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Association of Pericoronary Adipose Tissue Attenuation With High-Risk Coronary Plaque Features on Coronary CT Angiography: A Cross-Sectional Study

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Abstract

Background

Pericoronary adipose tissue attenuation (PCATa) is an imaging marker of the coronary inflammatory environment, but its relationship with high-risk coronary plaque (HRP) features remains incompletely characterized. We evaluated the association between PCATa and HRP on coronary CT angiography (CCTA).

Methods

This retrospective cross-sectional study included 1,482 adults who underwent clinically indicated CCTA and had diagnostic-quality examinations. Four predefined high-risk plaque features-low-attenuation plaque, positive remodeling, spotty calcification, and napkin-ring sign-were recorded at the patient level. The total number of recorded features was calculated for each participant (range, 0-4). Participants with two or more recorded features were classified as having a higher patient-level HRP feature burden. We measured PCATa in the proximal right coronary artery (RCA). We performed Spearman correlation, multivariable logistic regression, and receiver operating characteristic (ROC) analysis.

Results

A higher patient-level HRP feature burden was observed in 325/1,482 participants (21.9%). Mean PCATa was −79.22 ± 6.25 HU and was higher (less negative) in participants with a higher patient-level HRP feature burden than in those with a lower patient-level feature burden (−73.32 ± 5.37 vs −80.88 ± 5.42 HU; P<0.001). The total number of patient-level HRP features was positively correlated with PCATa (ρ=0.498; P<0.001). Each 1-HU increase in PCATa was associated with higher adjusted odds of a higher patient-level HRP feature burden (adjusted OR, 1.193; 95% CI, 1.132-1.257; P<0.001). PCATa discriminated between the two study-defined patient-level feature-burden categories, with an AUC of 0.839 (95% CI, 0.816-0.862). An exploratory threshold of −77.50 HU yielded 79.7% sensitivity and 73.5% specificity. Inter-reader agreement was high (κ=0.964; ICC=0.961).

Conclusion

Higher PCATa was associated with a greater patient-level HRP feature burden and demonstrated good discrimination for the study-defined higher feature-burden category. These findings suggest that PCATa may provide complementary information regarding coronary plaque characteristics. However, the study does not establish plaque-specific co-localization of high-risk features or Coronary Artery Disease - Reporting and Data System (CAD-RADS 2.0) same-plaque HRP classification.

Categories: Radiology, Cardiology
Keywords: adipose tissue, angiography, cardiovascular morbidity, coronary artery disease, high-risk coronary plaque

Introduction

Coronary artery disease (CAD) is a major cause of cardiovascular morbidity and mortality around the world. It is a common cause of acute coronary events, which occur when atherosclerotic plaques become unstable and rupture or erode [1]. Importantly, luminal stenosis alone does not capture plaque vulnerability [2]. Coronary computed tomography angiography (CCTA) can noninvasively assess coronary anatomy and morphology and detect high-risk plaque (HRP) features such as low-attenuation plaque, positive remodeling, spotty calcification, and the napkin-ring sign [3]. These characteristics have been linked to adverse cardiovascular events and offer data in addition to traditional stenosis severity assessment [4].

Pericoronary adipose tissue (PCAT) is anatomically and biologically connected to the coronary arterial wall and can react to local vascular inflammation [5]. Inflammatory changes of the coronary vessel can trigger molecular and structural changes of the adjacent pericoronary adipose tissue that lead to measurable changes in CT attenuation [6]. Thus, pericoronary adipose tissue attenuation (PCATa) is a new, non-invasive imaging biomarker of the pericoronary inflammatory milieu [7] and has been investigated as a marker of coronary vascular inflammation [8].

While these associations have been observed with elevated PCATa, the relationship between elevated PCATa and specific structural characteristics of plaque vulnerability seen on CCTA has not been fully characterized [9,10]. In particular, while some individual HRP characteristics have been linked to PCATa, the strength of these relationships varies by plaque features, and whether they are independent of traditional cardiovascular risk factors or coronary disease burden remains incompletely understood [9,11]. Clarifying this relationship is clinically relevant because PCATa and plaque morphology may provide complementary imaging information: PCATa may reflect the local inflammatory milieu, whereas CCTA-defined HRP features characterize structural and compositional manifestations of plaque vulnerability [8,11].

