Prolonged sitting time links to subclinical atherosclerosis

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Abstract

Background: This study investigates the association between daily sitting time and subclinical atherosclerosis by using coronary computed tomography angiography (CCTA).

Methods: The study enrolled 203 subjects (age 57.6 ± 8.8 years) who underwent CCTA at annual medical checkups. Sitting time was categorized as < 5 hours/day (short), 5 to 9 hours/day (moderate) and ≥10 hours/d (long). We analyzed the coronary calcium score, plaque characteristics, and severity of coronary artery stenosis, including the segment involvement score (SIS) and segment stenosis score (SSS).

Results: Subjects with longer sitting times tended to be male gender and have lower levels of high-density lipoprotein cholesterol (p for trend < 0.05). In addition, those with longer sitting time had higher SIS (1.2 ± 1.5 *vs.* 1.6 ± 2.1 *vs.* 2.3 ± 2.0 for short, moderate, and long sitting time, respectively) (p for trend = 0.015) and SSS (1.4 ± 2.0 *vs.* 1.9 ± 2.7 *vs.* 2.7 ± 2.6) (p for trend = 0.015), suggesting longer sitting time-correlated with the severity of coronary atherosclerosis. When considering the coronary plaque patterns, subjects with shorter sitting time (<5 hours/d) tended to have more calcified plaque and subjects with longer sitting time (≥10 hours/d) had more mixed plaque (p for trend = 0.018). After adjusting for age, gender, comorbidities, body mass index, and lipid profiles, increased sitting time was independently associated with the presence of mixed plaque, suggesting longer sitting time may be associated with higher risk of the formation of vulnerable plaque.

Conclusion: Longer sitting time was linked to the severity of subclinical atherosclerosis and the presence of high-risk vulnerable plaque in the general population.

Keywords: Atherosclerosis; Coronary computed tomography angiography; Coronary plaque; Physical activity; Sitting time

1. INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death around the world.[1] Although the treatment for CVD has greatly improved in recent years, mortality due to ischemic heart disease remains high globally.[2] The impact of CVDs and cerebrovascular diseases leads to excess death and massive additional financial burdens for health systems,[3] so it is important to screen and identify individuals at high risk as part of a strategy for primary and secondary prevention. Early identification of patients at risk and risk factor modification is crucial for CVD prevention and treatment.

Sedentary behavior is associated with multiple cardio-metabolic abnormalities, such as obesity, hypertension, diabetes, mortality, and even certain malignancies.[4–7] Therefore, a healthy lifestyle with regular exercise is suggested for the promotion of public health. Although sedentary behavior is considered as a risk factor for atherosclerosis, the association of physical activity and indices of coronary atherosclerosis, such as coronary artery calcium (CAC), has not been consistent. Various types of relationships between physical activity and CAC have been reported, including an inverse relationship, positive relationship, U/J-shaped relationship, and even no relationship.[8]

Possible reasons for the inconclusive association observed between physical activity and CAC included the variety of methods used to measure physical activity and inconsistent quality of questionnaires. In addition, a recent study reported the CAC index as the product of the CAC volume and CAC density. CAC volume correlated with future risk, but CAC density was inversely correlated with risk,[9] indicating that only CAC still has limitations in determining future cardiovascular risk.

Recent studies showed that high-volume, high-intensity exercise may increase CAC, but the coronary plaque patterns tend to be more calcified with fewer instance of mixed-type plaque.[10,11] This indicates that plaque patterns may provide additional risk stratification in characterizing the prognosis of coronary plaque.

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Mixed plaques contain both calcified and non-calcified material and are associated with the worst prognosis, whereas calcified plaques are associated with the best event-free survival.[8,12]

Sitting time is a parameter that is used for the evaluation of physical activity and is associated with total mortality, but the evidence is less clear for coronary artery disease.[13] Prolonged sitting time (>9 hours/d) has been shown to be associated with the incidence of nonalcoholic fatty liver disease,[14] and is a risk factor for obesity, suggesting that prolonged sitting time might be a risk factor for atherosclerosis. The association between daily sitting time and coronary atherosclerosis has remained undetermined and limited information has been reported about the association between atherosclerotic plaque patterns and sitting duration. Therefore, the aim of this study is to investigate the association between sitting time and CAC and the characteristics of coronary artery plaque as signals for subclinical atherosclerosis in the general population.

