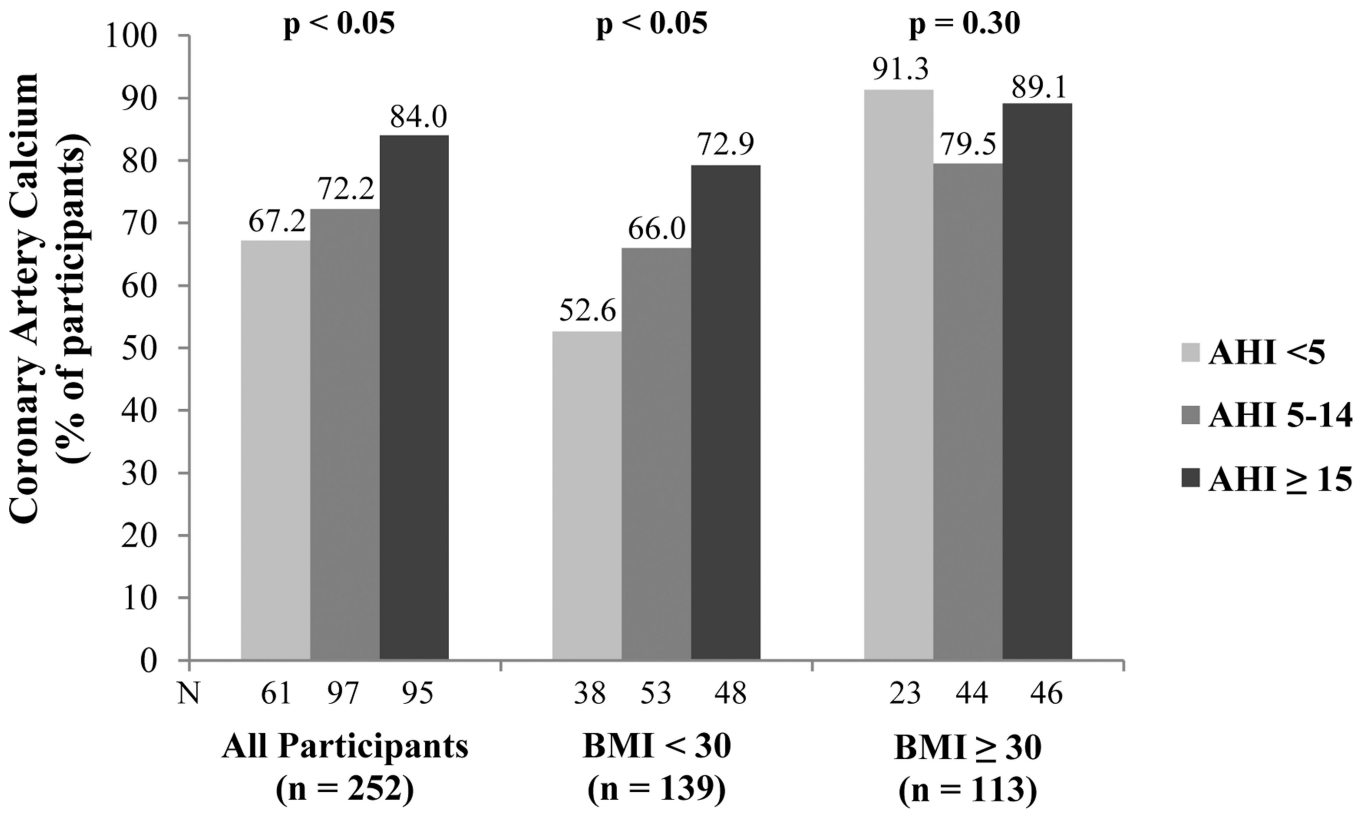

Figure 1.

Percentage of participants with coronary artery calcium by apnea hypopnea index in the overall sample and stratified by body mass index. AHI, apnea hypopnea index; BMI, body mass index.

