

Relationship Between Cardio-Ankle Vascular Index (CAVI) and the Severity of Coronary Artery Lesions: A Case-Control Study

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Introduction: The cardio-ankle vascular index (CAVI) is an important metric for evaluating arterial stiffness (AS). In this study, we used cardio-ankle vascular index (CAVI) to predict CAD severity of coronary artery lesions.

Methods: This case-control study was conducted between October 2019 and December 2022. There were 222 patients divided into two groups: those with chronic coronary artery disease (CAD group), with 160 patients and a control group (non-CAD group) with 62 patients. The CAVI measurement and severity of coronary artery lesion parameters were evaluated (severity of stenosis, number of coronary artery diseases, syntax scores, and Gensini scores).

Results: The CAVI in the CAD group (9.21 ± 0.79) was significantly higher than that in the non-CAD group (8.48 ± 0.62) ($p < 0.001$). CAVI, with a cut-off point ≥ 8.83 , was a significant predictor of chronic CAD (OR = 9.6; 95% CI: 4.0–18.8) with an area under the curve (AUC) of 0.796 (95% CI: 0.736–0.856; $p < 0.001$). CAVI was significantly higher in severe stenosis ($\geq 75\%$) compared to moderate stenosis (9.41 ± 0.81 vs 9.02 ± 0.75 , $p = 0.002$). CAVI was higher in multivessel disease compared to single-vessel disease (9.43 ± 0.80 vs 8.90 ± 0.70 , $p < 0.001$). CAVI increased with higher SYNTAX scores (mild, moderate, and severe: 9.09 ± 0.65 , 9.80 ± 1.05 , and 9.45 ± 0.46 , respectively; $p < 0.001$) and Gensini scores (mild, moderate, and severe: 8.90 ± 0.65 , 9.37 ± 0.95 , and 9.43 ± 0.59 , respectively; $p < 0.001$).

Conclusion: CAVI is higher in chronic CAD and can predict chronic CAD with a cut-off point of ≥ 8.83 . The CAVI is higher in patients with severe stenosis, multivessel coronary artery disease, higher syntax scores, and higher Gensini scores.

Keywords: CAVI, CAD, syntax scores, gensini scores

Introduction

Atherosclerosis is the leading underlying pathology of cardiovascular disease. As reported by the World Health Organization in 2018, cardiovascular conditions accounted for 17.8 million deaths globally, representing 44% of non-communicable disease mortality and 31% of all-cause mortality.¹ The incidence of atherosclerosis-related mortality has markedly declined in high-income countries owing to improvements in prevention and treatment strategies; however, it continues to increase in low- and middle-income regions.¹

Assessment of atherosclerosis using non-invasive methods plays a critical role in the diagnosis, treatment, monitoring, and prediction of cardiovascular disease events. A few methods for evaluating atherosclerosis include pulse wave velocity (PWV),^{2,3} carotid intima-media thickness (IMT), augmentation index (AI), β -parameter,^{4,5} and ambulatory arterial stiffness index (AASI). However, these indices have certain limitations, such as the dependence of PWV on blood pressure at the time of measurement,⁶ and the β parameter, while independent of blood pressure, only provides an assessment of a localized arterial segment.⁷ The Cardio-Ankle Vascular Index (CAVI), which was introduced in Japan in 2004, addresses the key limitations of earlier vascular stiffness indices. According to previous studies, CAVI is an index

associated with atherosclerosis from the aorta to the lower limb arteries,⁷ demonstrating a stronger correlation with the severity of atherosclerosis than IMT,⁸ PWV,^{9,10} and β .¹¹

Several studies have indicated that the CAVI is significantly higher in patients with CAD than in those without CAD^{12,13} even in the absence of symptoms.¹⁴ Furthermore, elevated CAVI is associated with the progression of CAD severity,^{15,16} the degree of coronary artery stenosis,^{17,18} and the number of stenosed coronary arteries.^{13,19} CAVI is strongly correlated with coronary artery lesions^{12,20} and is valuable in predicting atherosclerotic CAD.^{13,21} Consequently, several studies have proposed CAVI as an effective tool for screening CAD, predicting the extent of coronary artery damage, and forecasting cardiovascular events (CVE) associated with CAD.^{12,22} It offers multiple advantages over other atherosclerosis assessment indices.^{23,24}

