

Original Article

Left anterior descending artery pericoronary adipose tissue (PCAT) attenuation independently distinguishes ACS from stable coronary disease, and multivessel coronary CT angiography-PCAT combination improves diagnostic accuracy

Hong-Ye Zuo, Yong-Bo Li, Ya-Peng Dong, Xin-Xin Luo, Hong-Wei Lv, Yan-Ping Wang, Lan Fu

Department of CT Diagnostic, Cangzhou Central Hospital, Cangzhou 061000, Hebei, China

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Abstract: Objectives: The purpose of this paper is to determine if the volume and attenuation of CCTA-derived PCAT can be used to distinguish stable coronary artery disease (CAD) in people with verified CAD. Methods: This retrospective study enrolled 180 patients with coronary angiography (CAG)-verified CAD, including 100 SCAD and 80 ACS. We measured PCAT metrics of the left anterior descending artery (LAD), left circumflex artery (LCX), and right coronary artery (RCA). Intergroup differences, correlations with stenosis severity, receiver operating characteristic (ROC) curves, and multivariate logistic regression were analyzed. Results: PCAT volume and attenuation of three coronary arteries were significantly higher in ACS patients (all $P < 0.001$). LAD attenuation index correlated strongest with stenosis ($r = 0.72$, $P < 0.001$), with an area under the curve (AUC) of 0.781 (cutoff: -68.5 HU). The three-vessel combined model yielded an AUC of 0.892. Adjusted regression identified LAD attenuation as an independent discriminator (odds ratio [OR] = 1.255, $P < 0.001$). Conclusion: CCTA-based PCAT parameters allow non-invasive differentiation between SCAD and ACS. LAD attenuation index serves as an independent predictor, and multi-vessel combined analysis further improves diagnostic efficiency.

Keywords: CCTA, PCAT, SCAD, attenuation index, predictive value

Introduction

Coronary artery disease is the leading cause of cardiovascular disability and death worldwide. In clinical practice, coronary artery disease (CAD) is classified into stable coronary artery disease (SCAD) and acute coronary syndrome (ACS). The long-term cardiovascular risk, intervention strategies, and prognosis differ significantly between these two categories, and early non-invasive differentiation can effectively reduce the risk of adverse events such as myocardial infarction and malignant arrhythmias. Current clinically available diagnostic methods have notable limitations: electrocardiography and stress testing have relatively low diagnostic sensitivity; invasive coronary angiography, although the gold standard, is associated with invasiveness, high cost, and contrast-

related complications; and conventional coronary CT angiography can assess luminal stenosis and plaque morphology but cannot reflect the degree of perivascular inflammatory activity. Therefore, there is an urgent clinical need for novel non-invasive imaging biomarkers to facilitate rapid risk stratification in patients with coronary artery disease. The pathologic basis of coronary artery disease is coronary atherosclerosis. Luminal narrowing or occlusion can result in insufficient myocardial blood supply, leading to myocardial ischemia and angina pectoris, and in severe cases, may trigger myocardial infarction [1, 2]. In clinical practice, coronary artery disease is divided mainly into acute coronary syndrome and stable coronary artery disease. ACS is a critical condition, while SCAD, as the major chronic subtype of coronary artery disease, also holds significant clinical value in

terms of long-term standardized management [3]. Current clinical diagnosis primarily relies on symptoms, electrocardiography, stress testing, coronary CT angiography, and invasive coronary angiography. However, all these approaches have limitations in the assessment of stable coronary artery disease: electrocardiography and stress testing lack sufficient sensitivity, and although invasive coronary angiography remains the diagnostic gold standard, it carries the drawbacks of invasiveness and high cost [4]. Consequently, the search for accurate, non-invasive imaging biomarkers for the early diagnosis and risk stratification of coronary artery disease has become a research hotspot.

With the continuous progress in medical imaging technology, CCTA has been applied for many years to study the anatomy and atherosclerotic plaques of the coronary arteries non-invasively [5, 6]. In this way, one can observe the degree of narrowing in the lumen and changes in the blood vessel wall of plaques (calcified, non-calcified, and mixed plaques). CT can also be employed to evaluate the functional index and plaque in the detection of CT-FFR (CT-derived fractional flow reserve) and plaque to extend the application clinically [7]. Conventional CCTA can clearly depict the degree of coronary luminal stenosis and the morphology of calcified, non-calcified, and mixed plaques, and can also assess coronary flow reserve function by CT-FFR. However, both approaches can reflect only vascular anatomy and hemodynamic changes, and cannot directly quantify the chronic inflammatory state surrounding the coronary vessel wall, making it difficult to identify high-risk individuals with early plaque instability. However, previous CCTA examinations have been centered on changes in the lumen size and plaque, so pericoronary adipose tissue (PCAT) test has not been employed. PCAT is a newly-recognized type of subcutaneous fat that can provide energy and mechanical support for the body, and it is also involved in the development and progression of coronary atherosclerosis through the release of paracrine and perivascular signals [8]. Under physiologic conditions, pericardial adipose tissue can secrete anti-inflammatory factors to protect the vascular endothelium. When atherosclerotic lesions occur in the coronary arteries, local vascular inflammation reversely stimulates the surrounding adipose tissues, inducing inflammatory cell infiltration, adipose edema, and interstitial fibrosis, which directly alter adipose CT

attenuation and results in an increased attenuation index (HU value). Concurrently, inflammatory stimulation promotes adipose hyperplasia, leading to a parallel increase in PCAT volume. Together, these two indices can non-invasively reflect the degree of inflammatory activity in coronary atherosclerosis. Research shows that PCAT is involved in inflammation, oxidative stress, and endothelial dysfunction of the blood vessels. The microenvironment of atherosclerosis is characterized by local inflammation and is therefore different in terms of cell size, lipids, and inflammatory factors than in health. A modification in the CT attenuation properties of fat will alter the biomarker function in imaging and therefore affect the test results [9, 10]. Research has been conducted so far on the use of PCAT volume and attenuation index derived from CCTA. PCAT volume is the amount of adipose tissue; attenuation index, which is a CT value (HU), indicates a change in density and may be due to inflammation or fibrosis. Yuki and others (2023) have shown that an increase in the CT attenuation value of PCAT is associated with fragile coronary plaques and cardiovascular disease [11]. Nonetheless, the value of PCAT volume and attenuation index in diagnosing SCAD has not been fully explored, and especially in patients with confirmed coronary artery disease, its ability to differentiate SCAD is still somewhat weak.

Clinical Significance of PCAT Volume and Attenuation Metrics from CCTA for SCAD Identification.

This study investigates the application value of CCTA-derived PCAT volume and attenuation index in the diagnosis of SCAD. By retrospectively reviewing the CCTA images of people with coronary artery disease who have been confirmed by coronary angiography, we have quantified the volume and mean CT density of PCAT to determine whether they are related to the severity of coronary stenosis and plaque characteristics. In addition to the clinical signs of this disease, we would like to know how well PCAT indices can predict the development of SCAD and will then develop a new non-invasive diagnostic system for medical use.

Materials and methods

Subject screening

Study design and case retrieval: This study is a single-center retrospective observational

study. Stable coronary artery disease (SCAD) group (SCAD group, n = 100): Patients met the diagnostic criteria for stable coronary artery disease, with CAG confirming at least 50% luminal stenosis in one major coronary artery, and no acute coronary events in the preceding three months.

Acute coronary syndrome (ACS) group (ACS group, n = 80): Patients met the diagnostic criteria for acute myocardial infarction (STEMI/NSTEMI) or unstable angina, with CAG confirming the presence of a culprit vessel lesion, and all patients were diagnosed with acute coronary events upon admission.