2. METHODS

2.1. Study patients

This study employed a cross-sectional observational design and was conducted between February 2015 and June 2016. Participants were invited to participate and screened if they underwent coronary computed tomography angiography (CCTA) using 256-slice multidetector computed tomography as part of a general health evaluation at Taipei Veterans General Hospital. Participants had no history of coronary artery disease and no angina symptoms. They were invited to answer the questionnaires dealing with information about daily amounts of activity, sedentary behavior, food frequency, and smoking habits.

The study protocol and questionnaire information have been reported previously in our *VGH-HEALTHCARE* studies.[15,16] The study complied with the Declaration of Helsinki, and the study protocol was approved by the Institutional Research Board of Taipei Veterans General Hospital. All study patients gave informed consent before entering the study.

2.2. Sitting time and physical activity

Sitting time and physical activity levels were assessed using the validated Chinese version of the International Physical Activity Questionnaire Short Form (IPAQ-SF).[17,18] The IPAQ-SF measures the number of days and the duration of vigorous, moderate, and walking activities during the previous week.[19,20] These data were quantified, and the estimated metabolic equivalent of task (MET) for each individual was classified as high, moderate, or low physical activity according to the IPAQ-SF score.[19,20] Total weekday sitting time was measured by a single question that has shown acceptable validity and reliability: "During the last 7 days, how much time did you usually spend sitting on a weekday?" Sitting times were categorized into groups using cutoff points that have been commonly used in other studies, <5, 5–9, and ≥10 hours/d.[14,21]

2.3. CCTA measurement

CCTA was performed using multiple-detectors computed tomography (Definition Flash; Siemens Healthineers, Erlangen, Germany). Blood pressure and heart rate were measured beforehand. Beta-blockers or calcium channel blockers were given to patients who had an initial heart rate greater than 80 beats per minute when there were no contraindications. A prospective electrocardiography gated axial scan for calcium scoring was triggered at 75% of the R-R interval with the collimation set at 3.0 mm. The scanning sequence began approximately 1 cm above the left main coronary artery. CCTA parameters were set at 120 kV and 60 mA. A temporal resolution of 230 ms was achieved by using the half-scan reconstruction method with a 350-ms gantry rotation time.

The CCTA was performed by retrospective gated helical scanning with the parameters set at 64 × 0.5 mm × 128 × 0.625 mm collimation, 270-350 ms gantry rotation time, and 80-135 kV according to body size. The bolus-tracking method was used after injecting a total of 50-100 mL of iodinated contrast medium (Iopamiro 370; Bracco Imaging SpA, Milan, Italy; Ultravist 370; Bayer Pharma AG, Berlin, Germany) according to body size at a rate of 4.5-5.0 mL per second, followed by 50 mL of normal saline at a rate of 5.5 mL per second. The workstation automatically selected the best phase, and if the image quality was suboptimal, we manually reconstructed the phase with the best possible image quality, which was reconstructed into images with slice thicknesses of 0.75 mm and 0.9 mm with a 0.45 mm interval. All images were transferred to an external workstation (EBW, Amsterdam, Netherlands) for analysis.

Plaque morphology and the degree of coronary luminal stenosis were assessed according to established guidelines.[22,23] The severity of coronary atherosclerosis was scored as reported previously. The CAC was calculated by the Agatston method and graded as 0, 1 to 99, 100-399, and ≥400.[24] Several CCTA scores were measured including CAC, the segment involvement score (SIS), and the segment stenosis score (SSS).[25,26] The SIS was calculated as the total number of coronary artery segments that exhibited plaques, regardless of the degree of luminal stenosis within each segment (minimum = 0; maximum = 16). The SSS was used to measure the overall extent of coronary artery plaques.

Each individual coronary segment was graded as having no plaque to severe plaques (ie, scores from 0 to 3) based on the extent of obstruction of the luminal diameter of the coronary artery. The extent scores of all 16 individual segments were summed to yield a total score, which has a range from 0 to 48.[26,27] To investigate the association between daily sitting time and coronary plaque characteristics, we also analyzed the association between sitting time groups and the presence of significant (>50% stenosis) stenotic coronary plaque patterns including calcified, noncalcified and mixed-type plaques as in previous studies[11,28] (Fig. 1).