Recent studies have shown the significant association between the cardio-ankle vascular index (CAVI) and the progression of coronary artery disease (CAD) severity, highlighting its role as a predictor of cardiovascular (CV) events and coronary artery stenosis extent. Elevated CAVI values, particularly at or above 9.0, have been established as an optimal cutoff for predicting CV disease,²⁵ with psychological stress-induced increases in CAVI further emphasizing its prognostic value.²⁶ Beyond its predictive capacity, CAVI serves as a critical structural stiffness parameter, and its inclusion in risk assessment models has been shown to enhance predictive accuracy for CV events in patients without prior CVD history, opening new avenues for vascular function research.²⁷

However, variability remains in determining the CAVI cut-off point and establishing the sensitivity and specificity of the method for assessing CAD. Therefore, this study aimed to determine the cutoff point and use the CAVI to predict the severity of coronary artery lesions.

Materials and Methods

This case-control study was conducted between October 2019 and December 2022. Based on coronary angiography findings, the patients were divided into two groups: patients with chronic coronary artery disease (CAD group) and control group (Non – CAD group).

The study Group (CAD group) had percutaneous coronary angiography results showing $\geq 50\%$ stenosis of vessel diameter (significant stenosis). The control Group had percutaneous coronary angiography results showing $< 50\%$ stenosis of the vessel diameter (no stenosis or nonsignificant stenosis). The exclusion criteria for both the CAD and non-CAD groups included acute coronary syndrome, history of revascularization or coronary artery bypass grafting, comorbidities affecting CAVI measurement (bilateral peripheral artery disease, acute venous thrombosis of the limbs, Raynaud's syndrome, or limb amputation), and patients who did not consent to participate in the study.

Study Procedures

The study was conducted following these sequential steps:

Patient Recruitment

Patients with suspected chronic coronary artery disease (CCAD), as defined by the 2019 ESC guidelines, were screened for eligibility and subjected to the following assessments:

- + Medical History: Documentation of cardiovascular risk factors and comorbidities.
- + Clinical Examination: Comprehensive physical assessment of patients.
- + Laboratory and Diagnostic Evaluations: Analysis of blood biochemistry, complete blood count, electrocardiogram, and echocardiogram.
- + Indication for Coronary Angiography: Eligibility for percutaneous coronary angiography was determined based on predefined inclusion and exclusion criteria.

Cardio-Ankle Vascular Index (CAVI) Measurement Procedure

CAVI measurements were performed using the Vasera vs-1500N device (Fukuda-Denshi).

- + Measurement Environment: Measurements were conducted in a room shielded from electronic devices to prevent interference, with an ambient temperature maintained at 20–25°C to ensure patient comfort and muscle relaxation.

- + Patient Preparation: Patients were informed about the procedure to facilitate cooperation. They rested for ≥ 10 minutes in a supine position with arms and legs extended, muscles relaxed, and were instructed to remain still and silent.
- + Device Setup: Blood pressure cuffs, electrocardiogram electrodes, and a phonocardiogram microphone were attached to the patient.
- + Measurement Process: The device automatically (1) measured blood pressure in all four limbs simultaneously and (2) recorded pulse wave transit time from the aortic arch to the ankle. CAVI was calculated automatically using a pre-installed algorithm incorporating blood pressure and pulse wave transit time.

Percutaneous Coronary Angiography

Coronary angiography was performed according to standard protocols. Based on the results, patients were categorized into two groups: CAD (+) (study group) and CAD (-) (control group).