Diversity and Statistical Power of Samples: Cangzhou's primary cardiovascular medical centre is one of the six first-tier cardiovascular medical centres, and the number of monthly cardiac CT scans taken at the regional medical institution is between 450 and 550. In the past three months, about 1,400 to 1,500 cases have been collected to recruit roughly 180 qualified participants from all stages and different types of samples.

This study was approved by the Ethics Committee of Cangzhou Central Hospital. Owing to the retrospective nature of this study, the requirement for informed consent was waived.

Inclusion criteria: (1) Age 40-75 years, with complete medical records, imaging data, and laboratory test results; (2) All baseline demographic, biochemical, and clinical data (hypertension, diabetes, lipid profiles, statin medication history, BMI) were collected at the time of first admission, before coronary computed tomography angiography (CCTA) and coronary angiography; all patients underwent CCTA and coronary angiography within an interval of no more than 14 days; (3) Coronary angiography confirmed at least one major coronary artery with luminal stenosis $\geq 50\%$, establishing a diagnosis of coronary artery disease; (4) SCAD group: met the diagnostic criteria for stable coronary artery disease, with no acute chest pain, myocardial infarction, or other acute coronary events in the preceding 3 months; (5) ACS group: diagnosed with unstable angina, STEMI, or NSTEMI, with acute coronary-related clinical events at admission, and culprit vessel lesions identified on angiography.

Exclusion reasons: (1) Previous history of coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI); (2) Severe valvular heart disease or cardiomyopathy; (3) Active systemic inflammatory disease (e.g., rheumatoid arthritis, systemic lupus erythematosus); (4) Malignancy or active immunosuppressive therapy; (5) Poor CCTA image quality (severe motion artifacts or insufficient signal-to-noise ratio); (6) Missing clinical data; (7) Allergy to iodinated contrast media or severe renal insufficiency (eGFR < 30 mL/min) precluding tolerance of CCTA examination; (8) Large pericardial effusion or a huge mediastinal mass compressing the coronary arteries, which would interfere with precise segmentation and measurement of the PCAT boundary.

To reduce the influence on the PCAT attenuation index, the following confounding factors have been corrected: (1) The pharmacologic data were collected from the last three months. People who have been taking stable doses of statins regularly (more than five days a week) were excluded; those who have only been taking statins for a short time due to urgent reasons were recorded and classified for later study. (2) During CT scanning, tube voltage was automatically adjusted based on BMI (70-150 kV). (3) BMI was added as a covariate in the multivariate regression model to correct for any other effects.

All screened patients, reasons for exclusion, and the final derivation/validation cohorts are clearly presented in a flowchart in accordance with the STROBE statement (**Figure 1**).

The sample size of this study was estimated using the primary outcome indicator, the left anterior descending coronary artery pericoronary adipose tissue attenuation index (LAD PCAT attenuation index) - as the effect parameter. Based on the findings of a comparable study published by Kuneman et al. [12], the PCAT attenuation index around the culprit lesion was -63.8 ± 9.7 HU in the acute coronary syndrome (ACS) group and -69.6 ± 10.6 HU in the stable coronary artery disease (SCAD) group. Accordingly, the mean difference between the two groups was estimated to be approximately 5.8 HU, with a pooled standard deviation of approximately 10.2 HU. With a two-sided significance level (α) set at 0.05 and a statistical power ($1-\beta$) of 0.90, sample size cal-

PCAT volume and attenuation index derived in CAD patients

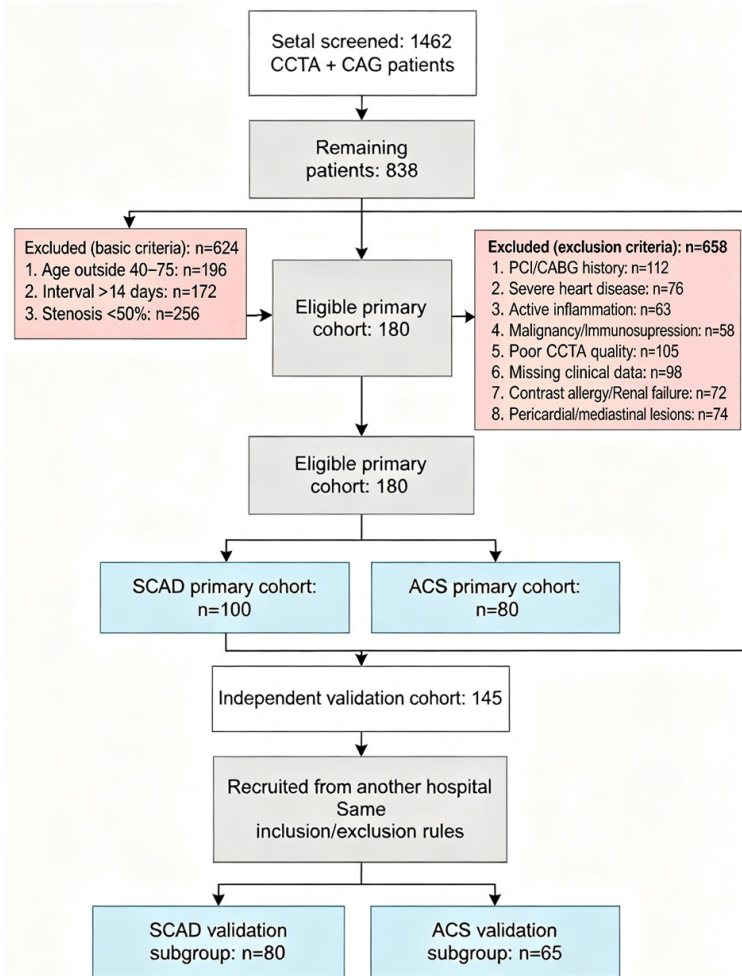


Figure 1. Flow chart of patient screening in the primary cohort and the independent external validation cohort. Note: The chart illustrates all screening procedures for study participants in accordance with the STROBE reporting guidelines. Initially, a total of 1,462 patients who underwent both CCTA and CAG were included in the screening for the primary cohort. After two rounds of selection based on the basic inclusion criteria and detailed exclusion criteria, 180 eligible patients were assigned to the SCAD group (n = 100) and the ACS group (n = 80). An independent validation cohort comprising 145 patients (SCAD: n = 80; ACS: n = 65) was recruited from another tertiary hospital using the same enrollment criteria. Abbreviations: CCTA, coronary computed tomography angiography; CAG, coronary angiography; SCAD, stable coronary artery disease; ACS, acute coronary syndrome; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting.

ulation was performed using an independent-samples t-test for comparison of two means, employing G*Power 3.1 software. The minimum required sample size was determined to be 65 cases per group. Considering the potential for missing data or suboptimal image quality inherent in retrospective studies, an additional 20% was added to the calculated sample size, resulting in a final target of at least 80 cases per group. Ultimately, 100 cases were

enrolled in the SCAD group and 80 cases in the ACS group, both exceeding the estimated minimum requirement, indicating that the study possessed adequate statistical power to detect between-group differences, with a relatively low probability of committing a type II error.

Data extraction and validation

CCTA scanning protocol: All CCTA images and the corresponding PCAT volume/attenuation index parameters were measured immediately after the completion of the scan, within the 14-day window between CCTA and invasive coronary angiography.

