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Original Article

Cardio-ankle vascular index as a screening tool for coronary artery disease in patients with metabolic dysfunction-associated steatotic liver disease

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ABSTRACT

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) is a common hepatic disorder that significantly increases cardiovascular risk. However, no established screening method exists for detecting subclinical coronary artery disease (CAD) in this high-risk population. The cardio-ankle vascular index (CAVI), a blood pressure–independent measure of arterial stiffness, may offer a non-invasive tool for early detection of coronary atherosclerosis.

Methods: We retrospectively analyzed 295 patients with MASLD who underwent both CAVI measurement and coronary computed tomography angiography at a single center. Significant CAD was defined as ≥50 % luminal stenosis. Receiver operating characteristic (ROC) analysis was used to evaluate diagnostic performance. Multivariable logistic regression was performed to identify independent associations between elevated CAVI and CAD. The incremental predictive value of CAVI was assessed using net reclassification improvement (NRI).

Results: Of the 295 patients, 110 (37.3 %) had significant CAD. Patients with CAD had significantly higher CAVI values (9.0 vs. 8.7, $p = 0.007$). The area under the ROC curve for CAVI predicting CAD was 0.614. A CAVI cut-off of 8.6 provided a sensitivity of 66.4 % and a specificity of 56.8 %. CAVI ≥8.0 was independently associated with CAD (OR 3.44, 95 % CI: 1.06–11.2, $p = 0.040$). Adding CAVI to a conventional risk model improved risk reclassification (NRI 0.4236, $p < 0.001$), although the C-statistic change was not significant (from 0.724 to 0.725, $p = 0.835$).

Conclusions: CAVI is independently associated with significant coronary stenosis in MASLD patients and may serve as a non-invasive screening tool to enhance cardiovascular risk stratification. Its moderate diagnostic accuracy suggests utility as a component of a multifactorial risk assessment strategy.

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a recently redefined condition encompassing hepatic steatosis in the presence of one or more cardiometabolic risk factors, such as obesity, hypertension, dyslipidemia, and type 2 diabetes mellitus [1]. As a hepatic manifestation of systemic metabolic dysfunction, MASLD affects over 20 % of the global population and has been increasingly linked to adverse cardiovascular outcomes [2]. Among these, coronary artery disease (CAD) represents a major cause of morbidity and mortality [3,4]. However, cardiovascular risk stratification in MASLD remains suboptimal, as no specific screening algorithm has been established for the early detection of subclinical CAD in this population [5].

The cardio-ankle vascular index (CAVI) is a non-invasive marker of arterial stiffness, calculated from the pulse wave velocity and blood pressure values obtained between the heart and the ankle [6]. Unlike pulse wave velocity measurements, CAVI is independent of blood pressure at the time of measurement, which enhances its reproducibility and reliability [7]. It reflects the functional and structural integrity of the arterial wall and is influenced by endothelial dysfunction, atherosclerotic burden, and vascular aging [8]. CAVI has been shown to correlate with established cardiovascular risk factors and has been proposed as a surrogate marker for atherosclerotic disease [9]. We previously

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proposed the CAVI cut-offs of less than 8 as normal, between 8 and 9 as borderline, and above 9 as abnormal [8]. However, the clinical utility of CAVI in specific high-risk populations, such as patients with MASLD, has not been fully clarified.

The aim of this study was to investigate whether CAVI is useful for identifying significant CAD in patients with MASLD. Specifically, we sought to determine the association between elevated CAVI values and the presence of ≥50 % coronary artery stenosis on coronary computed tomography angiography (CCTA), and to assess the diagnostic accuracy of CAVI as a potential screening tool for subclinical CAD in this population.

Methods

Ethical considerations

This study was conducted in accordance with the principles outlined in the Declaration of Helsinki. The study protocol was approved by the Institutional Review Board of Okayama University Hospital (2507-031). Owing to the retrospective nature of the study using anonymized clinical data, the requirement for informed consent was waived.

Study design and participants

Fig. 1 shows a flow diagram of the study design. This retrospective observational study included patients who underwent both CAVI measurement and clinically indicated CCTA at Okayama University Hospital between January 2011 and December 2020. Among these, 295 patients met the diagnostic criteria for MASLD and were included in the final analysis. Patients with known CAD, hemodialysis, chronic liver diseases, alcohol intake >30 g or 20 g/day for men or women, or medications associated with hepatic steatosis hematological disease were excluded. Clinical data including demographics, comorbidities, and medication use were obtained from medical records.