This study's novelty is the integrated assessment of quantitative PCATa and CCTA-derived HRP characteristics, allowing examination of the association between the local pericoronary inflammatory environment and specific morphological markers of plaque vulnerability. This study examines whether PCATa is associated with the presence of an HRP phenotype and the occurrence of individual HRP features.

This cross-sectional study assessed the association between PCATa and CCTA-derived high-risk coronary plaque characteristics. Specifically, we investigated the relationship between PCATa and four individual HRP features: low-attenuation plaque, positive remodeling, spotty calcification, and the napkin-ring sign. We also evaluated its association with the study-defined higher patient-level HRP feature-burden category, defined as the presence of two or more recorded features across the overall CCTA examination. Furthermore, we assessed whether this association remained evident after adjustment for relevant clinical and coronary characteristics. We hypothesized that higher PCATa would be associated with a greater prevalence of individual HRP features and higher patient-level HRP feature burden.

Materials And Methods

Study design and setting

This retrospective cross-sectional observational study was conducted at the study site and included consecutive adults who underwent clinically indicated CCTA between October 2025 and July 2026. Clinical data were retrieved retrospectively from institutional records, coronary plaque characteristics were assessed from archived CCTA data, and PCATa was determined from archived CCTA data. This study did not include any further imaging, lab testing, electrocardiography, or clinical follow-up.

Sample size

We initially screened 1,500 consecutive patients who underwent clinically indicated CCTA during the study period. The final analytic cohort comprised 1,482 patients after excluding 18 patients with non-diagnostic or poor-quality CCTA images. Of these, 325 (21.9%) met the study-defined criterion for a higher patient-level HRP feature burden, defined as the presence of two or more recorded HRP features across the overall CCTA examination. The remaining 1,157 (78.1%) had a lower patient-level feature burden, defined as fewer than two recorded features. The final sample size was thus not based on prospective recruitment to a pre-defined sample size, but on the number of eligible CCTA examinations available during the pre-defined study period.

Sampling technique

The technique used was consecutive sampling. We considered all patients with clinically indicated CCTA during the study period who met the inclusion criteria, in chronological order. We excluded patients only if the CCTA examination was non-diagnostic or image quality was poor for reliable analysis of coronary plaque characteristics and PCATa. Consecutive sampling was used to reduce investigator-driven selection of eligible examinations within the predefined study period. However, because the cohort consisted of patients referred for clinically indicated CCTA at a single center, external generalizability may be limited.

Study population and patient selection

We selected patients from the institutional radiology or cardiac CT database based on the study period. We

included adults ⩾18 years of age with a clinically indicated CCTA. Inclusion also required quality CCTA datasets to allow evaluation of at least one coronary artery, availability of the original CCTA images for quantitative PCATa assessment, and adequate clinical information for the prespecified covariates.

We excluded examinations with insufficient image quality for reliable assessment of coronary plaque and/or PCATa, such as high motion artifact, poor coronary artery opacification, or other significant artifacts that prevented evaluation of coronary plaque. Only patients with prior CABG or extensive coronary stenting that precluded a standard evaluation of the native coronary arteries or the prespecified measurement segment were excluded. We also excluded examinations that lacked adequate imaging information for plaque assessment or PCAT. After excluding 18 studies, we included 1,482 patients in the final study population. According to the prespecified patient-level feature-burden definition, 325 participants (21.9%) had a higher feature burden (two or more recorded HRP features), whereas 1,157 (78.1%) had a lower feature burden (fewer than two recorded features). Figure *1* summarises the selection of the final study cohort.