Siemens SOMATOM Force dual-source CT was used for the CCTA. Before the examination, if a person's heart rate was >70 bpm, 25-50 mg of oral metoprolol was administered to lower the heart rate; at the same time, everyone would receive 0.5 mg of nitroglycerin sublingually for vasodilation of the coronary arteries. A forward ECG-gated sequential scanning protocol has been chosen, and the range is now the tracheal bifurcation and cardiac diaphragm. Particular Settings are as follows: The tube voltage is 70-150 kV (automatically set according to body mass index), the tube current is 40-2600 mAs, the detector collimation is 2×192×

0.6 mm, the rotation duration is 0.25 s/rotation, and the pitch is between 0.15-3.2 (modulated according to heart rate). Iopromide (350 mg/mL) is an iodinated contrast agent; at a rate of 5.0 mL/s, a dual-syringe power injector will be employed to deliver a total volume of 60-80 mL, and then, at the same speed, 40 mL of saline was infused. Automatic bolus start was used; a region of interest (ROI) was set at the ascending aortic root, and then scanning

was started after it exceeded the threshold of 100 HU.

Methods for quantitative measurement of PCAT: Quantitative analysis of PCAT was performed with the help of Shukun PCAT analysis software. In terms of the position of the measurement segment and ROI division, the software automatically acquired the target vessel segments along the coronary artery course on CCTA axial images. According to the standardized measurement protocol, the segment of RCA was defined as 10-50 mm distal to its origin, while segments with anatomical variants or heavy calcification adjacent to the ostium were excluded to guarantee analysis on straight main-vessel segments. Both LAD and LCX segments were automatically selected for 40 mm in length starting from the left-main bifurcation. The perivascular adipose tissue zone was defined as the adipose region extending 4 mm outward from the outer vessel wall. We localized, then manually drew the ROI along the vessel wall margin. At this time, the axial image was displayed in real time; if there was a severely calcified lesion or stent artifact, that part was omitted, and otherwise, a continuous interval without interference next to it was chosen for further analysis to ensure that the total length of the vessel in the ROI meets the requirements. To know how far the PCAT boundary is, avoid interference, etc., the system automatically found and mapped adipose tissue in a 4 mm area outside the outer vessel wall to set that spot as the PCAT zone. The 4 mm margin is based on the general anatomical limit of PCAT; it does not interfere with the pericardial fat (which is outside the pericardium and usually more than 4 mm away from the coronary arteries) or epicardial fat tissue (which is more dispersed and not strictly limited to the area around the blood vessels). At the same time, there is a CT value screening mechanism in the program to keep only those voxels with CT values between -190 and -30 HU; this is the standard range for adipose tissue. This system does not include non-adipose tissue voxels, such as those in the vessel wall, calcified plaques and blood; these are out of this range, and thus the data are obtained from pure PCAT. PCAT Volume (units: cm³): With the help of 3D reconstruction technology, the total volume of all eligible voxels in a certain region that meet the CT value criterion can be determined by

the program. The number of voxels is then converted into a volume metric based on the voxel dimensions (which is determined by CT scan settings such as slice thickness and pixel spacing) to calculate the volume of PCAT accurately. PCAT attenuation index (unit: HU): The software takes the arithmetic mean of the CT values of all eligible voxels within the defined boundary. The above is the final average value, which is the PCAT attenuation index; it shows the density characteristics of PCAT and can be used to indirectly assess the degree of inflammation or fibrosis. Furthermore, to mitigate any measurement bias of PCAT caused by artifacts such as calcification and stents, in addition to selecting adjacent artifact-free segments for measurement, 0.75 mm original thin-slice images were used for analysis to minimize partial volume effects arising from the overlap of different tissue voxels. Concurrently, an artifact filtration algorithm was enabled during software operation to automatically identify and exclude voxels in high-density artifact regions associated with calcification (CT value >100 HU) and stent metal (CT value >1000 HU), retaining only pure PCAT voxels within the -190 to -30 HU range for parameter calculation, thereby reducing the impact of artifacts on the measurement results.

Assessment of coronary stenosis severity: Coronary angiography for stenosis grading was performed within 14 days after the completion of CCTA scanning, and the severity of stenosis was assessed on the day of angiography.

Coronary angiography (CAG) was used as the gold standard for assessing the severity of coronary stenosis. All angiographic examinations were performed within a 14-day window following the completion of CCTA. Two independent physicians with over 5 years of experience in cardiovascular interventional diagnosis and treatment reviewed the angiographic images in a blinded manner, with patient grouping, CCTA images, and PCAT measurements concealed during the reading process. In cases of disagreement between the two reviewers, they jointly reviewed multi-angle dynamic angiographic images and reached a consensus through discussion. Stenosis severity was quantified as the percentage of diameter stenosis, calculated as: stenosis rate (%) = (normal vessel diameter proximal to the lesion - minimal luminal diameter at the stenosis) ÷ normal vessel diameter proximal to the lesion × 100%.

Stenosis was then classified into four grades according to standard criteria: mild (<50%), moderate (50%-69%), severe (70%-99%), and total occlusion (100%). In the initial screening phase, only subjects with at least one major coronary artery stenosis $\geq 50\%$ were enrolled. For subsequent correlation analyses, the maximum stenosis percentage among the three major coronary arteries in each patient was used to represent the overall severity of coronary artery disease. For lesions that were difficult to assess, such as bifurcation lesions or those with diffuse calcification, measurements were comprehensively derived from multi-projection angiographic images to minimize measurement bias [13].

Data quality control and validation: To minimize the effect of manual operation and random errors on the results, multiple quality control measures were implemented: PCAT parameters for each coronary branch were measured three times independently, and the average of these three measurements was used as the final parameter value for that branch. Two physicians, each with over five years of experience in cardiovascular imaging diagnosis, independently performed all PCAT measurements for all cases without knowledge of each other's results; the average of the two physicians' measurements was used for subsequent statistical analysis. All measurements were conducted on original thin-slice images (slice thickness 0.75 mm), which minimize partial volume effects, i.e., CT value deviations caused by the overlap of different tissue voxels, thereby ensuring spatial resolution met the requirements for precise measurement.

Observational outcome measures

The primary outcome measure (left anterior descending artery PCAT attenuation index) was quantitatively analyzed based on CCTA images acquired within 14 days after admission. Secondary measures, including PCAT volume/attenuation of the three major epicardial coronary arteries, baseline laboratory lipid parameters, and percentage of coronary stenosis, were all measured at admission or within the 14-day window of the CCTA-CAG examination.

Primary outcome measure: The core evaluation index was the pericoronary adipose tissue (PCAT) attenuation index of the left anterior descending artery (LAD) measured by CCTA upon admission, which was analyzed for its

diagnostic efficacy in differentiating stable coronary artery disease (SCAD) from acute coronary syndrome (ACS). Quantitative evaluation metrics included the area under the receiver operating characteristic curve (AUC), optimal diagnostic HU threshold, diagnostic sensitivity, specificity, and overall accuracy. These metrics were recalculated in an independent external validation cohort to verify the stability and generalizability of the diagnostic results.

Secondary outcome measures: (1) Baseline demographic, clinical, and biochemical data at admission were collected for both groups. Intergroup comparisons were performed for age, sex, body mass index (BMI), total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), hypertension, diabetes mellitus, statin use, and other confounding variables. (2) Based on post-processed CCTA images, the PCAT volume and attenuation index of the three major coronary arteries—the left anterior descending artery (LAD), left circumflex artery (LCX), and right coronary artery (RCA)—were comprehensively acquired. Differences in each parameter between the SCAD group and the ACS group were compared. (3) After adjusting for confounding factors including age, lipid profiles, and underlying cardiovascular diseases, a multivariate logistic regression model was constructed to determine the odds ratios (ORs), 95% confidence intervals (CIs), and corresponding *P* values for each PCAT parameter as an independent predictor of ACS. (4) Multivariate logistic regression analysis was performed to establish the independent predictive value of PCAT parameters for ACS.