Statistical Analysis

Data were entered and managed using the Epidata software. Statistical processing and analysis were performed using SPSS version 20.0. Descriptive statistics included mean, median, and standard deviation for quantitative variables and frequency and percentage for qualitative variables. Pearson's, Fisher's exact, Kolmogorov–Smirnov, unpaired t-tests, and Mann–Whitney *U*-test were used, as appropriate. Receiver operating characteristic (ROC) curves were used to determine the area under the curve. Statistical significance was set at $P < 0.05$.

Ethics

This study was approved by the Ethics Committee of 108 Military Central Hospital (No.232/QĐ-VNC dated on 17th May, 2017) and adhered to the principles of the Helsinki Declaration. Written informed consent was obtained from all the patients.

Results

The results in Table 1 showed that there was no significant difference in the number of females and males and the mean age between the patients and controls. Hypertension was the most prevalent risk factor, whereas smoking had the lowest, with no significant differences between the two groups.

Table 2 showed the cardio-ankle vascular index measurement. The mean CAVI in the CAD (+) group was 9.20 ± 0.80 , which was significantly higher than that in the CAD (-) group (8.44 ± 0.63 , $p < 0.001$). The maximum CAVI in the CAD (+) group was 9.30 ± 0.81 , which was significantly higher than that in the CAD (-) group (8.54 ± 0.62 , $p < 0.001$).

Table 3 and Figure 1 show the CAVI as a Predictor of CCAD Risk. The area under the curve (AUC) for the mean CAVI was 0.796 (95% CI: 0.736–0.856, $p < 0.001$), and the maximum CAVI was 0.798 (95% CI: 0.738–0.857, $p < 0.001$). The cutoff point for predicting CCAD risk was ≥ 8.83 for mean CAVI, with an odds ratio (OR) of 9.6 (95% CI: 4.0–18.8), and ≥ 8.95 for maximum CAVI, with an OR of 9.9 (95% CI: 5.0–19.5).

Table 1 Patients Characteristics

Characteristics		CAD (-)	CAD (+)	p-Value
Female/Male, n (%)		37/25 (59.7/40.3)	91/69 (56.9/43.1)	0.705
Age, $\bar{X} \pm SD$ (years)		66.7 \pm 6.5	68.6 \pm 7.0	0.063
Age, n (%)	<65	21 (33.9)	43 (26.9)	0.436
	65-75	33 (53.2)	87 (54.3)	
	≥ 75	8 (12.9)	30 (18.8)	

(Continued)

Table 1 (Continued).

Characteristics		CAD (-)	CAD (+)	p-Value
Risk factors	Smoking	8 (12.9)	17 (10.6)	0.630
	Hypertension	52 (83.9)	138 (86.3)	0.651
	Diabetes	23 (37.1)	56 (35.0)	0.770
	Lipid disorders	50 (80.6)	143 (89.4)	0.083
	Obesity	28 (45.2)	84 (52.5)	0.327
	Stroke	3 (4.8)	14 (8.8)	-
	Renal failure (eGFR<60)	32 (51.6)	104 (65.0)	0.066

Table 2 Cardio-Ankle Vascular Index Measurement

Indexes	CAD (-)		CAD (+)		p-Value
	n	$\bar{X} \pm SD$	n	$\bar{X} \pm SD$	
CAVI right	62	8.43 ± 0.66	160	9.22 ± 0.80	<0.001
CAVI left	62	8.45 ± 0.62	160	9.18 ± 0.83	<0.001
CAVI mean	62	8.44 ± 0.63	160	9.20 ± 0.80	<0.001
CAVI max	62	8.54 ± 0.62	160	9.30 ± 0.81	<0.001

Table 4 and Table 5 showed cardio-ankle vascular index (CAVI) to predict the severity of coronary artery lesions. CAVI was significantly higher in severe stenosis ($\geq 75\%$) compared to moderate stenosis (9.41 ± 0.81 vs 9.02 ± 0.75 , $p = 0.002$). CAVI was higher in multivessel disease compared to single-vessel disease (9.43 ± 0.80 vs 8.90 ± 0.70 , $p < 0.001$). CAVI increased with higher SYNTAX scores (mild, moderate, and severe: 9.09 ± 0.65 , 9.80 ± 1.05 , and 9.45 ± 0.46 , respectively; $p < 0.001$) and Gensini scores (mild, moderate, and severe: 8.90 ± 0.65 , 9.37 ± 0.95 , and 9.43 ± 0.59 , respectively; $p < 0.001$).

