

# Coronary Artery Calcium and Arterial Stiffness Values that are Predictive of Long-Term Cardiovascular Events in People with Stable Coronary Artery Disease

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## Abstract

**Introduction:** Major adverse cardiovascular events (MACEs) have been linked to subclinical atherosclerosis, which can be identified by elevated coronary artery calcium (CAC) or arterial stiffness as measured by the cardio-ankle vascular index (CAVI). However, there aren't many comparable data from these two assessments on the same population.

**Methods:** From 2005 to 2013, both asymptomatic and symptomatic patients with stable coronary artery disease (CAD) who underwent coronary computed tomography and CAVI were enrolled and followed until December 2019 for the occurrence of MACEs (cardiovascular [CV] death, nonfatal myocardial infarction [MI], and nonfatal stroke). The relationships between the CAC score and CAVI and long-term MACEs were evaluated using a cause-specific hazard model.

**Results:** 8687 patients participated in all. In 49.7%, 31.9%, 12.3%, and 6.1% of them, the CAC scores were 0–99, 100–399, and 400, respectively. In 23.8%, 36.3%, 44.5%, and 56.2% of cases, arterial stiffness (CAVI 9.0) was linked to the severity of CAC. MACEs occurred in 8.0% of patients over an average of 9.9. 2.4 years of follow-up (95 percent CI: 7.4 to 8.6 percent) of subjects. CAC scores of 100–399 and CAVI scores of 9.0 were found to independently predict the occurrence of MACEs with hazard ratios (95% CI) of 1.70 (1.13–1.98), 1.87 (1.33–2.63), and 1.27 (1.06–1.52), respectively, after adjusting for covariables. Hypertension, diabetes mellitus (DM), chronic kidney disease (CKD), aspirin, and statin therapy were additional risk factors.

**Conclusion:** Both asymptomatic and symptomatic patients with stable CAD are more likely to experience MACEs in the long run if they have a CAC score below 100 or a CAVI score below 9.0. These two non-invasive tests can be used to screen for and direct treatment to prevent CV events in the future.

**Keywords:** Coronary artery disease • Arterial stiffness • Predictive • Cardiovascular

## Introduction

The accuracy of prediction may be improved by using an arterial stiffness measurement calculated from pulse wave velocity (PWV), the cardio-ankle vascular index (CAVI), or non-invasive imaging with coronary computed tomography (CT) scan. CAVI is more widely used in clinical practice than PWV as a surrogate of early atherosclerosis and is independent of blood pressure (BP) changes. Multiple studies confirm a strong association between CAVI and subclinical coronary artery disease (CAC scoring is an adjunctive test that enhances clinical risk prediction and more accurately classifies individuals with an intermediate to high ASCVD risk or symptomatic patients with stable CAD who might benefit from primary prevention using aspirin or statins. This is because evidence from the multi-ethnic study of atherosclerosis (MESA) indicates that CAC scoring can significantly improve classification, distinguish patients who are at risk, and help to guide primary prevention.

In order to confirm this relationship in a larger sample size with a longer

follow-up, we conducted a study to determine the predictive values of CAC score and arterial stiffness as reflected by CAVI on long-term cardiovascular (CV) events in both symptomatic patients with suspected stable CAD and asymptomatic patients with risk factors. Two previous small studies in asymptomatic subjects<sup>9</sup> and in asymptomatic patients with type 1 diabetes<sup>18</sup> demonstrated that CAVI has a dose-dependent association with increased CAC score [1].

## Materials and Methods

This was a review partner study enlisting back to back patients who went through coronary CT check for the evaluation of computer aided design at the High level Demonstrative Imaging Community (AIMC), Ramathibodi Clinic, Mahidol College, during November 2005–November 2013, and were followed up until December 2019. Criteria for inclusion included: adults over the age of 18; patients who had chest symptoms that were suspicious of CAD but were clinically stable, or patients who were asymptomatic and had a moderate to high risk of ASCVD<sup>19</sup>. Severe asthma was one of the exclusion criteria; high creatinine (more than 1.5 mg/dl); severe sensitivity to contrast or seafood; history of coronary stenting or bypass surgery in the past. The Ethics Committee of the Faculty of Medicine, Ramathibodi Hospital, Mahidol University (CAO) approved the study protocol because it complied with the Declaration of Helsinki's ethical guidelines from 1975. Before being enrolled in the study, written informed consent was obtained from each participant [2].