2.4. Laboratory measurements

Venous blood samples were collected after an overnight fast. Biochemical serum markers were measured with a TBA-c16000 automatic analyzer (Toshiba Medical Systems, Tochigi, Japan). Other biochemical variables were also measured, including albumin, cholesterol, triglyceride, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), blood urea nitrogen, creatinine, aspartate aminotransferase, and alanine aminotransferase (ALT), uric acid, and fasting blood glucose. These parameters were measured using a previously described method.[29]

2.5. Statistical analysis

All the data were expressed as a frequency (percentage) or the mean ± SD. All the participants were divided into three groups according to hours spent on sitting daily (<5, 5–9, and ≥10 hours/d). Continuous parametric data between the participants in the three sitting time groups were compared using a one-way analysis of variance. Categorical data were compared between the sitting time groups using a Chi-square test or Fisher's exact test. Plaque with >50% stenosis was identified as significant plaque according to a previous study investigating the risk ratio of variable plaque patterns in coronary artery.[28,30]

A multivariate logistic regression analysis was performed to determine the risk of the presence of significant plaques for

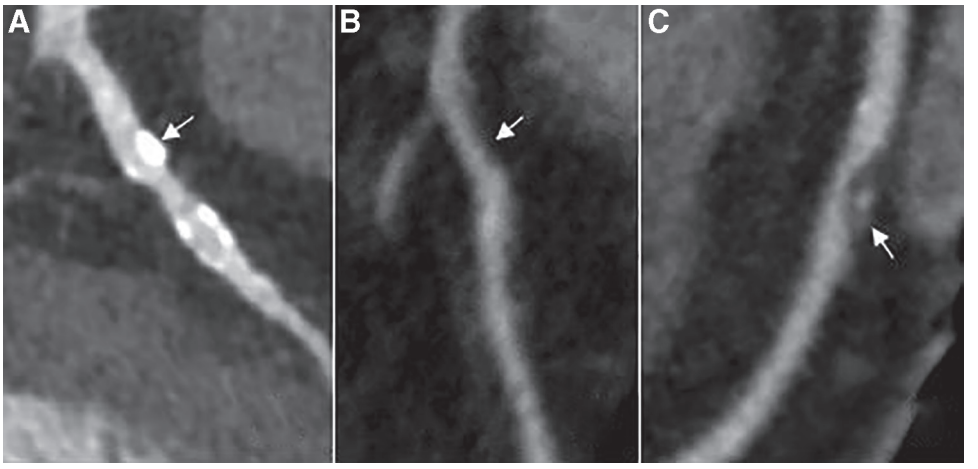

Fig. 1 Longitudinal views (arrows) of significant calcified plaques (A), non-calcified (soft) plaque (B), and mixed plaque (C) in the coronary artery.

every 1-hour increase in sitting time per day. In model 1, the crude odds ratio (OR) was determined. In model 2, the OR was adjusted for age, gender, history of hypertension, diabetes, and smoking. In model 3, the OR was adjusted for age, gender, history of hypertension, diabetes mellitus, smoking, physical activity, BMI, HDL-C, and LDL-C. The *p*-value was two-sided, and $p < 0.05$ was considered statistically significant. Statistical analysis was performed utilizing SPSS software (Version 15.0; SPSS Inc).

3. RESULTS

The study enrolled a total of 203 subjects (age 57.6 ± 8.8 years, 149 male and 54 female) who received CCTA and completed the IPAQ-SF. These included 66 subjects with sitting time <5 hours/d (short), 108 with sitting time of 5 to 9 hours/d (moderate), and 29 subjects with sitting time ≥10 hours/d (long). Table 1 shows the demographic data of the study subjects in three sitting time groups. Subjects in the long sitting time group were predominantly male (63.6%, 75.0%, and 89.7% for short, moderate, and long sitting time, respectively) and the prevalence was significantly higher with increasing time spent sitting (*p* for trend = 0.007).

In addition, HDL-C was found progressively lower with increasing sitting time (49.9 ± 14.9 mg/dl *vs.* 45.7 ± 13.0 mg/dl *vs.* 42.8 ± 9.9 mg/dl, respectively) (*p* for trend = 0.017) and creatinine was found progressively higher with increasing sitting time (*p* for trend = 0.040). Although subjects with longer sitting times had lower cholesterol values, there was no statistical difference in LDL-C between sitting time groups. Other demographic data were similar between the three groups, including age, BMI, the prevalence of diabetes mellitus, blood pressure, and other biochemical data.