Definition of MASLD

MASLD was defined based on recent international consensus criteria [1]: the presence of hepatic steatosis in conjunction with at least one of the following five cardiometabolic abnormalities: (i) body mass index ≥23 kg/m^2 or waist circumference >94 cm (men), >80 cm (women); (ii) blood pressure ≥130/85 mmHg or use of antihypertensive medication; (iii) fasting plasma glucose ≥100 mg/dL, 2-hour glucose ≥140 mg/dL during oral glucose tolerance test, hemoglobin A1c ≥5.7 %, or presence of type 2 diabetes mellitus; (iv) triglycerides ≥150 mg/dL or treatment with lipid-lowering agents; (v) high-density lipoprotein cholesterol <40 mg/dL (men) or <50 mg/dL (women), or treatment with lipid-lowering agents. Hepatic steatosis was assessed with the fatty liver index >60 [10].

Measurement of CAVI

CAVI was measured using a VaSera VS-1500 device (Fukuda Denshi, Tokyo, Japan). Measurements were conducted in the supine position after at least 5 min of rest [9]. The average of the left- and right-side measurements was used. CAVI values were analyzed both as a continuous variable and as a categorical variable (<8.0, 8.0–8.9, ≥9.0) to evaluate associations with CAD.

CCTA image acquisition

CCTA images were obtained as described previously [11]. Beta-blockers and sublingual nitroglycerin were administered as appropriate to optimize image quality [12]. CCTA images were assessed visually by experienced cardiologists blinded to clinical data. Significant stenosis was defined as luminal narrowing of ≥50 % in at least one major coronary artery segment [12].

Statistical analysis

Continuous variables were expressed as mean ± standard deviation or median (interquartile range) and compared using the paired Student's *t*-test or the Mann–Whitney *U* test. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Receiver-operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic accuracy of CAVI for detecting significant coronary stenosis. The optimal cut-off value was determined using the Youden index. C-statistics were calculated as the area under the ROC curves. A two-sided *p*-value <0.05 was considered statistically significant. All analyses were conducted using SPSS (version 29.0; IBM Corp., Armonk, NY, USA) and the R statistical package (version 4.1.1; R Foundation for Statistical Computing, Vienna, Austria).

Results

Patient characteristics

Among the 295 patients with MASLD analyzed, 110 (37.3 %) had significant coronary artery stenosis (≥50 %) (Table 1). Patients with

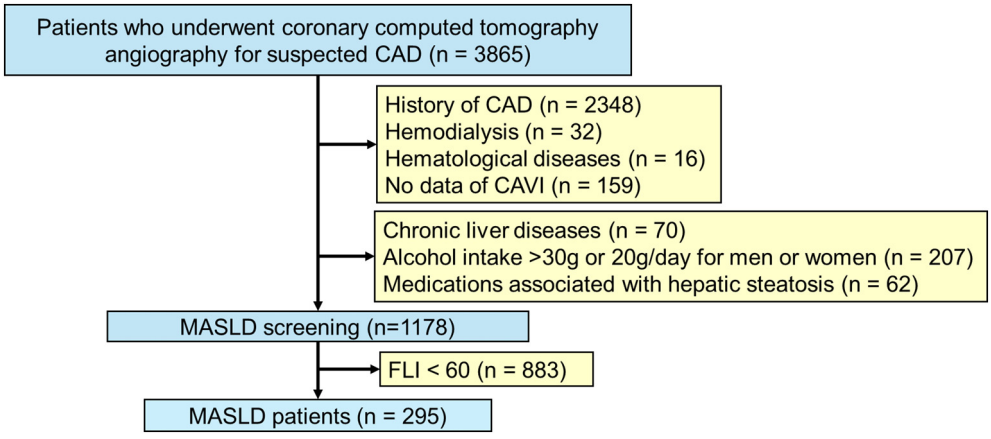

Fig. 1. Flow diagram of this study population.
From an initial cohort of patients evaluated by coronary computed tomography angiography, those meeting exclusion criteria were removed, and a final sample of 295 patients with assessable data was included in the analysis.
CAD, coronary artery disease; CAVI, cardio-ankle vascular index; FLI, fatty liver index; MASLD metabolic dysfunction-associated steatotic liver disease.