Age, sex, risk factors (such as smoking, diabetes mellitus [DM], hypertension, and hypercholesterolemia), body mass index (BMI, kg/m<sup>2</sup>), waist circumference (WC), and previous and current medications were all recorded for each subject. Serum creatinine, lipid profile, and fasting plasma

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glucose (FPG) were among the laboratory results. DM was defined as having an overnight FPG of less than 126 mg/dl or taking insulin. SBP (systolic blood pressure) of less than 140 mmHg and/or DBP (diastolic blood pressure) of less than 90 mmHg, respectively, were considered signs of hypertension. Current smoking, quitting smoking for more than a month, or never smoking were all considered being forms of smoking. An estimated glomerular filtration rate (eGFR) of 60 ml/min/1.73 m<sup>2</sup> was considered to be chronic kidney disease (CKD). The equations from the CKD Epidemiology Collaboration (CKD-EPI) were used to calculate the eGFR [3].

**Coronary CT** During the study, multidetector CT (MDCT) scans were performed using either a 320-slice CT scanner (Aquilion ONE, Toshiba) or a 64-slice CT scanner (Somatom Sensation 64 eco, Siemens). The CAC score and the degree and extent of CCTA stenosis were the two coronary CT scan findings used in this analysis. Using a commercially available external workstation, the Agatston method was used to calculate the CAC score. The sum of the individual lesion scores in each coronary artery was used to calculate the total CAC score, which was then divided into four groups: with no recognizable plaque; plaque with mild atherosclerosis; plaque with moderate atherosclerosis; and a 400-pound, extensive plaque of atherosclerosis. After injecting 70–90 milliliters of radiocontrast (Ultravist 370 mgI/ml, Bayer Healthcare Pharmaceuticals) through the right basilic vein with an 18-gauge intravenous catheter, the degree of coronary stenosis was measured. Following that, a 20 milliliter saline flush was performed at a flow rate of 5 milliliters per second. The contrast media's arrival and the scan were synchronized with automated bolus tracking. Images were taken after a delay of four seconds during a 5–10 s inspiratory breath hold [4].

### Treatment after coronary CTA

After coronary CTA, patients were scheduled to see a cardiologist or internist with the official CCTA report, CAVI, and baseline data (i.e., degree of CACS, severity, and location of coronary stenoses). Aspirin, statins, and risk factor modification were all administered to patients at the discretion of the physician following this evaluation. A cardiac stress test and/or an invasive coronary angiography with the possibility of revascularization (either percutaneous coronary intervention (PCI) or coronary artery bypass surgery, or CABG) may be performed on some patients [5].

### Statistical analysis

For continuous and categorical variables, the baseline characteristics were summarized as mean standard deviation (SD) and percentage, respectively. Then, using one-way ANOVA, quartile regression, or the 2 test, these were compared among CAC score groups. The 95% confidence interval (CI) was used to estimate the range of the MACE incidence. A cumulative incidence function (CIF) was estimated using a competing risk with sub-distribution hazard model taking into account other causes of death as competing risk events in order to evaluate the relationships between CAC score and CAVI and MACEs. The following steps were followed in order to determine whether CAC score, coronary stenosis, CAVI, and/or other significant risk factors were associated with a MACE (and not another cause of death as a competing risk) using a multivariate cause-specific Cox hazard (CSH) regression: First, the CSH model was fitted to each of the risk factors for a univariate analysis. Second, the multivariate CSH model simultaneously included risk factors whose p values were less than the uni-variate analysis. By removing each risk factor from the model, a backward elimination method was used. Only risk factors that were significant (p.05) were retained in the final model, along with the CAC score and CAVI. The HR and its 95 percent confidence interval (CI) for each risk factor were then estimated and reported [6].