3.1. CCTA data of the patients by sitting time groups

Although not statistically significance, subjects with longer sitting time tended to have higher calcium scores (41.0 ± 95.1 *vs.* 52.8 ± 114.0 *vs.* 80.5 ± 118.2, respectively), especially in the left anterior descending coronary artery (24.8 ± 63.9 *vs.* 29.7 ± 71.5 *vs.* 59.7 ± 101.5 respectively) (*p* for trend = 0.036) (Table 2). Regarding the severeity of coronary atherosclerosis, subjects with longer sitting time tended to have higher SIS (1.2 ± 1.5 *vs.* 1.6 ± 2.1 *vs.* 2.3 ± 2.0, respectively) (*p*=0.051, P for trend = 0.015) and SSS (1.4 ± 2.0 *vs.* 1.9 ± 2.7 *vs.* 2.7 ± 2.6) (*p* for trend = 0.015) (Table 2 and Fig. 3A), suggesting that longer sitting time correlates with coronary atherosclerosis severity. In addition, SIS was correlated to CCTA score (r = 0.786, $p < 0.001$), > 50% stenosis calcified plaque (r = 0.280, $p < 0.001$), and > 50% stenosis mixed plaque (r = 0.338, $p < 0.001$). SSS was correlated with the CCTA score (r = 0.805, $p < 0.001$), calcified plaque (r = 0.358, $p < 0.001$), non-calcified plaque (r = 0.150, $p = 0.032$), and mixed plaque (r = 0.377, $p < 0.001$) (Supplement Table 1, http://links.lww.com/JCMA/A117). There was no significant correlation between MET activity and SIS (r = 0.12, $p = 0.087$), SSS (r = 0.076, $p = 0.281$) or CCTA score (r = 0.089, $p = 0.205$).

Sitting time and the presence of significant coronary plaque

When considering significant plaque characteristics (> 50% stenosis) in three sitting groups, subjects with shorter sitting time (<5 hours/d) tended to have higher amounts of calcified plaque and subjects with longer sitting time (≥10 hours/d) tended to have higher amounts of mixed plaque (*p* for trend = 0.018) (Table 2 and Fig. 3B). Fig. 3C shows the sitting time distributions according to dominant plaque patterns. In the presence of various significant plaques, there was a significant association between longer sitting time (≥10 hours/d) and the presence of mixed-type plaque, suggesting a close relationship between long sitting time and the presence of high-risk plaque within the coronary artery.

To further explore the role of sitting time in the presence of various plaque patterns, a logistic regression was performed to determine the risk of the presence of different significant plaques according to increasing sitting time. The results were adjusted for variables including age, gender, hypertension, diabetes, smoking, BMI, HDL, LDL, and physical activity (MET) (model 3). As a result, increased sitting time was independently associated with a higher risk of the presence of significant mixed plaque in the coronary artery (OR, 1.33; 95% CI, 1.04-1.69, $p = 0.022$) (Table 3).

4. DISCUSSION

The main findings showed a significant association between increasing increased sitting time and unfavorable coronary atherosclerosis. Sitting time was associated with more mixed plaques but less calcified plaques, as well as the severity of coronary artery stenosis. Furthermore, there was a significant association between sitting time and the presence of high-risk plaque in the coronary arteries, independent of physical activity and

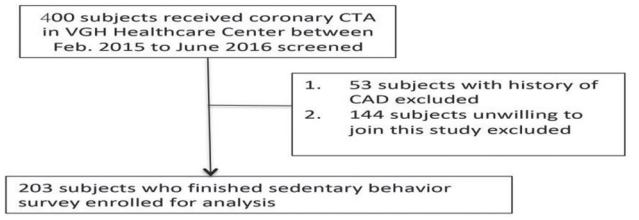

Fig. 2 Study flow.

comorbidities, This suggests that avoiding sedentary behavior and sitting for a long time is crucial for CVD prevention.

Low physical activity and sedentary behavior have been associated with the development of chronic conditions, including obesity, hypertension, diabetes, CVD, mortality, and even certain malignancies.[4–7] Although some of these conditions can be treated pharmacologically, more effort needs to be done to reduce their risk through lifestyle modifications. Many studies support that adequate physical activity reduces CV risk.[31,32] In a cohort of 8425 generally healthy men with an average of 8.4 years of follow-up, higher levels of cardiorespiratory fitness at baseline were shown to be associated with fewer CV events.[33]