Table 1
Baseline clinical characteristics of patients with and without significant coronary artery stenosis.

Variables	All $n = 295$	With significant stenosis $n = 110$	Without significant stenosis $n = 185$	p-Value
Age, years	65 ± 11	68 ± 9	63 ± 12	<0.001
Men	195 (66.1)	78 (70.9)	117 (63.2)	0.204
Body mass index, kg/m^2	27.2 ± 3.8	27.3 ± 4.2	27.1 ± 3.6	0.802
Waist circumference, cm	94.1 ± 9.7	94.7 ± 10.5	93.8 ± 9.2	0.476
Current smoker	59 (20.0)	17 (15.5)	42 (22.7)	0.174
Hypertension	214 (72.5)	92 (83.6)	122 (65.9)	0.002
Diabetes mellitus	137 (46.4)	59 (53.6)	78 (42.2)	0.070
Dyslipidemia	195 (66.1)	83 (75.5)	112 (60.5)	0.014
Statins	132 (44.7)	64 (58.2)	68 (36.8)	0.001
Hypoglycemic agents	103 (34.9)	51 (46.4)	52 (28.1)	0.002
Platelet count, ×10^9/L	236.0 ± 67.5	220.7 ± 60.4	245.1 ± 70.0	0.003
AST, IU/L	25 ± 12	26 ± 15	24 ± 9	0.161
ALT, IU/L	28 ± 19	28 ± 20	28 ± 18	0.950
GGT, U/L	34 [23–55]	33 [21–54.5]	35 [23–55]	0.817
eGFR, mL/min/1.73m^2	70.1 ± 16.6	68.1 ± 15.6	71.3 ± 17.2	0.105
Triglyceride, mg/dL	153 [117–217]	151 [114–211.5]	158 [117–229]	0.515
LDL cholesterol, mg/dL	120.1 ± 32.6	115.6 ± 33.4	71.3 ± 17.2	0.065
CAVI	8.8 ± 0.8	9.0 ± 0.8	8.7 ± 0.8	0.007

Values are expressed as mean ± standard deviation or median [interquartile range] for continuous variables, and as number (percentage) for categorical variables.
AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, γ-glutamyl transpeptidase; eGFR, estimated glomerular filtration rate; LDL, low-density lipoprotein; CAVI, cardio-ankle vascular index.

stenosis were significantly older than those without ($p < 0.001$), and more frequently had hypertension ($p = 0.002$) and dyslipidemia ($p = 0.014$). Statin use ($p = 0.001$) and hypoglycemic agent use ($p = 0.002$) were also significantly more prevalent. Although the proportion of patients with diabetes mellitus was higher in the stenosis group, the difference did not reach statistical significance ($p = 0.070$). Similarly, male sex ($p = 0.204$) and current smoking ($p = 0.174$) showed no significant differences. Anthropometric parameters, including body mass index ($p = 0.802$) and waist circumference ($p = 0.476$), were comparable between groups. Platelet count was significantly lower in the stenosis group ($p = 0.003$), while liver enzymes and estimated glomerular filtration rate ($p = 0.105$) did not differ significantly. Lipid values, including triglycerides and low-density lipoprotein cholesterol, were also similar between groups ($p = 0.515$ and $p = 0.065$, respectively). Importantly, CAVI was significantly elevated in patients with significant stenosis (9.0 vs. 8.7, $p = 0.007$), supporting its association with coronary atherosclerosis.

After excluding individuals with a history of CAD, hemodialysis, hematologic disorders, missing CAVI data, chronic liver disease, excessive alcohol intake (>30 g/day for men or >20 g/day for women), or medications known to induce hepatic steatosis, a total of 1178 patients remained eligible for analysis (Online Table 1). Among these, 380 (32.3 %) had significant coronary artery stenosis (≥50 %). Notably, CAVI was significantly higher in patients with stenosis compared to those without (8.6 vs. 8.4, $p < 0.001$). In a separate analysis of 883 patients without MASLD (fatty liver index <60; Online Table 2), 270 (30.6 %) had significant coronary stenosis. Consistent with findings in the MASLD subgroup, CAVI was significantly elevated in those with stenosis (8.4 vs. 8.3, $p = 0.019$).