CAVI showed a weak but statistically significant positive correlation with the degree of coronary artery stenosis ($r = 0.250$, $p = 0.001$), syntax score ($r = 0.275$, $p < 0.001$), and Gensini score ($r = 0.187$, $p = 0.018$).

Table 3 CAVI as a Predictor of CCAD Risk

		CAVI mean	CAVI max
The area under the curve (AUC)	AUC (95% CI)	0.796 (0.736–0.856)	0.798 (0.738–0.857)
	p-value	<0.001	<0.001
The cut-off point for predicting CCAD risk	Cut-off value	8.83	8.95
	OR (95% CI)	9.6 (4.0–18.8)	9.9 (5.0–19.5)
	p-value	<0.001	<0.001

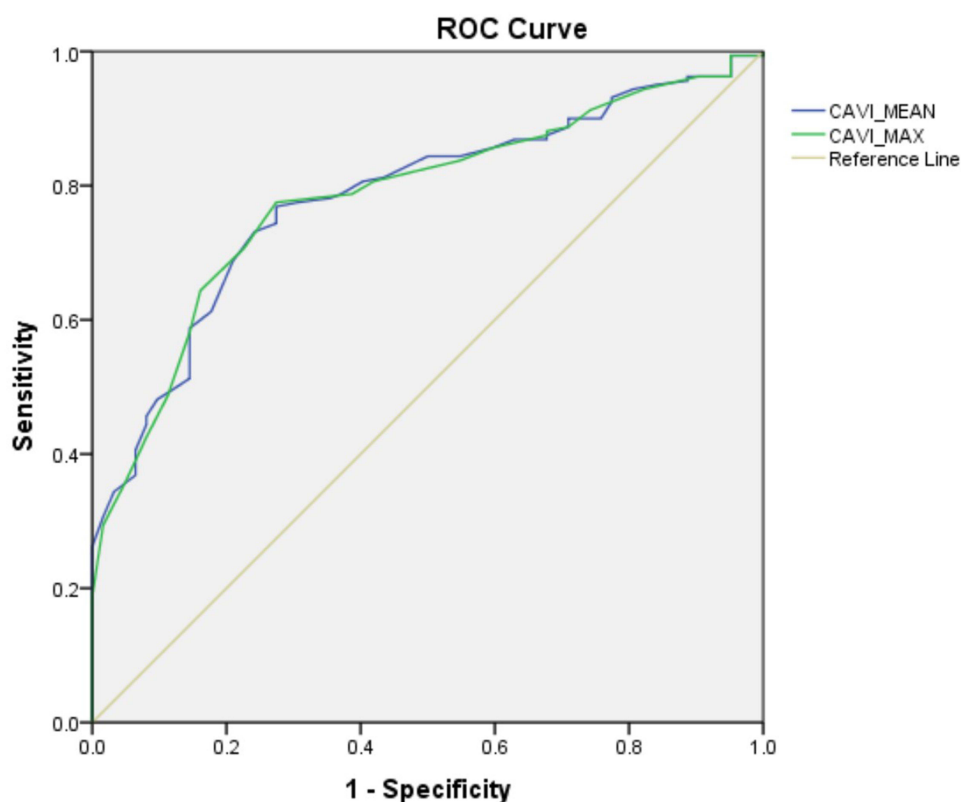


Figure 1 ROC for CAVI as a Predictor of CCAD Risk.