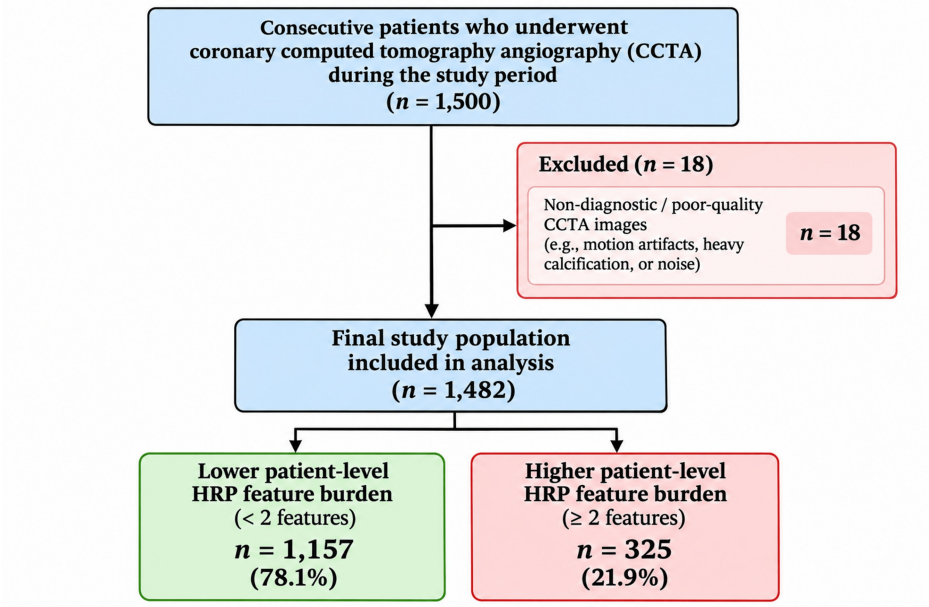


FIGURE 1: Flowchart of patient selection and study cohort formation

Image credit: Created by the authors using Python version 3.14.7 (Python Software Foundation, Fredericksburg, VA, US) within the JupyterLab environment (version 4.6.4; (Computer software). GitHub. https://github.com/jupyterlab/jupyterlab).

Clinical and cardiovascular variables

We abstracted demographic and clinical variables from existing medical records using a standardized data-collection form. Variables included age, sex, body mass index (BMI), hypertension, diabetes mellitus, dyslipidemia, smoking status, family history of coronary artery disease, previous coronary artery disease or myocardial infarction, and statin therapy. For the association between PCATa and HRP, we considered the following variables as potential confounders.

Smoking status was categorized as never, former, or current smoking. We obtained cardiovascular comorbidities and medications from documentation in the medical record at the index CCTA or before the index CCTA examination.

CCTA acquisition protocol

CCTA examinations were performed at the Radiology Department of Farooq Hospital, Lahore, Pakistan, using an Aquilion ONE PRISM CT scanner with prospective ECG-triggering (Canon Medical Systems Corporation, Ōtawara, Japan). Tube voltage ranged from 100 to 120 kVp, depending on the patient's body habitus, and was controlled by automatic exposure control with tube current. Iodinated contrast material (ICM) was administered intravenously at approximately 50-80 mL at a 5-6 mL/s injection rate, followed by the institutional saline flush protocol, which contained approximately 350-370 mgI/mL of ICM.

Pre-image acquisition heart-rate optimization was performed. Beta-blocker therapy was used as needed to reach a target heart rate of <60-65 beats/min, if no contraindications existed. To support coronary vasodilation and coronary lumen visualization, we used sublingual nitroglycerin, unless contraindicated,

before image acquisition.

We used prospective ECG triggering to acquire images, and centered the main reconstruction window at approximately 70-80% of the R-R interval for patients with well-controlled heart rates. We reconstructed images at approximately 0.5 mm slice thickness using the scanner's vendor-specific cardiac reconstruction algorithm, with iterative reconstruction applied where available. These datasets were then transferred to the dedicated CT workstation for coronary plaque analysis, where they were used to characterize and quantitatively assess PCATa. The same institutional CCTA acquisition and image reconstruction protocol was used throughout the study period. Tube voltage was adjusted according to patient body habitus within the specified range. However, individual patient-level acquisition and reconstruction parameters were not independently extracted from Digital Imaging and Communications in Medicine (DICOM) metadata or statistically compared between the lower and higher patient-level HRP feature-burden groups.

Coronary artery and plaque assessment

We evaluated the left main coronary artery, left anterior descending artery, left circumflex artery, and right coronary artery for coronary atherosclerotic plaque. We noted the maximum percent diameter stenosis and classified plaque composition as calcified, noncalcified, or mixed.

Two experienced readers trained in cardiovascular CT interpretation independently assessed coronary plaque characteristics using multiplanar and cross-sectional reconstructions of the original CCTA datasets.

Assessment of high-risk coronary plaque features

Four predefined HRP features were assessed: low-attenuation plaque, positive remodeling, spotty calcification, and napkin-ring sign. We evaluated each feature on multiplanar and cross-sectional CCTA images.