Table 2
Multivariable logistic regression analysis for predictors of significant coronary artery stenosis.

	Odds ratio (95 % CI)	p-Value
CAVI ≥8.0	3.44 (1.06–11.2)	0.040
Age ≥65 years	2.02 (1.11–3.68)	0.022
Male	1.51 (0.83–2.760)	0.181
Body mass index, per 1 point	1.02 (0.94–1.11)	0.632
Current smoker	0.474 (0.22–1.00)	0.050
Hypertension	2.28 (1.14–4.55)	0.020
Diabetes mellitus	0.58 (0.23–1.47)	0.249
Dyslipidemia	1.14 (0.57–2.29)	0.716
Statin	2.12 (1.11–4.06)	0.023
Hypoglycemic agents	3.20 (1.23–8.35)	0.017

CAVI, cardio-ankle vascular index; CI, confidence interval.

Diagnostic accuracy of CAVI for identifying patients with significant coronary artery stenosis

The prevalence of significant stenosis in patients with MASLD increased with higher CAVI levels: 15 % in patients with CAVI <8.0, 36 % in those with CAVI 8.0–8.9, and 47 % in those with CAVI ≥9.0. ROC curve analysis yielded an area under the curve of 0.614 (95 % confidence interval: 0.549–0.679) for CAVI in predicting significant stenosis (Fig. 2). Fig. 3 illustrates the diagnostic performance of the CAVI for detecting significant coronary artery stenosis (≥50 %) at three different cut-off values: 8.0, 8.6, and 9.0. At a cut-off of 8.0, CAVI demonstrated very high sensitivity (95.5 %) but low specificity (15.7 %), indicating its utility for screening with minimal false negatives, albeit at the expense of increased false positives. A cut-off value of 8.6, identified as optimal in this cohort, provided balanced diagnostic performance, with a sensitivity of 66.4 % and a specificity of 56.8 %. When the cut-off was raised to 9.0, specificity improved to 72.4 %, but sensitivity declined to 41.8 %, resulting in a higher rate of missed diagnoses.

Among the broader cohort of 1178 patients including both MASLD and non-MASLD individuals, the prevalence of significant coronary stenosis increased with higher CAVI levels: 25 % for CAVI <8.0, 35 % for CAVI 8.0–8.9, and 41 % for CAVI ≥9.0. In this group, a CAVI cut-off of 8.2 yielded a sensitivity of 68.2 % and a specificity of 53.3 % for detecting significant stenosis. Similarly, in the subgroup of 883 patients without MASLD (fatty liver index <60), the prevalence of stenosis also rose across increasing CAVI categories: 26 % for CAVI <8.0, 35 % for CAVI 8.0–8.9, and 36 % for CAVI ≥9.0. For this group, a CAVI cut-off of 8.1 demonstrated a sensitivity of 66.7 % and a specificity of 53.3 %.

Incremental diagnostic value of CAVI for significant coronary artery stenosis

In multivariable logistic regression analysis adjusted for age, sex, body mass index, current smoking, hypertension, diabetes, dyslipidemia, statin use, and anti-hyperglycemic medication, a CAVI ≥8.0 was independently associated with the presence of significant coronary stenosis (odds ratio 3.65, 95 % CI: 1.12–11.89, $p = 0.03$) (Table 2). When CAVI (as a binary variable using the cut-off of 8.6) was added to a baseline model consisting of conventional risk factors (age, sex, body mass

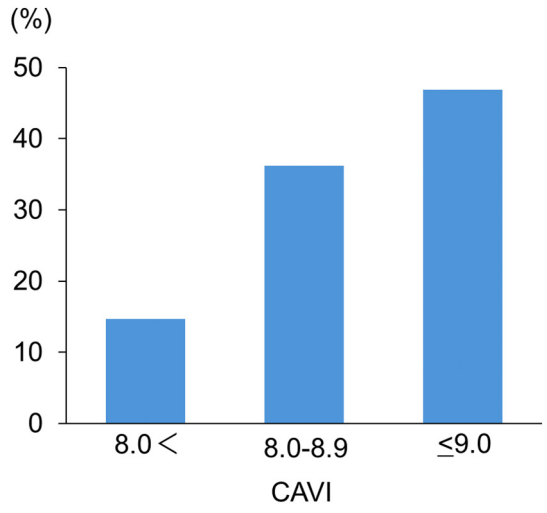

Fig. 2. Prevalence of significant coronary artery stenosis across CAVI strata. The proportion of patients with significant stenosis increases in a stepwise manner with rising CAVI categories, suggesting a positive association between arterial stiffness and coronary atherosclerosis.
CAVI, cardio-ankle vascular index.