According to another cohort of 10 690 asymptomatic patients who underwent CAC scanning, exercise may play a protective role, especially among those with high CAC.[34] Therefore, the 2016 European guidelines have recommended at least 150 minutes a week of moderate-intensity aerobic physical activity or 75 minutes a week of vigorous-intensity aerobic physical activity for healthy adults of all ages.[35] The American Heart Association also recommends routine assessment and promotion of physical activity in healthcare settings for primary prevention.[36]

In the current study, we observed a linear association between sitting time and the severity of coronary atherosclerosis, which was independent of physical activity. This suggests that prolonged sitting time is associated with a higher risk of atherosclerosis in this Asian population. Subjects with less sitting time had more calcified plaque and less mixed plaque. Those with longer sitting time had less calcified plaque and more mixed plaque, indicating that prolonged sitting time (≥10 hours/d) was associated with high-risk plaque formation independent of CV risk factors and physical activity. There are limited data on the association between sitting time and plaque characteristics, but several studies show evidence of morphology modification of coronary artery plaque after exercise. One study compared 108 healthy male marathon runners with sedentary men anf found more coronary artery calcification in the runners after adjustment for both age and Framingham risk score.[35] In addition, it has been reported that male athletes demonstrated predominantly calcified plaques (72.7%), whereas sedentary males showed predominantly mixed morphology plaques (61.5%).[10]

In a cohort of 284 middle-aged men engaged in competitive or recreational leisure sports, participants in the group with >2000 MET minutes per week had a higher prevalence of CAC and atherosclerotic plaques.[11] The most active group, however, had a more benign composition of plaques, with less mixed plaques and more often only calcified plaques.[11] Several features have been identified as characteristics of vulnerable plaque, and plaque with spotty calcium was found to be the most frequent high-risk plaque feature, followed by positive remodeling, low-Hounsfield-units plaque, and napkin ring sign.[30] Hou et al. estimated the 3-year-probability of major CV events as 6% for calcified plaque, 23% for non-calcified plaque, and 38% for mixed plaque in a cohort of people with suspected CAD, which supports that the risk is highest in the presence of mixed plaque.[12] Avoiding risky plaque composition (less calcified plaque, more mixed plaque) is crucial to prevent adverse CV events.[11]

We observed a positive relationship between sitting time and the formation of high-risk coronary plaque independent of physical activity and underlying lipid profiles. The reason why more sitting time was associated with higher mixed plaque pattern remains undetermined. However, some clinical observations show that prolonged sedentariness is an independent risk factor, even if physical activity is considered. Hamilton et al.[36] suggested that sitting too much may affect the cellular processes responsible for metabolic abnormalities differently than structured exercise. Longer sitting time was found to be correlated with risk of obesity, CVD, metabolic syndrome, and even cancer, which was independent of time spent on moderate to vigorous physical activity.[36,37] This suggests that longer sitting time may be seen as an important risk factor for CVD independent of physical activity.

One recent meta-analysis showed a dose-response association between sitting time, time watching television, and CV mortality in those who are 'inactive', who represent the lowest quartile of physical activity.[38] In addition, longer sitting may represent a specific lifestyle pattern such as long time spent watching television, snacking, obesity and other CV risk factors. Although the baseline LDL-C levels were similar in our sitting time groups, longer sitting still carries a trend of reduced HDL-C which is considered as a protective factor in maintaining health in the CV system.

Comorbidities were adjusted for, including age, gender, physical activity, BMI, and lipid profiles, but the results still showed that sitting time was independently associated with the presence of mixed plaque. This suggests that longer sitting time may be associated with higher risk of the formation of vulnerable plaque, even in subjects who have few CV risks and remain asymptomatic in daily life. A recent study also reported that longer sitting time is a risk factor for fatty liver after adjusting for the volume of physical exercise[14] and showed that overall, avoiding sedentariness is crucial for CVD prevention.

Prolonged sitting time may be associated with a low level of physical activity, which could be a risk factor for cardiovascular disease. However, our study did not find a significant correlation between MET and SIS or SSS. This result hints that the significant correlation between long sitting time and coronary atherosclerosis is independent of total physical activity. Recently, Pereira et al. randomized subjects into a "move group" that had increased daily activity and a "stand group" that stood during work, and the results demonstrated that the stand group had a greater reduction of cardiovascular risk profiles.[39] This study supported our current finding of prolonged sitting time being linked to the risk of cardiovascular disease. Furthermore, it also supported that sedentary behavior, especially prolonged sitting should be avoided to improve cardiovascular health. In a recent study from Spain conducted by Perez-Lasierra et al.,[40] long sitting time was associated with increased prevalence of subclinical atherosclerosis, which was independent of physical activity.