Independent cohort validation method: To validate the stability and generalizability of the optimal diagnostic threshold derived from this study, an independent external validation cohort was concurrently established at another tertiary hospital in the same city, applying the same inclusion and exclusion criteria. A total of 145 patients were eventually enrolled, including 80 cases in the SCAD group and 65 cases in the ACS group.

The validation cohort adhered to measurement standards, analytical software, vessel segment selection, and artifact exclusion principles that were entirely consistent with those of the primary cohort. Two radiologists with over 5 years of experience in cardiovascular imaging independently and blindly measured the LAD PCAT

attenuation values; disagreements were resolved through discussion to reach a consensus.

The optimal threshold derived from the primary cohort (-68.5 HU) was directly applied to the validation cohort, and the sensitivity, specificity, positive predictive value, negative predictive value, and AUC for differentiating SCAD from ACS were recalculated. The Z-test was used to compare the AUCs between the primary and validation cohorts. The Kappa coefficient was employed to evaluate the consistency of diagnostic results between the two cohorts: Kappa ≥ 0.75 indicated excellent agreement, 0.40–0.74 indicated fair to good agreement, and < 0.40 indicated poor agreement.

Statistical analysis

SPSS 22.0 was employed for the analysis of the data in this paper. Quantitative indicators that are normally distributed are presented as the mean $\pm$ standard deviation ($\bar{x} \pm s$), and qualitative indicators are displayed as n and %. For measured data not meeting the normal distribution, the Mann-Whitney test was adopted. For measured data meeting the normal distribution, one-way analysis of variance was adopted. A χ^2 test was carried out on the categorical variable. Using coronary artery disease type (SCAD = 0, ACS = 1) as the binary dependent variable, univariate logistic regression was first performed, incorporating age, hypertension, diabetes mellitus, lipid parameters, and PCAT parameters of each coronary artery as independent variables, and variables with $P < 0.1$ were selected as candidates for the multivariate model. Subsequently, a multivariate logistic regression model was constructed using the forward stepwise LR method, adjusting for cardiovascular confounding factors including age, hypertension, and low-density lipoprotein cholesterol, while eliminating variables without statistical significance. The model output included regression coefficient β , odds ratio (OR), 95% confidence interval of OR, and corresponding P values. The Hosmer-Lemeshow test was used to assess the goodness-of-fit of the model, with $P > 0.05$ indicating satisfactory model calibration. In addition, for correlation and ROC curve analyses, Pearson correlation analysis was employed to explore the linear association between each PCAT parameter and the severity of coronary stenosis, with the correlation coefficient r and P value reported. In this study, variables with $P < 0.1$ by univariate logistic regres-

sion were included as candidates for the multivariate model. Hypertension had a P value of 0.083 by univariate analysis, which was close to the screening threshold. Additionally, hypertension is a traditional core risk factor for coronary heart disease; therefore, it was retained in the multivariate regression model to adjust adequately for clinical confounding. Receiver operating characteristic (ROC) curves were used to calculate the area under the curve (AUC), 95% confidence interval, optimal diagnostic threshold, sensitivity (Sen), specificity (Spe), positive predictive value, and negative predictive value for single-vessel and multi-vessel combined PCAT parameter diagnostic models. Comparisons of AUC values between two groups were performed using the Z-test. Diagnostic consistency between the independent validation cohort and the original cohort was evaluated using the Kappa coefficient. $P < 0.05$ was regarded as significant.

Results

Contrast of baseline characteristics of subjects

A comparison was made between the original main cohort and the external validation cohort regarding all baseline general data, including age, sex, BMI, smoking history, hypertension, diabetes, various lipid profiles, statin use, and other indicators. There were no statistically significant differences in all baseline indicators between the two groups (all $P > 0.05$), indicating that the baseline characteristics were balanced and comparable. Detailed comparative data are shown in **Table 1**.

The contrast of baseline clinical characteristics of subjects in the primary cohort (**Table 2**) showed no significant intergroup differences in age ($P = 0.372$), gender composition ($P = 0.839$), BMI ($P = 0.392$), smoking history ($P = 0.567$), hypertension prevalence ($P = 0.730$), diabetes prevalence ($P = 0.618$), TC ($P = 0.120$), LDL-C ($P = 0.237$) and HDL-C ($P = 0.284$). Only triglycerides exhibited a statistically significant difference between groups ($P = 0.049$). These findings demonstrate that the majority of baseline confounding factors were balanced between the two groups.

Contrast of PCAT volume and attenuation index in subjects

The measurement results of PCAT parameters (**Figure 2**) showed that there were visible dis-

Table 1. Comparison of baseline data between the original main cohort and the external validation cohort

Indicator	Original main cohort (n = 180)	External validation cohort (n = 145)	Statistical measures	P value
Age (years)	61.74±10.97	58.86±7.93	t = 1.875	0.062
Male [n (%)]	103 (57.22%)	87 (60.00%)	$\chi^2 = 0.225$	0.613
BMI (kg/m ²)	25.35±3.36	24.97±3.15	t = 1.042	0.298
Smoking history [n (%)]	80 (44.44%)	64 (44.14%)	$\chi^2 = 0.003$	0.958
Hypertension [n (%)]	114 (63.33%)	89 (61.38%)	$\chi^2 = 0.131$	0.718
Diabetes [n (%)]	61 (33.89%)	46 (31.72%)	$\chi^2 = 0.171$	0.711
Statin use [n (%)]	9 (5.00%)	7 (4.83%)	$\chi^2 = 0.004$	0.679
Total cholesterol TC (mmol/L)	4.96±1.18	4.88±1.13	t = 0.619	0.536
Triglycerides TG (mmol/L)	1.81±0.60	1.76±0.57	t = 0.763	0.446
LDL-C (mmol/L)	3.32±0.97	3.26±0.91	t = 0.570	0.569
HDL-C (mmol/L)	1.18±0.34	1.20±0.31	t = 0.547	0.585

Note: BMI, body mass index; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

Table 2. Comparison of baseline characteristics between SCAD and ACS groups

Variable	SCAD group (n = 100)	ACS group (n = 80)	Statistic	P
Age (years)	62.34±8.71	60.92±9.23	t = 0.896	0.372
Male [n (%)]	52 (65.00%)	51 (63.80%)	$\chi^2 = 0.041$	0.839
BMI (kg/m ²)	25.13±3.24	25.63±3.52	t = 0.857	0.392
Smoking history [n (%)]	38 (48.00%)	42 (52.50%)	$\chi^2 = 0.328$	0.567
Hypertension [n (%)]	56 (70.00%)	58 (72.50%)	$\chi^2 = 0.119$	0.730
Diabetes mellitus [n (%)]	32 (40.00%)	29 (36.30%)	$\chi^2 = 0.248$	0.618
TC (mmol/L)	4.82±1.12	5.13±1.23	t = 1.563	0.120
TG (mmol/L)	1.72±0.58	1.93±0.62	t = 1.987	0.049
LDL-C (mmol/L)	3.23±0.92	3.42±1.03	t = 1.185	0.237
HDL-C (mmol/L)	1.21±0.35	1.15±0.32	t = 1.074	0.284

Note: BMI, body mass index; TC, total cholesterol; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SCAD, stable coronary artery disease; ACS, acute coronary syndrome.

cy (AUC = 0.781, optimal cut-off -68.5 HU, Sen 82.50%, Spe 80.00%). The multi-parameter model (integrating PCAT parameters from three vessels) achieved the optimal predictive performance (AUC = 0.892, Sen 85.67%, Spe 87.92%, Acc 86.79%), with a significantly higher AUC compared to any single parameter ($P < 0.05$) (**Figures 3 and 4**).