Low-attenuation plaque was defined as non-calcified plaque with internal attenuation <30 HU. Positive remodeling was defined as a remodeling index >1.1, calculated as the outer vessel diameter at the plaque site divided by the mean outer vessel diameter of the proximal and distal reference segments. According to Coronary Artery Disease-Reporting and Data System (CAD-RADS) 2.0, focal/punctate calcification within a coronary plaque was defined as spotty calcification.

The napkin-ring sign was described as a central area of low-attenuation plaque in contact with the lumen with a higher-attenuation peripheral rim. The definitions of low attenuation and remodeling are (specifically) defined in CAD-RADS 2.0.

Definition of HRP

Four predefined high-risk coronary plaque features were assessed: low-attenuation plaque, positive remodeling, spotty calcification, and napkin-ring sign [12]. We recorded each feature as present or absent at the patient level based on the overall CCTA examination. We calculated the total number of recorded features for each patient, yielding a score ranging from zero to four. For the binary analysis, patients with two or more recorded features were classified as having a higher patient-level HRP feature burden. In contrast, those with fewer than two features were classified as having a lower feature burden. Because plaque-specific co-localization was not available in the analytical dataset, this classification represents the cumulative patient-level burden of recorded features. It does not establish that two or more features were present within the same individual plaque.

Quantification of PCATa

PCATa was measured by AutoPlaque v2.5 (Cedars-Sinai Medical Center, Los Angeles, USA). We chose the proximal right coronary artery (RCA) as the standard coronary segment for PCAT evaluation. The analysis segment started 10 mm from the RCA ostium and ended 50 mm distal to the ostium, excluding the first 10 mm adjacent to the coronary ostium. PCAT was defined as adipose tissue surrounding the coronary artery with CT attenuation values between −190 and −30 Hounsfield units (HU). The pericoronary adipose tissue compartment was defined as the three-dimensional space extending up to 3 mm radially outward from the outer coronary vessel wall, corresponding approximately to the typical diameter of the proximal RCA. The mean PCATa was determined by averaging all voxels in the predefined PCAT compartment that had an attenuation between −190 and −30 HU. The same protocol for acquisition and analysis was applied to all participants for all PCAT measurements performed on the original CCTA datasets. Two experienced readers independently quantified PCATa while blinded to clinical data and plaque classification. We used the average of the two readers' measurements for the primary analysis.

Figure *2* provides a schematic illustration of the proximal RCA measurement segment and the PCAT compartment used for PCATa assessment.

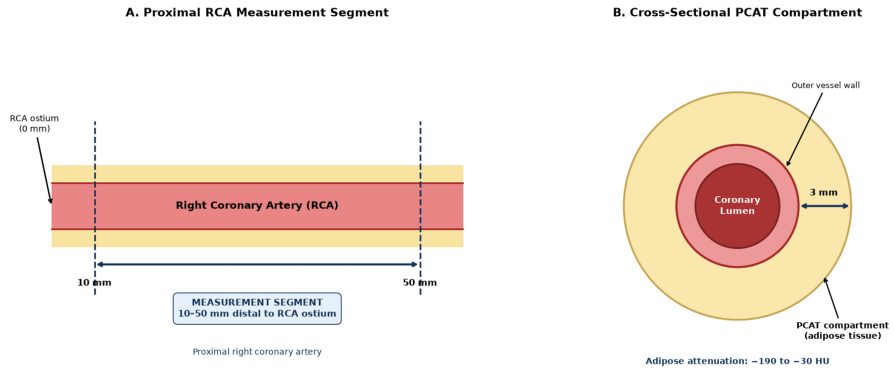


FIGURE 2: Schematic illustration of pericoronary adipose tissue attenuation (PCATa) measurement in the proximal right coronary artery

Panel A illustrates the proximal right coronary artery (RCA) measurement segment extending from 10 to 50 mm distal to the coronary ostium. Panel B demonstrates the pericoronary adipose tissue compartment extending up to 3 mm radially from the outer vessel wall. Adipose tissue voxels were identified using CT attenuation thresholds of −190 to −30 HU. This is a conceptual schematic based on the study measurement protocol and is not an actual coronary computed tomography angiography (CCTA) image.

Image credit: Created by the authors using Python version 3.14.7 (Python Software Foundation, Fredericksburg, VA, US) within the JupyterLab environment (version 4.6.4; (Computer software). GitHub. https://github.com/jupyterlab/jupyterlab).