There were some limitations to the present study. First, our sample size is relatively small and further studies with larger sample sizes are still needed to validate our findings. Second, the sitting time and physical activity were not collected by the IPAQ, and the use of self-reported information could be subject to bias. Third, the time spent sitting may be associated with

Table 1

Baseline characteristics of study population (n = 203)

Sitting time	<5 h/d N = 66	5-9 h/d N = 108	≥10 h/d N = 29	*p*
Ave. sitting (h/d)	3.2 ± 0.8	6.6 ± 1.2	11.0 ± 1.5	< 0.001
Age (y)	56.9 ± 7.6	58.8 ± 9.2	55.2 ± 9.5	0.105
Male, n (%)	42 (63.6%)	81 (75%)	26 (89.7%)	0.026
BMI (kg/m^2)	24.6 ± 2.8	25.1 ± 3.1	25.6 ± 2.1	0.265
Smoking, n (%)	8 (12.1%)	22 (20.4) %	5 (17.2%)	0.376
Drinking, n (%)	26 (39.4%)	53 (49.1%)	13 (44.8%)	0.460
Hypertension, n (%)	14 (22.2%)	40 (38.5%)	10 (34.5%)	0.093
Diabetes, n (%)	5 (7.9%)	11 (10.6%)	4 (13.8%)	0.678
SBP (mmHg)	124.8 ± 18.1	123.6 ± 19.3	124.3 ± 19.3	0.916
DBP (mmHg)	78.2 ± 10.3	78.1 ± 10.4	81.9 ± 13.2	0.222
Cholesterol (mg/dL)	219.2 ± 40.7	204.3 ± 37.8	201.5 ± 35.6	0.029
Triglyceride (mg/dL)	135.9 ± 95.2	130.3 ± 65.9	143.9 ± 67.1	0.682
HDL-C (mg/dL)	49.9 ± 14.9	45.7 ± 13.0	42.8 ± 9.9	0.034
LDL-C (mg/dL)	139.4 ± 34.8	130.6 ± 34.7	128.8 ± 35.8	0.216
ALT (U/L)	29.8 ± 20.9	28.4 ± 18.8	27.9 ± 13.5	0.862
Creatinine (mg/dL)	0.9 ± 0.2	0.9 ± 0.2	1.0 ± 0.2	0.025
Uric acid (mg/dL)	6.6 ± 1.6	6.6 ± 1.4	6.9 ± 1.4	0.534
Glucose (mg/dL)	95.4 ± 18.8	96.0 ± 16.8	94.2 ± 14.7	0.881
MET (min/wk)	1,889.7 ± 1,143.4	1,779.5 ± 1,047.2	1,475.5 ± 958.1	0.221

Values data are n (%) or mean ± SD.

ALT = alanine aminotransferase; BMI = body mass index; BUN = blood urea nitrogen; DBP = diastolic blood pressure; HDL-C = high-density lipoprotein-cholesterol; LDL-C = low-density lipoprotein-cholesterol; MET = metabolic equivalent of task; SBP = systolic blood pressure.

other unhealthy activities, such as snacking or consuming alcohol, about which information is not included in IPAQ.

Although the backgrounds of various sitting time groups were similar, those with prolonged stilling time tended to have higher baseline blood pressure and more diabetes as well. Furthermore, those with prolonged sitting time had a lower HDL-cholesterol (p = 0.034) and took more statins. Although previous studies have reported that sedentary behavior may cause reduced estrogen metabolism, increased estrogen levels[41–43] and declined immune system,[44] the current study only measured parameters related to cardiometabolic risks such as lipid profiles, blood sugar, and liver function. Serum hormone levels were not checked in our study.

Another limitation is that all of the participants were enrolled from those who received a comprehensive annual checkup at a healthcare center, and all subjects were asymptomatic. Therefore, detailed history of medication was not available, and the possibility of selection bias cannot be excluded. Finally, larger studies on different racial populations are still needed to investigate whether our findings could be applied to other populations.

In conclusion, longer sitting time was linked to subclinical atherosclerosis severity and the presence of high-risk vulnerable plaque. In addition to the promotion of adequate physical activity, avoiding prolonged sitting time and sedentary lifestyle should be advised for CVD prevention.