Correlation analysis between PCAT parameters and the severity of coronary artery stenosis

tinctions in all measured indicators ($P < 0.001$). Specifically, in the three vascular regions (LAD, LCX, and RCA), PCAT volume in the ACS group was markedly greater than that of the SCAD group. The attenuation index of adipose tissue in these three vascular regions was also markedly higher in the AG than in the SG.

Diagnostic efficacy of PCAT parameters in differentiating SCAD from ACS

Predictive performance of PCAT parameters for myocardial infarction: The LAD attenuation index demonstrated the best predictive effica-

Further Pearson correlation analysis showed (**Table 3**) that both the PCAT attenuation index and volume of the LAD, LCX, and RCA were positively correlated with the severity of coronary artery stenosis to varying degrees. Among these, the LAD PCAT attenuation index had the strongest correlation ($r = 0.72$, $P < 0.001$). Although the correlations of LCX-derived PCAT parameters were relatively lower, both LCX PCAT attenuation index ($r = 0.63$, $P < 0.001$) and LCX PCAT volume ($r = 0.59$, $P < 0.001$) exhibited moderate-to-strong positive correlations.

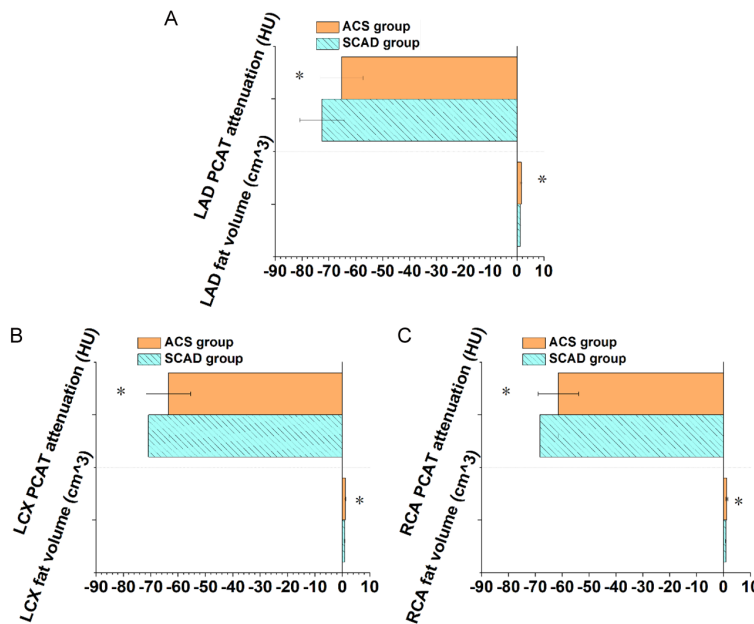


Figure 2. Comparison of PCAT volume and attenuation index between ACS and SCAD groups for three main coronary arteries (A: LAD PCAT metrics comparison; B: LCX PCAT metrics comparison; C: RCA PCAT metrics comparison). Note: * $P < 0.05$ versus SCAD group; PCAT, peri-coronary adipose tissue; ACS, acute coronary syndrome; SCAD, stable coronary artery disease; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

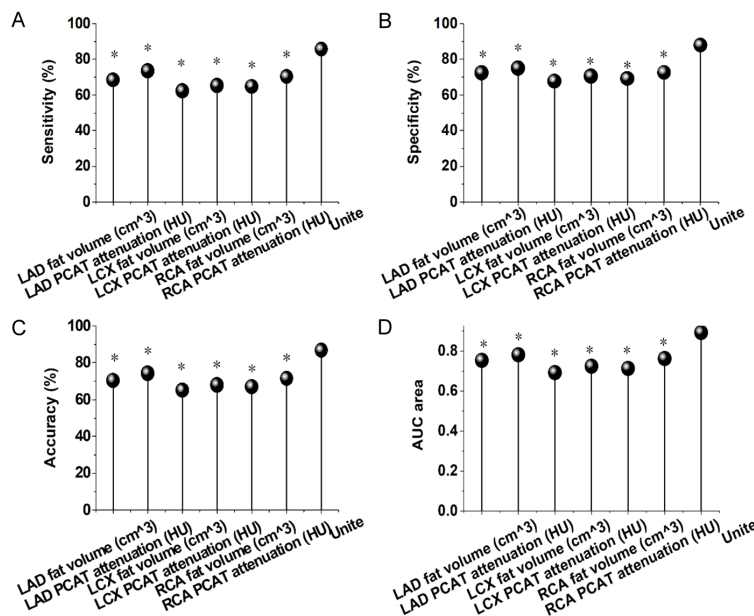


Figure 3. Diagnostic Sen, Spe, Acc and AUC area of PCAT parameters for SCAD. Note: A. Sensitivity of each PCAT parameter. B. Specificity of each PCAT parameter. C. Accuracy of each PCAT parameter. D. AUC values of each PCAT parameter. *Compared to the three-vessel combined model, $P < 0.05$; Sen, sensitivity; Spe, specificity; Acc, accuracy; PCAT, peri-coronary adipose tissue; AUC, area under curve; HU, Hounsfield unit; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

Multivariate logistic regression analysis for differentiating SCAD from ACS

The primary outcome variable in this study was the clinical classification of coronary artery disease, with assignment criteria as follows: stable coronary artery disease (SCAD) was coded as 0, and acute coronary syndrome (ACS) was coded as 1. The predictive variables incorporated into the analysis included the binary variables of hypertension and diabetes mellitus, as well as the continuous variables of low-density lipoprotein cholesterol (LDL-C) and the left anterior descending coronary artery pericoronary adipose tissue attenuation index (LAD PCAT attenuation index). The assignment rules for binary variables were: absence of a history of hypertension or diabetes mellitus = 0, and presence of a history = 1. Age, LDL-C, and the LAD PCAT attenuation index were all entered into the model as raw measured values, without any categorical transformation, and then univariate logistic regression analysis was conducted first. As shown in **Table 4**, hypertension, LDL-C and the attenuation index of LAD PCAT were all associated with ACS (all $P < 0.1$).

Based on the above analysis, the significant variables were then entered into a multivariate logistic regression model, with adjustment for confounding factors. As shown in **Table 5**, the LAD PCAT attenuation index could distinguish between SCAD and ACS independently; the odds ratio is 1.255, with a 95% confidence interval of 1.138-1.384 and $P < 0.001$.