Table 1

Demographic and clinical characteristics by apnea hypopnea index and in overall sample

Variable	Apnea Hypopnea Index			Total sample (N = 252)	p-value[a]
	< 5 (n = 61)	5–14 (n = 97)	≥ 15 (n = 94)		
Age, mean (SD) (years)	59.1 ± 7.8	60.9 ± 7.6	62.0 ± 6.5	60.9 ± 7.3	0.05
Male	29 (48%)	48 (49%)	64 (68%)	141 (56%)	0.008
Female	32 (52%)	49 (51%)	30 (32%)	111 (44%)	
European American	29 (52%)	66 (68%)	59 (63%)	156 (62%)	0.84
African American	32 (48%)	31 (32%)	35 (37%)	96 (38%)	
BMI, mean (SD) (kg/m^2)	29.0 ± 5.8	29.9 ± 4.5	30.4 ± 5.0	29.9 ± 5.0	0.26
AHI, mean (SD)	2.7 ± 1.1	9.0 ± 2.7	26.9 ± 12.3	14.1 ± 12.7	
Current/past smoker	33 (54%)	60 (62%)	46 (49%)	137 (55%)	0.19
Diabetes Mellitus	10 (16%)	22 (23%)	19 (20%)	50 (20%)	0.63
Prehypertension	13 (21%)	19 (20%)	18 (19%)	50 (20%)	0.94
Hypertension[c]	43 (71%)	65 (67%)	71 (75%)	179 (71%)	0.53
Dyslipidemia[d]	45 (77%)	81 (83%)	65 (69%)	192 (76%)	0.10
CAC score, median (range)	18.3 (0 – 786)	18.1 (0 – 1359)	64.2 (0 – 1115)	38.5 (0 – 1359)	0.01[b]

[a] P values based on Pearson χ^2 test and analysis of variance.

[b] P value based on Kruskall-Wallis test. BMI, body mass index; AHI, apnea hypopnea-index; CAC, coronary artery calcium.

[c] Hypertension was defined as diastolic blood pressure ≥ 90 mm Hg and/or systolic blood pressure ≥ 140 mm Hg, or self-reported usage of antihypertensive medication.

[d] Dyslipidemia was defined as having blood lipid concentrations within the following parameters, HDL cholesterol ≤ 40 mg/dL, total cholesterol ≥ 200 mg/dL, or self-reported treatment for dyslipidemia.

Table 2

Logistic regression analysis of adjusted odds ratios of coronary artery calcium in relation to apnea hypopnea index in the total sample and stratified by body mass index

AHI Score (compared to < 5)		**OR**	**95% CI**	**p-value**
		Adjusted for age, gender, race/ethnicity, smoking status, diabetes, hypertension, and dyslipidemia		
Total Sample (n = 252)	5–14	0.97	(0.46 – 2.06)	0.94
	≥ 15	2.33	(1.01 – 5.38)	0.04
AHI Score (compared to < 5)		**OR**	**95% CI**	**p-value**
		Adjusted for above covariates + BMI		
Total Sample (n = 252)	5–14	0.72	(0.33 – 1.61)	0.43
	≥ 15	1.71	(0.71 – 4.13)	0.23
AHI Score (compared to <5)		**OR**[a]	**95% CI**	**p-value**
BMI < 30 (n = 139)	5–14	1.12	(0.40 – 3.14)	0.83
	≥ 15	2.70	(0.88 – 8.33)	0.08
AHI Score (compared to <5)		**OR**[a]	**95% CI**	**p-value**
BMI ≥ 30 (n = 113)	5–14	0.29	(0.05 – 1.62)	0.16
	≥ 15	0.73	(0.12 – 4.48)	0.74

[a]Adjusted for age, gender, race/ethnicity, smoking status, diabetes, hypertension, and dyslipidemia. AHI, apnea-hypopnea index; OR, odds ratio; CI, confidence interval; BMI, body mass index.