Discussion

Non-invasive assessments of the extent and progression of coronary artery atherosclerosis play a crucial role in screening, monitoring disease progression, guiding treatment, and preventing cardiovascular events in chronic coronary artery

Table 4 The Relationship Between Cardio-Ankle Vascular Index (CAVI) and the Severity of Coronary Artery Lesions

Characteristics	n	CAVI ($\bar{X} \pm SD$)	p-Value	
Degree of Coronary Artery Stenosis	Stenosis <75%	88	9.02 ± 0.75	0.002
	Stenosis ≥75%	72	9.41 ± 0.81	
Number of Significantly Stenosed Coronary Arteries	Single-vessel disease	70	8.90 ± 0.70	<0.001
	Multivessel disease	90	9.43 ± 0.80	
Syntax Score Risk Stratification	Mild (<23)	127	9.09 ± 0.78	0.002
	Moderate (23–33)	14	9.80 ± 1.05	
	Severe (≥33)	19	9.45 ± 0.46	
Gensini Score Risk Stratification	Mild (<20)	63	8.90 ± 0.65	<0.001
	Moderate (20–50)	61	9.37 ± 0.95	
	Severe (≥50)	36	9.43 ± 0.59	

Table 5 Correlation Between CAVI and Coronary Artery Lesions

Indexes	Correlation Coefficient	
	r	p-Value
Degree of coronary artery stenosis in the CAD group	0.250	0.001
Syntax score	0.275	<0.001
Gensini score	0.187	0.018

disease (CCAD). This study was designed to evaluate the characteristics of CAVI in patients with CCAD and its relationship with coronary artery lesions and cardiovascular events.

Our study results revealed that the mean CAVI in the CAD (+) group was 9.20 ± 0.80 , which was significantly higher than that in the CAD (-) group (8.48 ± 0.63 , $p < 0.01$). These findings are consistent with the analyses of case-control studies conducted by both international and domestic researchers in similar populations. Specifically, Miyoshi et al reported that the CAVI in the group with significant coronary artery stenosis ($\geq 50\%$ vessel diameter reduction on percutaneous coronary angiography) was higher than that in the group with non-significant stenosis ($< 50\%$) (9.1 ± 1.3 vs 8.7 ± 1.2 ; $p = 0.02$). A similar study by Izuhara et al also reported results that were consistent with the aforementioned findings. In their study, CAVI in the group with significant coronary artery stenosis was 8.7 ± 1.8 , which was higher than that in the group without significant stenosis (8.3 ± 1.7 , $p = 0.027$).⁸

Numerous studies have investigated the relationship between the CAVI and coronary artery disease (CAD). Patients with stable angina pectoris exhibited significantly higher CAVI values compared to the control group (9.71 ± 1.42 vs 9.10 ± 1.41 ; $p = 0.0178$).¹⁶ In patients with acute myocardial infarction (AMI) undergoing emergency coronary artery reperfusion, Sairaku et al observed that CAVI was significantly higher than that in patients with stable angina pectoris undergoing elective coronary revascularization (9.3 ± 1.3 vs 10.0 ± 1.7 ; $p = 0.0012$). This difference persisted even after adjusting for clinical variables that differed between the two groups. Additionally, CAVI significantly decreased six months post-intervention (10.9 ± 1.6 vs 10.0 ± 1.5 ; $p = 0.019$).¹⁵