This illustration is intended to clarify the measurement protocol and does not represent an actual patient-derived CCTA image.

Image interpretation and reader blinding

Two experienced readers trained in cardiovascular CT interpretation, coronary plaque characterization, and PCATa assessment independently reviewed CCTA images. Both readers independently reviewed the CCTA examinations, and their evaluations were blinded to one another and to the pertinent clinical and outcome data. The assessment included PCATa quantification and evaluation of predetermined HRP characteristics (low-attenuation plaque, positive remodeling, spotty calcification, and napkin-ring sign).

We minimized information bias through independent image interpretation and by assessing clinical variables and the primary study outcome. We resolved discrepancies between the two readers by consensus.

Assessment of measurement reproducibility

We used the intraclass correlation coefficient (ICC) to assess interobserver reproducibility for quantitative PCATa, and Cohen's κ statistic for categorical HRP features. We reported the ICC and κ coefficients with their corresponding 95% confidence intervals (CIs).

Data management and quality control

We collected clinic and imaging data in a standardized data-collection database. Each participant received an individual study identification number, and identifying information was removed from the analytical dataset. The database included demographic characteristics, cardiovascular risk factors, coronary plaque distribution and stenosis, plaque composition, individual HRP characteristics, total number of patient-level HRP features, HRP classification, and PCATa.

We first examined the data for completeness and internal consistency before statistical analysis. All variables included in the primary analysis were complete; therefore, no imputation was required. We measured imaging variables from the CCTA datasets, not from radiology reports alone, because quantitative PCATa is based on measurements of the actual images.

Statistical analysis

We performed statistical analyses using IBM SPSS Statistics for Windows, Version 26 (Released 2019; IBM Corp., Armonk, New York, United States). We presented data as mean ± SD for continuous variables, median (IQR) for continuous variables, and frequencies and percentages for categorical variables. We analyzed group data with an independent-samples t-test, Mann-Whitney U test, or Pearson χ^2 test, as appropriate. Multivariable binary logistic regression was used to estimate the association between PCATa and the study-

defined higher patient-level HRP feature-burden category. Two multivariable logistic regression models were constructed to evaluate the association between PCATa and higher patient-level HRP feature burden. Model 1 included PCATa as the primary predictor and was adjusted for age, sex, BMI, hypertension, diabetes mellitus, dyslipidemia, smoking status, and statin use. These covariates were selected based on their established clinical relevance as potential cardiovascular risk factors and possible confounders of the association between PCATa and HRP.

Model 2 included all variables from Model 1, with maximum coronary stenosis added to account for the potential influence of coronary disease severity on HRP. Participants classified as having no coronary plaque (n=742) had a maximum coronary stenosis value of 0% in the analytical dataset. These observations were retained in Model 2 rather than treated as missing. Overall, maximum stenosis values were available for all 1,482 participants. This additional adjustment was intended to assess whether the association between PCATa and HRP persisted after accounting for the degree of coronary stenosis. Adjusted odds ratios (aORs) with 95% CIs were reported for both models. PCATa was entered as a continuous predictor per 1-HU increase. We determined correlations between the total number of patient-level HRP features and PCATa using Spearman's correlation. Multicollinearity among model covariates was assessed using variance inflation factors and tolerance statistics.

We used Cohen's κ and ICC to measure interobserver agreement. We evaluated the discriminative performance of PCATa for distinguishing between the study-defined lower and higher patient-level HRP feature-burden categories using receiver operating characteristic (ROC) analysis. For the primary binary logistic regression analyses, the patient-level HRP feature-burden classification recorded by Reader 1 was used as the dependent variable. Patients with two or more recorded HRP features were classified as having a higher patient-level feature burden, while those with fewer than two features were classified as having a lower feature burden. Reader 2's assessments were used to evaluate inter-reader agreement. A two-sided $P<0.05$ was considered statistically significant.

Ethics and confidentiality

The study was conducted in accordance with the Declaration of Helsinki and applicable institutional requirements for research involving human participants. This retrospective cross-sectional study used previously collected clinical and imaging data. The protocol for this study was approved by the institutional Research Ethics Committee/Institutional Review Board of Allama Iqbal Medical College, Lahore, Pakistan (IRB approval number: ERB761/ 5/08-10-25). The IRB granted a waiver of informed consent because the study involved retrospective analysis of previously collected, de-identified clinical and imaging data.