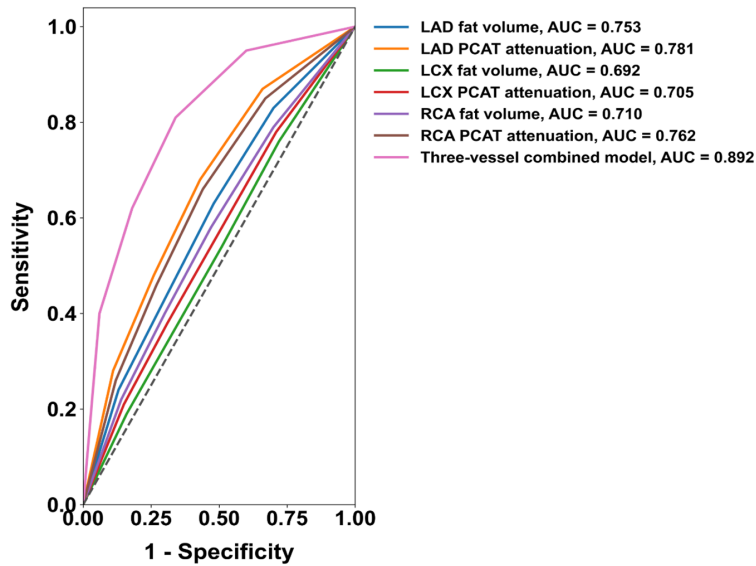


Figure 4. ROC curves of various peri-coronary adipose tissue parameters for differentiating stable coronary artery disease. Note: The figure includes curves for seven diagnostic indicators: left anterior descending artery fat volume, left anterior descending artery peri-coronary adipose tissue attenuation index, left circumflex artery fat volume, left circumflex artery peri-coronary adipose tissue attenuation index, right coronary artery fat volume, right coronary artery peri-coronary adipose tissue attenuation index, and the combined three-vessel diagnostic model. Abbreviations: AUC, area under the receiver operating characteristic curve; PCAT, peri-coronary adipose tissue; SCAD, stable coronary artery disease; ACS, acute coronary syndrome; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; HU, Hounsfield unit; Sen, sensitivity; Spe, specificity; Acc, accuracy.

Table 3. Pearson correlation analysis between PCAT parameters and the severity of coronary artery stenosis

Parameter	Correlation coefficient r	P value
LAD PCAT attenuation index	0.72	<0.001
LCX PCAT attenuation index	0.63	<0.001
RCA PCAT attenuation index	0.65	<0.001
LAD PCAT volume	0.66	<0.001
LCX PCAT volume	0.59	<0.001
RCA PCAT volume	0.62	<0.001

Note: The severity of coronary artery stenosis was defined as the maximum percentage of diameter stenosis among the three major coronary arteries (LAD, LCX, and RCA) for each patient. LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; PCAT, pericoronary adipose tissue.

Typical coronary CTA imaging findings in patients

Axial coronary CTA images were compared between the two groups of patients in the SCAD

group and the ACS group. The imaging findings of typical cases are shown in **Figure 5**.

Independent cohort validation results of the key threshold

The independent validation cohort included 145 patients, comprising 80 SCAD cases (48 males, 32 females, aged 58.2 ± 7.6 years) and 65 ACS cases (39 males, 26 females, aged 59.5 ± 8.1 years). Baseline characteristics showed no significant differences from the original cohort ($P > 0.05$). The diagnostic efficacy of the LAD attenuation index with a cut-off value > 68.5 HU for differentiating SCAD from ACS, as well as the consistent results between the two cohorts, are presented in **Table 6**.

Discussion

Currently, a major challenge in CAD management is the inability to detect changes in clinical status promptly [14].

CAG is the traditional diagnostic method; it is relatively invasive and expensive. Standard CTA is based on the degree of luminal narrowing and cannot detect early-stage diseases well. With the progress in imaging technology in recent years, PCAT has gradually been used to assess the degree of coronary inflammation and atherosclerosis [15]. This article innovatively and systematically evaluated the application value of PCAT volume and attenuation index in the diagnosis of SCAD, providing important references for clinical practice. According to the data, the quantity and absorption of PCAT are considerably higher in the LAD, LCX, and RCA areas of ACS subjects than in SCAD subjects. This finding is highly consistent with the pathologic features of more pronounced PCAT inflammatory responses reported in previous studies of patients with acute coronary events. The attenuation index of the LAD area (AUC = 0.781) was relatively high, possibly due to anatomic factors such as a broader perfusion territory and a large proportion of lesions.

Table 4. Univariate logistic regression analysis of risk factors for acute myocardial infarction

Risk Factor	β value	OR value	95% CI	P value
Age	0.031	1.031	0.986-1.079	0.172
Hypertension	0.247	1.280	0.964-1.698	0.083
Diabetes	0.195	1.215	0.901-1.639	0.201
LDL-C	0.176	1.192	1.012-1.404	0.036
LAD PCAT attenuation index	0.284	1.328	1.201-1.468	<0.001

Note: Dependent variable: classification of coronary artery disease, with stable coronary artery disease (SCAD) coded as 0 and acute coronary syndrome (ACS) coded as 1. Binary independent variables: hypertension and diabetes mellitus, coded as 0 for absence and 1 for presence. Continuous independent variables: age, low-density lipoprotein cholesterol (LDL-C), and left anterior descending artery peri-coronary adipose tissue attenuation index, which were directly entered as raw numerical values without the need for categorical assignment or transformation. In the univariate analysis, hypertension had a *P* value of 0.083, which was close to the predefined variable selection threshold of *P*<0.1 in this study. Moreover, hypertension is a well-recognized classic confounder for cardiovascular disease; therefore, it was retained in the multivariable model to adjust for confounding effects. Abbreviations: SCAD, stable coronary artery disease; ACS, acute coronary syndrome; LDL-C, low-density lipoprotein cholesterol; LAD, left anterior descending artery; PCAT, peri-coronary adipose tissue; OR, odds ratio; CI, confidence interval.

Table 5. Multivariate logistic regression analysis of risk factors for acute myocardial infarction

Risk factor	β value	OR value	95% CI	P value
Hypertension	0.182	1.199	0.879-1.636	0.247
LDL-C	0.124	1.132	0.948-1.353	0.169
LAD PCAT attenuation index	0.227	1.255	1.138-1.384	<0.001

Note: LAD, left anterior descending artery; PCAT, peri-coronary adipose tissue; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio; CI, confidence interval.

At the same time, the diagnostic performance of LCX markers was also relatively low (AUC = 0.692), which may be due to technical reasons, such as a large variation in the trajectory of large vessels and the effect of cardiac motion. The anatomy of the LCX has a convoluted path in the atrioventricular groove, and multiple branches originate from different places (such as the early obtuse marginal artery), precluding precise measurement within the 10-40 mm segment; some PCAT might be missed, and the results will be inaccurate. Additionally, the LCX is also affected by atrial and ventricular motion, and its displacement is relatively large at about 2-3 mm compared with only 1-1.5 mm for the LAD; therefore, there is still some blurring even after prospective ECG gating, which reduces the accuracy of the measurement. To address the problem above, we carried out segmented LCX quantification in the ranges of -20 mm and 20-40 mm, adjusted the phase window to 75%-

80% of the R-R interval, but it was not very effective; therefore, AI-driven automatic segmentation that considers the shape of LCX is needed. These results suggest that in clinical applications, priority should be given to PCAT features in the LAD region, combined with multi-vessel assessment to improve diagnostic Acc [16, 17].