Our study results demonstrated that CAVI varied across different syntax score risk levels and exhibited a weak but statistically significant positive correlation with the syntax score ($r = 0.275$, $p < 0.001$). In a study of 121 non-type 2 diabetes patients with suspected coronary artery disease (CAD) undergoing percutaneous coronary angiography, Gokdeniz et al found that CAVI was positively correlated with the SYNTAX score ($r = 0.537$, $p < 0.001$) and was an independent predictor of intermediate to high SYNTAX scores. With a cutoff value of 8.6, the CAVI had an AUC of 0.877 (95% CI: 0.805–0.929), with a sensitivity of 68.9% and specificity of 93.5%.²⁸ Korkmaz et al, in a study of 172 patients with acute CAD, reported that CAVI was independently correlated with SYNTAX score (95% CI: 15–29, $p = 0.021$). The Syntax scores for normal, borderline, and abnormal CAVI levels were 4.0 ± 3.7 , 11.1 ± 7.2 , and 14.1 ± 9.1 , respectively ($p = 0.009$).²⁹ CAVI varied across different risk levels according to the Gensini score and showed a weak but statistically significant positive correlation with the Gensini score ($r = 0.187$, $p = 0.018$). Miyoshi et al reported that CAVI was positively correlated with the coronary artery stenosis index (modified Gensini score) in patients with acute coronary syndrome undergoing percutaneous coronary angiography ($r = 0.32$, $p < 0.01$).¹² In a study of 548 subjects undergoing coronary angiography with optical coherence tomography to assess atherosclerotic plaque morphology, Sakaguchi et al found that CAVI correlated with the logarithm of the Gensini score and was higher in the group with coronary artery stenosis ($p < 0.001$). However, no correlation was observed between the CAVI and plaque morphology on optical coherence tomography.³⁰

The area under the curve (AUC) for the mean CAVI was 0.796 (95% CI: 0.736–0.856, $p < 0.001$), and the maximum CAVI was 0.798 (95% CI: 0.738–0.857, $p < 0.001$). In a study by Nakamura et al, the AUC was 0.775, with a cutoff point of 8.81, yielding a sensitivity of 83.9% and specificity of 70%.¹³

CAVI have consistently identified by numerous studies as an independent predictor of coronary artery disease (CAD). Analyses by Izuhara et al⁸ and Miyoshi et al¹² in patients with confirmed CAD using percutaneous coronary angiography

demonstrated that CAVI was independently correlated with CAD. Additionally, a study by Park et al in asymptomatic CAD patients diagnosed using multidetector computed tomography showed that a CAVI ≥ 8.0 independently correlated with the degree of coronary artery stenosis, even after adjusting for age, sex, and cardiovascular risk factors such as hypertension, type 2 diabetes, and dyslipidemia (OR = 3.143, 95% CI: 1.004–9.842; $p = 0.049$).¹⁴ Based on research findings, the cutoff value for CAVI in detecting the presence of coronary artery disease (CAD) varies between 7.8 and 9.0 across different studies. Some studies have reported that a CAVI ≥ 8.0 correlates with coronary artery stenosis of $\geq 50\%$,^{14,31} while a CAVI range of 8.91–9.0 correlates with stenosis of $\geq 75\%$.^{8,13}

Our study on CAVI in coronary artery disease (CAD) among Vietnamese patients yielded results consistent with those of studies conducted in Japan and other countries worldwide, confirming that CAVI is associated with the severity of coronary artery lesions and has predictive value for CAD.^{25–27} Furthermore, the value of CAVI lies in its noninvasive nature, simple measurement process, and minimal technical requirements while providing relatively stable results, making it widely applicable.

A limitation of this study was the exclusion of patients with a low ABI (<0.9), severe left ventricular dysfunction, and valvular heart disease, which restricts the generalizability of the findings. Nevertheless, the CAVI remains a valuable and effective tool for CAD screening, particularly in high-risk populations.

Conclusion

The CAVI is higher in chronic CAD and can predict chronic CAD with a cutoff point of ≥ 8.83 . The CAVI is higher in patients with severe stenosis, multivessel coronary artery disease, higher syntax scores, and higher Gensini scores.

Data Sharing Statement

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

The study was approved by the Institutional Review Board of 108 Military Central Hospital (No.232/QĐ-VNC dated 17th May, 2017). This study adhered to the principles of the Declaration of Helsinki for Biomedical Research.

Consent for Publication

All the authors have read and agreed to the published version of the manuscript.

Author Contributions

All authors made a significant contribution to the work reported, whether in the conception, study design, execution, acquisition of data, analysis, and interpretation, or in all these areas, took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no conflicts of interest in this work.

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