Results

As shown in Table *1*, out of 1,482 participants, 753 (50.8%) were male subjects, with a mean age of 57.02 ± 11.11 years and a mean BMI of 27.20 ± 3.83 kg/m².

Characteristic	N = 1,482
Demographic characteristics	
Age, years	57.02 ± 11.11
Male sex	753 (50.8%)
Body mass index, kg/m²	27.20 ± 3.83
Clinical characteristics	
Hypertension	612 (41.3%)
Diabetes mellitus	228 (15.4%)
Dyslipidemia	592 (39.9%)
Smoking status	
Never	779 (52.6%)
Former	381 (25.7%)
Current	322 (21.7%)
Family history of CAD	494 (33.3%)
Previous CAD/MI	188 (12.7%)
Statin use	473 (31.9%)

CCTA characteristics	
Maximum coronary stenosis, %	0 (0–24)
Left main plaque	118 (8.0%)
LAD plaque	435 (29.4%)
LCX plaque	363 (24.5%)
RCA plaque	398 (26.9%)
Plaque type	
No plaque	742 (50.1%)
Calcified	149 (10.1%)
Non-calcified	408 (27.5%)
Mixed	183 (12.3%)
PCAT characteristics	
PCAT attenuation, reader 1, HU	−79.20 ± 6.29
PCAT attenuation, reader 2, HU	−79.24 ± 6.33
Mean PCAT attenuation, HU	−79.22 ± 6.25
High-risk plaque characteristics (Reader 1)	
Low-attenuation plaque	322 (21.7%)
Positive remodeling	270 (18.2%)
Spotty calcification	241 (16.3%)
Napkin-ring sign	157 (10.6%)
Total number of patient-level HRP features	0.67 ± 1.19
Higher patient-level HRP feature burden (≥2 features)	325 (21.9%)

TABLE 1: Baseline clinical, demographic, CCTA, and PCAT characteristics of the study population (N=1,482)

Data are presented as mean ± SD, median (IQR), or n (%), as appropriate. CAD: coronary artery disease; MI: myocardial infarction; LAD: left anterior descending; LCX: left circumflex; RCA: right coronary artery; PCAT: pericoronary adipose tissue; HU: Hounsfield units.

Hypertension was present in 612 (41.3%) participants, dyslipidemia in 592 (39.9%), and diabetes in 228 (15.4%). Non-calcified plaque was the most common plaque type, occurring in 408 (27.5%) participants, while mean PCATa was −79.22 ± 6.25 HU. Overall, 325 participants (21.9%) had a higher patient-level HRP feature burden, defined as two or more recorded HRP features across the overall CCTA examination.

As shown in Figure 3, among the 1,482 participants, 742 (50.1%) had no coronary plaque, while 408 (27.5%) had non-calcified plaque, 183 (12.3%) had mixed plaque, and 149 (10.1%) had calcified plaque.