The data from this study demonstrated that both PCAT volume and attenuation index around the three major coronary arteries were significantly higher in ACS patients than in SCAD patients, a finding highly consistent with several previous authoritative studies. Goeller et al. confirmed through a case-control study that pericoronary adipose tissue inflammatory activity surrounding culprit lesions in ACS was significantly higher than that around stable stenotic lesions in stable coronary artery disease [18]. The prospective CRISP-CT study conducted by Oikonomou et al. confirmed

that an elevated pericoronary adipose tissue attenuation index is significantly correlated with aggravated coronary artery inflammation and an increased risk of future cardiovascular events, thereby providing robust evidence for the close association between PCAT inflammatory burden, plaque vulnerability, and acute coronary events [19]. Lin et al. also demonstrated in a cross-sectional study that PCAT attenuation values could differentiate the severity of coronary artery disease, with higher inflammatory burden corresponding to higher adipose CT Hounsfield unit values [20]. At the pathophysiologic level, there exists a paracrine interaction pathway between coronary vessels and surrounding pericoronary adipose tissue. Vulnerable plaques in ACS continuously release large amounts of pro-inflammatory mediators such as IL-6 and TNF- α , which induce inflammatory cell infiltration, interstitial edema, and fibrosis to the adjacent adipose tissue, directly

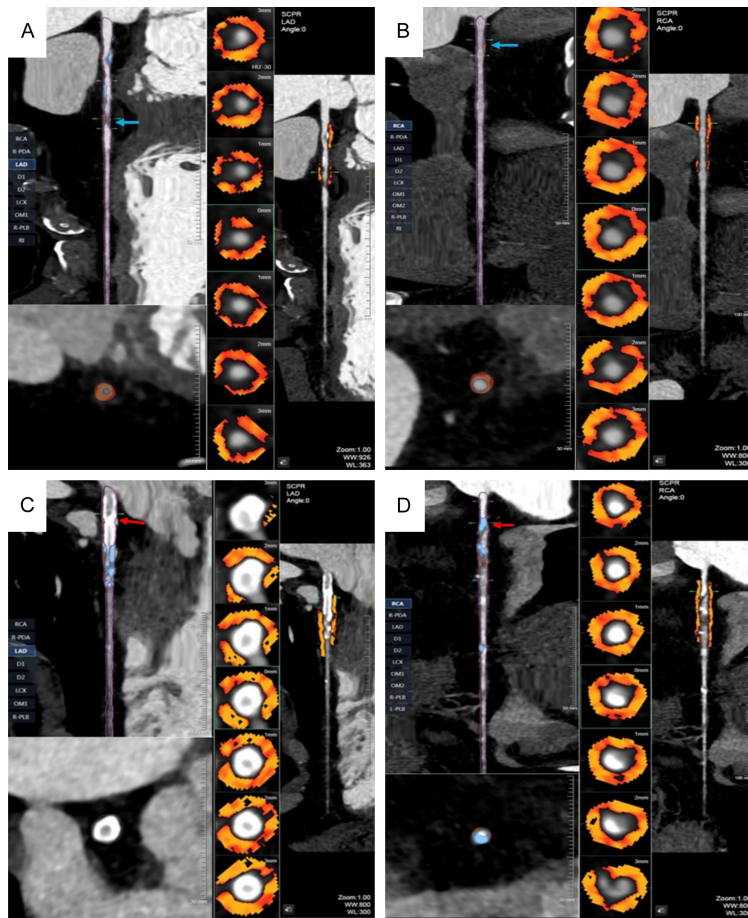


Figure 5. Typical CCTA images of SCAD and ACS. A. Axial LAD image of SCAD patient. B. Axial RCA image of SCAD patient. C. Axial LAD image of ACS patient. D. Axial RCA image of ACS patient. Note: Patients with SCAD (A, B) mainly presented calcified/fibrous plaques and low-attenuation PCAT with clear boundaries; patients with ACS (C, D) showed vulnerable plaques and significantly increased PCAT attenuation with blurred borders. The arrows denote the target segments for PCAT quantitative measurement. Abbreviations: CCTA, coronary computed tomography angiography; SCAD, stable coronary artery disease; ACS, acute coronary syndrome; LAD, left anterior descending artery; RCA, right coronary artery; PCAT, pericoronary adipose tissue.

leading to increased adipose CT density and elevated PCAT attenuation index. This pattern of inflammatory regulation has been jointly corroborated by the three cited studies. In our study, the LAD PCAT exhibited the optimal diagnostic performance, while the discriminative ability of the LCX was relatively weaker, which may be attributable to coronary anatomic characteristics: the LAD follows a relatively straight course with less interference from cardiac motion during measurement, whereas the LCX lies within the atrioventricular groove and undergoes substantial displacement throughout the cardiac cycle, rendering it more suscep-

tible to measurement bias. Moreover, the multi-vessel combined model constructed in this study achieved an AUC of 0.892, outperforming single-vessel parameters, a finding that aligns with the consensus of the three references, as combined assessment across multiple coronary arteries can comprehensively capture the global coronary inflammatory burden and thereby improve the accuracy of risk stratification in high-risk coronary artery disease.

Compared to conventional CCTA examination, PCAT parameter analysis has unique clinical advantages. Traditional CTA mainly relies on morphologic assessment of lumen stenosis and is not sensitive to early vascular wall lesions. This study found that alterations in the PCAT attenuation index may precede the development of coronary lumen stenosis, providing a novel non-invasive approach for risk identification in CAD (particularly early-stage lesions). Notably, the myocardial infarction prediction model established by integrating PCAT volume and attenuation index from LAD, LCX, and RCA achieved an AUC of 0.892, significantly higher than the predictive performance of any single PCAT parameter ($P < 0.05$).

These results not only confirm the significant value of multi-parameter integration in CAD risk stratification (particularly for myocardial infarction risk prediction) but also establish a data foundation for the future development of an intelligent CAD risk assessment system based on imaging parameters [21]. From a clinical translation perspective, PCAT analysis does not require additional scanning or contrast agent injection. It can be obtained through post-processing of conventional CTA images, which is highly operable and easy to implement. Our findings have important guiding significance for clinical practice. First, PCAT

Table 6. Independent cohort validation results of the key threshold

Metric	Validation cohort (n = 145)	Original cohort (n = 180)	Inter-cohort comparison result
Sensitivity (%)	72.5 (58/80)	74.0 (74/100)	-
Specificity (%)	69.2 (45/65)	71.3 (57/80)	-
Positive predictive value (%)	70.7 (58/82)	72.5 (74/102)	-
Negative predictive value (%)	71.0 (45/63)	72.8 (57/78)	-
AUC	0.703 (95% CI: 0.621-0.785)	0.715 (95% CI: 0.643-0.787)	Z = 0.382, P = 0.703
Kappa value with the original cohort	-	-	0.61 (P<0.001)

Note: AUC, area under curve; CI, confidence interval; SCAD, stable coronary artery disease; ACS, acute coronary syndrome; Sen, sensitivity; Spe, specificity.

parameters can serve as a powerful supplement for the diagnosis of SCAD, especially for patients with clinical suspicion but uncertain results from traditional examinations. Second, thresholds such as an LAD attenuation index >-68.5 HU or an RCA adipose tissue volume >1.07 cm³ can provide an objective basis for clinical decision-making. More importantly, the combined diagnostic model established in this article increased the diagnostic Acc of SCAD to 86.79%, markedly higher than the ~70% diagnostic accuracy of conventional CTA. These findings provide new imaging evidence for CAD (clinical classification of patients with diagnosed coronary artery disease) and personalized treatment decision-making. Compared to similar domestic and international studies, the innovation of this research is primarily reflected in three aspects: first, this study is the first to compare systematically the characteristics of PCAT between patients with stable coronary artery disease (SCAD; note: here SCAD specifically refers to stable coronary artery disease, not spontaneous coronary artery dissection) and those with acute coronary syndrome (ACS, including acute myocardial infarction); second, it clarified the hierarchical predictive value of PCAT parameters (volume and attenuation index) from different coronary branches, specifically the LAD, LCX, and RCA, in forecasting myocardial infarction; third, it developed a multi-parameter integrated prediction model for myocardial infarction (incorporating PCAT volume and attenuation index from multiple vessels). Although multiple international studies (such as the CRISP-CT study) have reported an association between PCAT attenuation index and cardiovascular event risk, they have not specifically evaluated its predictive efficacy for myocardial infarction risk in patients with SCAD. This study addresses a