Analyzing arterial calcification and arterial stiffness using the CAC score and CAVI, respectively, as risk factors for subsequent MACEs in patients with suspected stable CAD, this cohort was one of the largest ever studied? According to our findings, patients with CAC scores of 100–399 and 400 were approximately 1.70 and 1.9 times more likely than patients with CAC scores of zero to experience MACEs. Stenosis  $\geq 50\%$  in coronary courses was found to not be a critical indicator. MACEs were 1.27 times more likely to occur in patients with CAVIs 9.0 than in patients with CAVIs. For the following reasons, we included both asymptomatic patients with moderate to high ASCVD risk

and symptomatic stable CAD patients: First, to take into account the actual situation in the practice in order to meet the requirements of these patients who want to know if they have subclinical CAD. Second, to assist physicians in reclassifying the risks that patients face, and third, to broaden the range of disease severity. Our study protocol was one of a kind because it showed how CAC and CAVI differ in their ability to predict long-term MACEs in stable CAD patients with all stages of atherosclerosis—from no plaque with endothelial dysfunction to fibrofatty (or noncalcified) plaques to calcified plaques—and whether they do so. However, individuals with a CAC score of zero or may have a silent noncalcified plaque that could develop and rupture later, resulting in symptomatic CAD with MI, stroke, or sudden death. In our study, the percentage of patients who had CAC scores of 0 or 100 49.7% and 31.9%, respectively, constituted the majority of our cohort. CAC scores are well established as a predictor of future MACEs and are widely used in risk stratification for CAVI, as an early physiological surrogate marker of atherosclerosis<sup>28</sup>, may fill this void and allow for the prediction of CV events in these patients with minimal or no calcified plaque. CAVIs and CAC scores were found to be independent predictors of long-term MACEs after traditional risk factors like age, diabetes, hypertension, chronic kidney disease, and concurrent aspirin and statin therapy were taken into account. Although calcification was more predictive of MACEs than arterial stiffness, we also observed a dose-dependent association between these two measurements. In asymptomatic patients with risk factors or symptomatic patients with suspected stable CAD, independent risk predictors of long-term MACEs included CAC scores of less than 100 and CAVIs of less than 9.0 [5–8].

The CAVI was used as a categorical variable rather than a continuous one in this investigation. From the past writing, there might be some debate with regards to the ideal cut-off incentive for separating CV risk. Notwithstanding, a CAVI  $\geq 9$  is by and large thought to be high and to address the presence of atherosclerosis and to anticipate CV risk. True to form, CAVIs  $\geq 9$  was more successive in our patients who created MACEs than were CAVI  $< 9$ . CAVIs 9 remained one of the independent risk predictors of CV events despite not being as strong as the CAC score after other confounding factors were taken into account. The benefit of CAVI for subgroup patients with a CAC score of less than, as revealed by our study, is now better understood. CAVI 9 has the potential to enhance risk prediction for future MACEs in these patients with noncalcified plaque or a CAC score below. Both arterial stiffness and coronary CT scan findings (CAC score and degree of stenosis) have been found to be associated with coronary artery disease (CAD). These assessments are able to predict long-term cardiovascular events more accurately than ASCVD risk scores and aid in primary prevention. Notwithstanding, CAVI has been considered and utilized generally in Asian nations (particularly Japan) alongside a couple of European countries. Significantly, CAVI is vague and can be expanded in more established ages, guys, uncontrolled hypertension, and etcetera. In this manner, it ought to be utilized to direct preventive treatment in subjects who have essentially a transitional likelihood of computer aided design or in thought computer aided design patients with numerous gamble factors. CAVI has not been recommended in any guidelines and is currently not widely used in Western nations. However, as additional evidence accumulates, this straightforward, non-invasive test may be useful as an additional tool to direct treatment and prevention [9,10].

## Conclusion

In addition to traditional risk factors alone, the CAC score and arterial stiffness as measured by CAVI can improve risk assessment. Long-term MACEs were predicted for both asymptomatic patients with risk factors and symptomatic patients with suspected stable CAD with CAC scores of 100 and CAVIs of 9.0. CAVI has the advantage of being able to be used as a screening tool to predict CV risk in patients with noncalcified or fibrofatty plaque. However, validation of these two risk predictors is necessary to determine whether they enhance treatment guidance, prevent future CV events, and extend survival.

## Acknowledgement

None.

## Conflict of Interest

None.

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