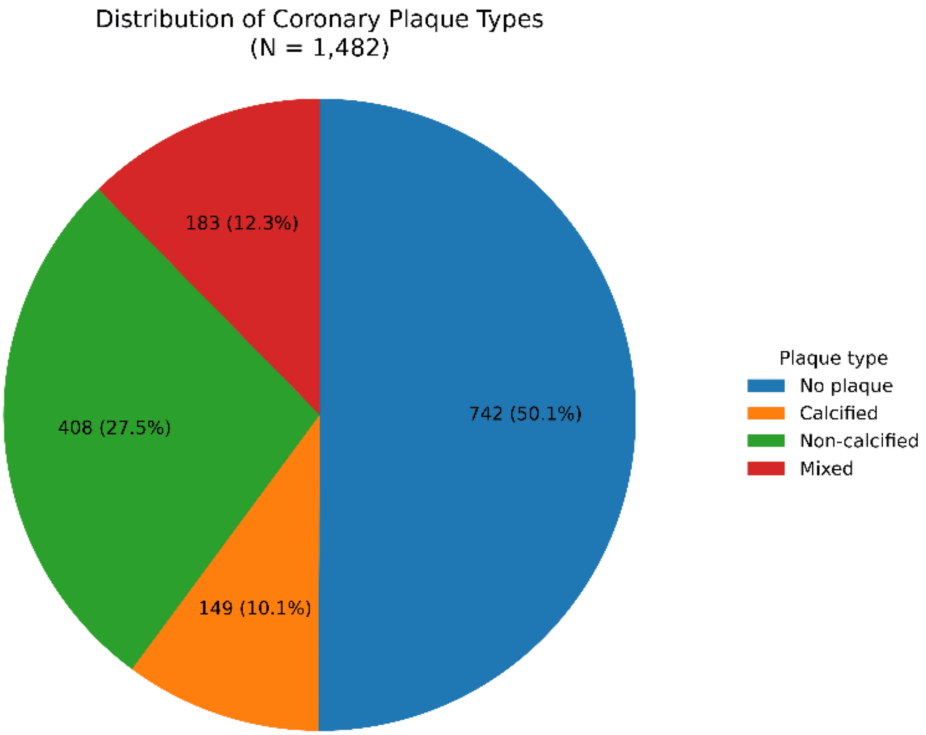


FIGURE 3: Distribution of coronary plaque types in the study population (N = 1,482)

Values within the pie segments represent the number of participants and the corresponding percentage.

Image credit: Created by the authors using Python version 3.14.7 (Python Software Foundation, Fredericksburg, VA, US) within the JupyterLab environment (version 4.6.4; (Computer software). GitHub. https://github.com/jupyterlab/jupyterlab).

Among participants with coronary plaque, non-calcified plaque was the most common type. Mean PCATa demonstrated a moderate positive correlation with the total number of recorded patient-level HRP features (Spearman's ρ=0.498, P<0.001) among all 1,482 participants.

This indicates that higher (less-negative) PCATa was associated with a greater cumulative number of recorded features at the patient level. This was an unadjusted correlation analysis.

Mean PCATa increased progressively from −81.07 HU among participants with zero recorded patient-level HRP features to −69.14 HU among those with four recorded features, as shown in Figure *4*.

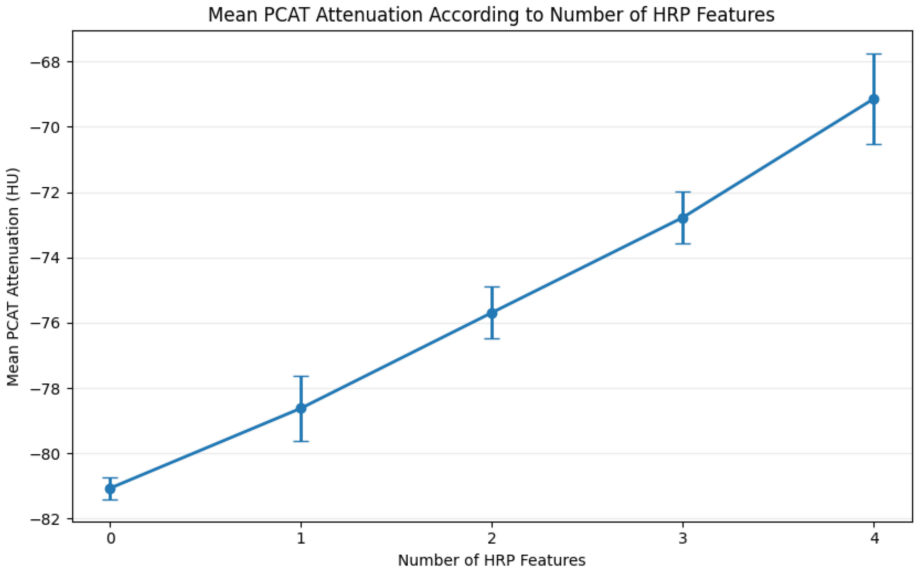


FIGURE 4: Mean PCATa according to the number of high-risk plaque features

Error bars represent 95% confidence intervals. PCATa: pericoronary adipose tissue attenuation.

Image credit: Created by the authors using Python version 3.14.7 (Python Software Foundation, Fredericksburg, VA, US) within the JupyterLab environment (version 4.6.4; (Computer software). GitHub. https://github.com/jupyterlab/jupyterlab).

This pattern indicates a positive relationship between PCATa and the cumulative number of patient-level features, with higher (less-negative) PCATa values observed as the number of recorded features increased.