Table 2

Coronary computed tomography angiography (CCTA) findings and calcium scores of the patients by sitting time (n = 203)

Sitting time	<5 h/d (n = 66)	5-9 h/d (n = 108)	≥10 h/d (n = 29)	*p*	*p* for trend
CCTA score, total	41.0 ± 95.1	52.8 ± 114.0	80.5 ± 118.2	0.267	0.104
LM calcium score	1.2 ± 8.9	2.5 ± 9.2	1.1 ± 4.6	0.511	0.950
LAD calcium score	24.8 ± 63.9	29.7 ± 71.5	59.7 ± 101.5	0.095	0.036
LCX calcium score	3.2 ± 10.5	6.4 ± 30.8	2.1 ± 4.4	0.555	0.833
RCA calcium score	11.8 ± 37.5	14.2 ± 42.6	17.6 ± 28.1	0.794	0.504
Coronary calcium score, n (%)					
0	34 (51.5%)	60 (55.6%)	9 (31%)	0.127	
0-99	24 (36.4%)	31 (28.7%)	12 (41.4%)		
≥100	8 (12.1%)	17 (15.7%)	8 (27.6%)		
SIS	1.2 ± 1.5	1.6 ± 2.1	2.3 ± 2.0	0.049	0.015
SSS	1.4 ± 2.0	1.9 ± 2.7	2.7 ± 2.6	0.051	0.015
Plaque characteristics[a]					
Calcified plaque, n (%)	6 (9.1%)	3 (2.8%)	1 (3.4%)	0.162	0.119
Non-calcified plaque, n (%)	2 (3%)	3 (2.8%)	1 (3.4%)	0.981	0.953
Mixed plaque, n (%)	1 (1.5%)	5 (4.6%)	4 (13.8%)	0.038	0.018

Values data are n (%) or mean ± SD.

CAD = coronary artery disease; CCTA = Coronary computed tomography angiography; LAD = left anterior descending artery; LCX = left circumflex artery; LM = left main artery; RCA = right coronary artery; SIS = segment involvement score; SSS = segment stenosis score.

[a]Significant plaque formation (>50% stenosis).