gap highlighted by Cheng et al. [22]. A detailed case-control method will be employed in this paper to fill that gap. The standardized measurement methods used in this article provide reproducible technical specifications for subsequent research. It should be pointed out that this paper concentrates on the diagnostic capability of PCAT indices and does not compare them to functional indices, such as CT-FFR; this is also a constraint. CT-FFR is a function that reflects changes in coronary hemodynamics; therefore, one can know how much myocardial blood flow has been reduced by narrowing of the coronary arteries directly. PCAT index is mainly related to the inflammatory status of the coronary arteries. Both metrics provide complementary information for CAD diagnosis. In retrospect, had CT-FFR data been included, we could have further explored the correlation between PCAT parameters (such as the LAD attenuation index) and CT-FFR values (e.g., whether increased PCAT inflammation correlates with a reduced CT-FFR ≤0.8), thereby validating whether combining PCAT and CT-FFR could further enhance the diagnostic efficacy for SCAD. Future studies will incorporate CT-FFR measurements to establish a multidimensional diagnostic model integrating anatomic, inflammatory (PCAT-based), and functional (CT-FFR-based) indicators, which is expected to strengthen the evidence for the superior diagnostic value of PCAT parameters.

However, in this article, there may be selection bias; although PCAT measurement was performed using a standardized protocol, it may still be subject to partial volume effects; functional parameters such as coronary FFR were not included for comparative analysis. Future studies could further deepen the research in

the direction of multicenter large-sample validation, development of deep learning automatic measurement algorithms, and combination with hemodynamic measurements. It is particularly worth exploring whether dynamic changes in PCAT parameters can serve as biomarkers for evaluating remedy response and predicting the progression of the disease, which will open up new research directions for the management of CAD. From the perspective of pathophysiological mechanisms, the results of this article support the theoretical framework of “vascular-adipose tissue dialogue”. PCAT is no longer only an energy-storage device; now, it functions to release paracrine and perivascular factors that can promote the development of atherosclerosis. Inflammation of the blood vessels releases pro-inflammatory cytokines that can travel to the nearby fat tissue and promote the enlargement of adipocytes, infiltration by inflammatory cells, and extracellular matrix remodeling. As shown in the pictures, there is an increase in the extent of fat expansion and an increase in CT density values [23, 24]. Therefore, PCAT indices can be used to reflect pathologic changes in the vascular wall. In terms of clinical translation and application, this paper puts forward that PCAT evaluation should be added to the current diagnostic and treatment protocols for CAD. For patients undergoing CTA examination, routine measurement of PCAT parameters can be added; for people at medium or low risk, an abnormal PCAT may be one of the reasons why they need more aggressive intervention, and patients who have had revascularization surgery can use PCAT to determine if the treatment is effective. With the development of artificial intelligence, it is hoped that automatic PCAT parameter quantification and real-time analysis will be achieved to extend the clinical application of PCAT. In short, according to the above quantitative PCAT analysis based on CCTA, the attenuation index of the LAD and integrated diagnostic models for SCAD showed good predictive values. This represents a novel, non-invasive, and reproducible imaging biomarker that will help us better diagnose coronary artery disease (CAD). In the future, standardization and automation of this technology will be carried out, and many applications in the overall management of CAD will also be explored. Further correlation analysis shows that both the PCAT attenuation index

and volume are positively associated with the degree of coronary artery stenosis; among them, the LAD PCAT attenuation index shows a relatively strong correlation and is considered a marker of the extent of coronary artery disease in the inflammatory state of pericoronary adipose tissue. At the same time, univariate and multivariate logistic regressions also showed that after controlling for conventional cardiovascular risk factors, the LAD PCAT attenuation index was still an independent predictor of acute myocardial infarction; thus, it may offer new information for the risk model that does not contain typical indicators. Typical coronary CTA images show that the lower pericoronary fat density is lower in patients with SCAD and relatively higher in patients with ACS; thus, it can be seen that the PCAT parameter discrepancy is objective and reliable from an imaging perspective. Based on the independent cohort validation results, the cutoff value for the LAD PCAT attenuation index developed in this paper has good generalization and stability, and can provide an objective, non-invasive imaging basis for the early diagnosis of high-risk individuals with coronary artery disease [25]. According to the results of the new independent cohort validation in this study, the original threshold of LAD attenuation index >-68.5 HU is still consistently accurate across various centers (AUC = 0.703 vs. 0.715) and has a good level of agreement (Kappa = 0.61). This is to address the problem of single-center threshold determination. The validation process further supports the reliability of PCAT parameter thresholds as auxiliary diagnostic indicators for SCAD, providing more robust evidence for subsequent clinical application.

The present study had several inherent limitations. First, this was a single-center retrospective investigation, and all enrolled participants were patients with coronary artery disease confirmed by coronary angiography from Cangzhou and eastern Hebei Province. Owing to the influence of local dietary habits, lifestyle patterns, and the prevalence characteristics of hypertension and diabetes mellitus, the generalizability of our findings to other populations is subject to certain constraints. Although we implemented rigorous inclusion and exclusion criteria, achieved balanced baseline characteristics between the two groups, and employed standardized PCAT measurement

protocols, we cannot completely rule out potential interference of selection bias and unmeasured confounding factors. Furthermore, this study only explored the value of PCAT parameters in differentiating stable coronary artery disease from acute coronary syndrome, without incorporating coronary hemodynamic functional indices such as CT-FFR for comparative analysis. Despite the fact that post-hoc power analysis and an independent external validation cohort confirmed the statistical robustness of the diagnostic threshold derived in this study, long-term follow-up data are lacking, which precluded verification of the sustained predictive performance of PCAT parameters over time. Future multicenter, large-scale prospective studies encompassing diverse geographic populations, incorporating functional parameters such as CT-FFR, and extending the follow-up duration are warranted to validate the clinical use of PCAT parameters in the classification, risk stratification, and prognostic evaluation of coronary artery disease.

Conclusion

The volume and attenuation index of pericoronary adipose tissue (PCAT) in coronary CT images were effective indicators to distinguish between stable coronary artery disease (SCAD) and acute coronary syndrome (ACS) in patients with known coronary heart disease. The degree of coronary artery stenosis was positively correlated with the PCAT attenuation level and volume. After adjusting for conventional cardiovascular risk factors, the LAD PCAT attenuation index remained an independent discriminator between SCAD and ACS. Representative coronary CT angiography images show decreased attenuation of pericoronary fat in people with SCAD, and marked increases in pericoronary fat attenuation were observed in those with ACS. Imaging evidence suggested that PCAT can reflect the condition of coronary artery inflammation. In addition, the optimal threshold for the LAD PCAT attenuation index that was determined in this study also performed well for the separate validation group and was consistent with the initial findings, so current results are reliable and consistent. The diagnostic model combining multi-vessel PCAT parameters achieves optimal discriminatory efficacy,

providing a non-invasive, high-precision imaging basis for clinical classification, early risk stratification, personalized assessment, and clinical decision-making in patients with coronary artery disease.

Disclosure of conflict of interest

None.

Address correspondence to: Hong-Ye Zuo, Department of CT Diagnostic, Cangzhou Central Hospital, No. 16, Xinhua West Road, Yunhe District, Cangzhou 061000, Hebei, China. Tel: +86-0317-96789; E-mail: hongyezuo@163.com

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