index, current smoking, hypertension, diabetes mellitus, dyslipidemia, statin use), the global chi-square value increased from 43.732 to 45.984 ($p < 0.001$), and the net reclassification improvement was 0.4236 (95 % CI: 0.1907–0.6564, $p < 0.001$). However, the increase in the C-statistic from 0.724 to 0.725 was not statistically significant ($p = 0.835$) (Table 3).

Discussion

In this study, we demonstrated that the prevalence of significant coronary artery stenosis increased stepwise with higher CAVI values among patients with MASLD. Although the optimal cut-off value of CAVI for predicting significant stenosis was identified as 8.6, the diagnostic performance of CAVI alone was moderate. Nevertheless, the addition of CAVI to a prediction model based on conventional cardiovascular risk factors resulted in a modest but meaningful improvement in risk classification. These findings suggest that CAVI may be a valuable adjunctive tool for identifying significant CAD in patients with MASLD.

Previous studies have highlighted the elevated risk of atherosclerotic cardiovascular disease in patients with non-alcoholic fatty liver disease,

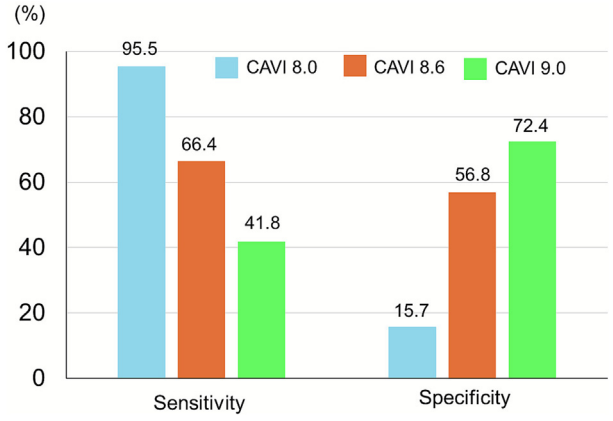

Fig. 3. Sensitivity and specificity of CAVI for detecting significant coronary artery stenosis at three different cut-off values (8.0, 8.6, and 9.0).
Lower thresholds yielded higher sensitivity but lower specificity, while higher thresholds improved specificity at the expense of sensitivity.
CAVI, cardio-ankle vascular index.

which largely overlaps with MASLD [4,13,14]. However, tools for early cardiovascular screening in this population remain limited. CAVI has been validated in large cohorts as a predictor of cardiovascular events [9,15]. Our findings build on this evidence and suggest that CAVI could be a useful screening marker in MASLD populations, which often present with multiple metabolic comorbidities that may mask early coronary disease.

In addition to the MASLD subgroup, our findings in the overall cohort ($n = 1178$) and the non-MASLD subgroup ($n = 883$) further support the utility of CAVI in stratifying CAD risk. In both populations, the prevalence of significant coronary stenosis increased stepwise with higher CAVI categories, echoing the trend observed in MASLD. Specifically, among patients with CAVI ≥9.0, the prevalence of stenosis was 41 % in the overall cohort and 36 % in the non-MASLD subgroup, compared to 25–26 % in those with CAVI <8.0. Notably, optimal cut-off values for predicting significant stenosis differed slightly across groups—CAVI 8.6 in the MASLD group, CAVI 8.2 in the overall population, and CAVI 8.1 in the non-MASLD group, suggesting that the discriminatory performance of CAVI may vary depending on underlying metabolic context. These results highlight the potential for population-specific thresholds when applying CAVI as a screening tool in broader clinical settings.