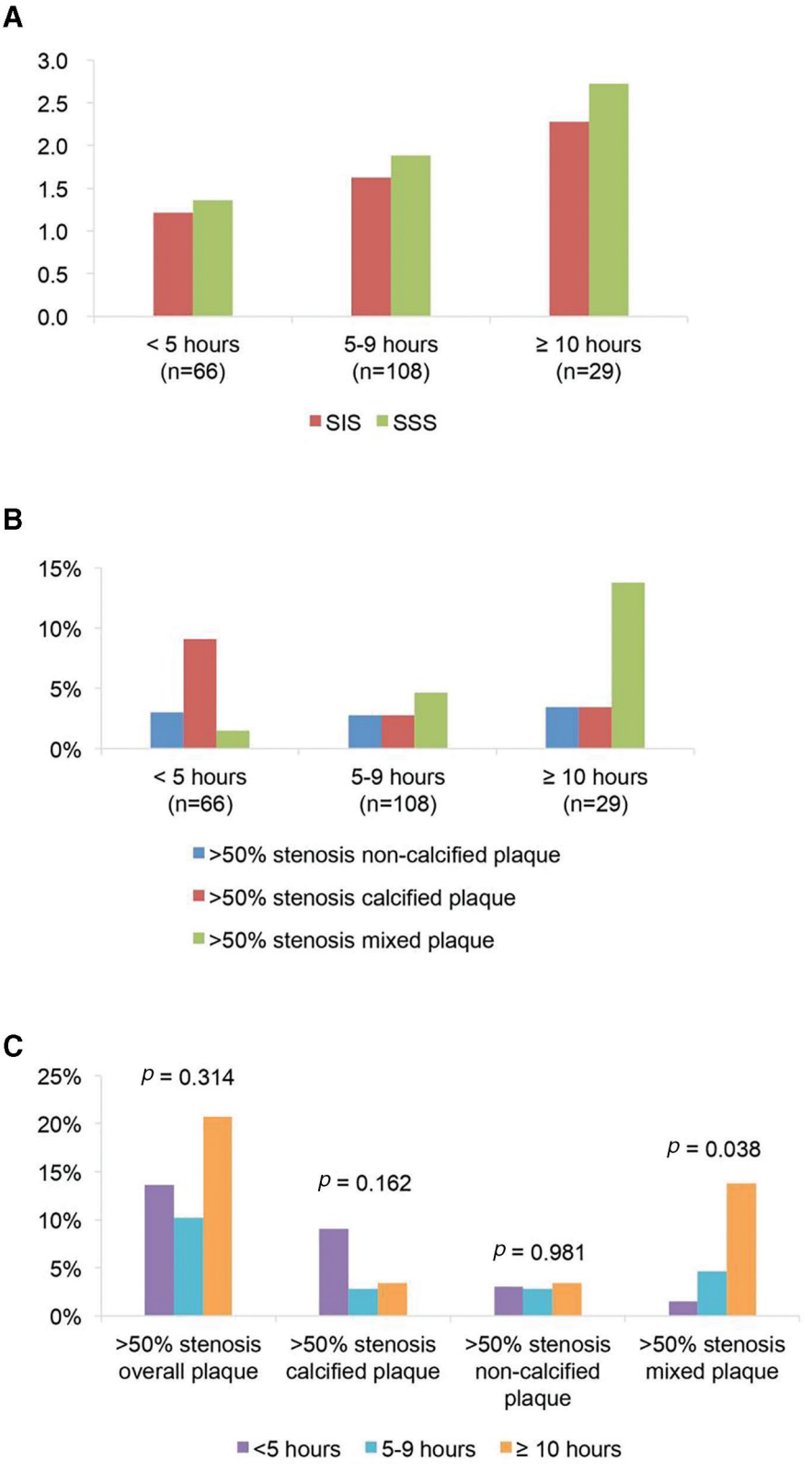

Fig. 3 (A) The association of segment involvement score (SIS) and segment stenosis score (SSS) in variable sitting time groups. (B) The association of significant (>50% stenosis) coronary plaque in variable sitting time groups. (C) The association between dominant plaque patterns and sitting hour categories.

Table 3

Multivariate adjusted association of daily sitting time with >50% stenotic coronary plaque

			Sitting time per day			
	Increasing 1 h sitting/day	*p*	<5 h/d	5-9 h/d	>10 h/d	*p* trend
Coronary plaque[a]						
Model 1	1.07 (0.92-1.23)	0.389	1 (referent)	0.72 (0.28-1.84)	1.65 (0.53-5.17)	0.580
Model 2	1.05 (0.90-1.23)	0.519	1 (referent)	0.56 (0.21-1.49)	1.48 (0.45-4.89)	0.793
Model 3	1.07 (0.91-1.26)	0.425	1 (referent)	0.60 (0.21-1.68)	1.52 (0.44-5.27)	0.701
Calcified plaque[a]						
Model 1	0.86 (0.66-1.12)	0.261	1 (referent)	0.29 (0.07-1.18)	0.36 (0.04-3.11)	0.126
Model 2	0.81 (0.59-1.10)	0.170	1 (referent)	0.16 (0.03-0.78)	0.30 (0.03-2.80)	0.062
Model 3	0.87 (0.63-1.21)	0.414	1 (referent)	0.20 (0.04-1.09)	0.41 (0.04-4.24)	0.160
Non-calcified (soft)[a]						
Model 1	0.93 (0.67-1.27)	0.635	1 (referent)	0.91 (0.15-5.62)	1.14 (0.10-13.13)	0.953
Model 2	0.91 (0.66-1.25)	0.550	1 (referent)	0.87 (0.14-5.44)	0.99 (0.08-11.82)	0.962
Model 3	0.89 (0.63-1.24)	0.476	1 (referent)	0.88 (0.13-5.91)	0.88 (0.07-11.32)	0.908
Partial calcified (mixed)[a]						
Model 1	1.34 (1.08-1.66)	**0.007**	1 (referent)	3.16 (0.36-27.62)	10.40 (1.11-97.63)	**0.023**
Model 2	1.35 (1.07-1.71)	**0.012**	1 (referent)	2.63 (0.29-23.56)	9.12 (0.93-89.46)	**0.036**
Model 3	1.33 (1.04-1.69)	**0.022**	1 (referent)	2.54 (0.28-23.13)	8.14 (0.82-81.28)	**0.048**

Model 1 was unadjusted.

Model 2 was adjusted for age, gender, hypertension, diabetes, and smoking.

Model 3 was adjusted for age, gender, hypertension, diabetes, smoking, BMI, HDL, LDL, and physical activity (MET).

[a]>50% stenosis.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://links.lww.com/JCMA/A117.

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