Regarding the optimal cut-off, our analysis identified a CAVI threshold of 8.6 as the best balance of sensitivity and specificity for predicting significant coronary stenosis. This is consistent with previous studies suggesting that CAVI values above 8.0 indicate increased cardiovascular risk [8]. Lowering the cut-off to 8.0 improved sensitivity to 95.5 % at the expense of specificity, whereas raising the cut-off to 9.0 improved specificity but decreased sensitivity, which is in line with our previous recommendation of diagnostic criteria of CAVI [8]. Therefore, the choice of cut-off should depend on the clinical context: a lower cut-off may be preferable in primary prevention to rule out patients with significant coronary stenosis, whereas a higher cut-off could be used in settings requiring higher diagnostic precision.

Despite its independent association with coronary artery stenosis, the discriminative ability of CAVI alone was modest, with a C-statistic of 0.614 and limited incremental improvement in the area under the curve when added to conventional risk models. However, the net reclassification improvement analysis showed significant enhancement in risk stratification, indicating that CAVI may provide additional value when integrated with existing clinical risk assessments. These findings are in line with previous reports that CAVI correlates with the incident cardiovascular events, particularly in high-risk populations [9,16]. Future studies should explore multimodal screening approaches combining CAVI with other non-invasive modalities, such as coronary artery calcium scoring or biomarker panels, to optimize early detection strategies for CAD in patients with MASLD.

Importantly, previous interventional studies have demonstrated that lifestyle modifications and pharmacological treatments targeting cardiovascular risk factors—such as blood pressure control, lipid-lowering therapy, glycemic control, and weight reduction—can lead to improvements in CAVI values [17–20]. This dynamic responsiveness of CAVI to treatment suggests that it reflects not only fixed structural damage but also reversible functional components of arterial stiffness. However, this modifiability raises critical considerations regarding the interpretation of CAVI in clinical screening. Specifically, in patients already undergoing intensive risk factor management (e.g. those on statins or antihypertensives), CAVI values may appear deceptively low despite the presence of subclinical atherosclerosis. Conversely, elevated CAVI in untreated individuals may represent both risk and opportunity for early intervention. Therefore, while CAVI shows promise as a non-invasive and practical screening tool in the MASLD population, its utility must be interpreted within the context of the patient's treatment status and longitudinal risk trajectory.

Several limitations of this study should be acknowledged. First, the study population comprised individuals with MASLD who underwent clinically indicated CCTA. Therefore, the findings may not be

Table 3
Incremental predictive value of CAVI when added to a conventional risk model for significant coronary artery stenosis.

Model	Global chi-square score		C-statistic		NRI (95 % CI)	
		p-Value		*p*-Value		*p*-Value
Age, sex, risk factors[a]	43.732		0.724			
With CAVI added	45.984	<0.001	0.725	0.835	0.4236 (0.1907–0.6564)	<0.001

CAVI, cardio-ankle vascular index; CI, confidence interval; NRI, net reclassification index.
[a] Risk factors included body mass index, current smoking, hypertension, diabetes mellitus, dyslipidemia, statin use.

generalizable to asymptomatic MASLD patients in the general population. Further studies are warranted to validate these findings in broader cohorts and across different ethnic groups. Second, the diagnosis of MASLD was based on the fatty liver index and metabolic parameters, rather than imaging confirmation or liver biopsy, which could lead to misclassification. Third, although CAVI and CCTA were performed within a clinically relevant timeframe, the timing was not standardized, and temporal variations might have affected the results. Fourth, while the study adjusted for major confounding factors, residual confounding by unmeasured variables (e.g. inflammatory markers, lifestyle factors) cannot be ruled out. Lastly, due to the modest discriminative power of CAVI alone, further validation in larger, prospective, and multi-ethnic cohorts is necessary.

Conclusion

This study demonstrates that increased CAVI values are independently associated with the presence of significant coronary artery stenosis in patients with MASLD. Although CAVI alone may not offer sufficient accuracy as a standalone diagnostic tool, it may serve as a useful component of an integrated, non-invasive screening strategy for early CAD detection in this high-risk population. Future studies should focus on refining prediction models that combine CAVI with other clinical, biochemical, and imaging markers to enhance cardiovascular risk stratification in MASLD.

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Declaration of competing interest

The authors declare that there is no conflict of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jjcc.2025.12.006.

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