As shown in Table *2*, participants with a higher patient-level HRP feature burden (n=325, 21.9%) were older than those with a lower feature burden (n=1,157, 78.1%) (63.96 ± 10.36 vs. 55.07 ± 10.53 years, P<0.001) and had a slightly higher BMI (27.88 ± 3.75 vs. 27.01 ± 3.83 kg/m², P<0.001).

Characteristic	Lower feature burden (n = 1,157)	Higher feature burden (n = 325)	P value
Age, years	55.07 ± 10.53	63.96 ± 10.36	<0.001
Male sex, n (%)	564 (48.7)	189 (58.2)	0.003
BMI, kg/m²	27.01 ± 3.83	27.88 ± 3.75	<0.001
Hypertension, n (%)	406 (35.1)	206 (63.4)	<0.001
Diabetes mellitus, n (%)	140 (12.1)	88 (27.1)	<0.001
Dyslipidemia, n (%)	397 (34.3)	195 (60.0)	<0.001
Smoking status, n (%)	-	-	0.001
Never	623 (53.8)	156 (48.0)	—
Former	307 (26.5)	74 (22.8)	—
Current	227 (19.6)	95 (29.2)	—
Family history of CAD, n (%)	393 (34.0)	101 (31.1)	0.329
Previous CAD/MI, n (%)	106 (9.2)	82 (25.2)	<0.001
Statin use, n (%)	335 (29.0)	138 (42.5)	<0.001
Maximum stenosis, %, median (IQR)	0 (11)	44 (20)	<0.001
LM plaque, n (%)	61 (5.3)	57 (17.5)	<0.001
LAD plaque, n (%)	195 (16.9)	240 (73.8)	<0.001
LCX plaque, n (%)	159 (13.7)	204 (62.8)	<0.001
RCA plaque, n (%)	187 (16.2)	211 (64.9)	<0.001
Plaque type, n (%)	-	-	<0.001
No plaque	742 (64.1)	0 (0.0)	—
Calcified	45 (3.9)	104 (32.0)	—
Non-calcified	322 (27.8)	86 (26.5)	—
Mixed	48 (4.1)	135 (41.5)	—
Mean PCAT attenuation, HU	−80.88 ± 5.42	−73.32 ± 5.37	<0.001

TABLE 2: Clinical, demographic, and CCTA characteristics according to patient-level HRP feature-burden category

Note: Data are presented as mean ± standard deviation, median (interquartile range), or number (percentage), as appropriate. Independent-samples t-tests were used for continuous variables with approximately normal distributions, the Mann–Whitney U test for maximum coronary stenosis, and chi-square tests for categorical variables. Maximum coronary stenosis data were available for all participants (lower-burden group: n=1,157; higher-burden group: n=325; total N=1,482). P-values for smoking status and plaque type represent overall comparisons.

HRP: high-risk plaque; CAD: coronary artery disease; MI: myocardial infarction; BMI: body mass index; LM: left main; LAD: left anterior descending; LCX: left circumflex; RCA: right coronary artery; PCAT: pericoronary adipose tissue; HU: Hounsfield units.

Participants in the higher feature-burden category also had higher prevalences of hypertension, diabetes mellitus, and dyslipidemia, as well as previous CAD/MI and statin use. Current smoking was more common in this group, and sex distribution also differed between groups, whereas family history of CAD did not differ significantly. The higher feature-burden category had a higher frequency of plaque in the left median (LM); left anterior descending (LAD), left circumflex (LCX); right coronary artery (RCA), and maximum coronary stenosis was significantly greater in this group (median, 44%; IQR, 20) than in the lower feature-burden group (median, 0%; IQR, 11; P<0.001). Mean PCATa was significantly higher (less negative) in participants with a higher feature burden than in those with a lower feature burden (−73.32 ± 5.37 vs. −80.88 ± 5.42 HU, P<0.001). Calcified and mixed plaques were more frequent in the higher feature-burden category, whereas participants with no plaque were classified in the lower feature-burden category.

Higher PCATa remained independently associated with higher patient-level HRP feature burden in both models (Model 1: adjusted OR=1.240, 95% CI: 1.200-1.282, P<0.001; Model 2: adjusted OR=1.193, 95% CI: 1.132-1.257, P<0.001). In Model 1, age, male sex, hypertension, and dyslipidemia were also significant predictors, whereas in Model 2, BMI and maximum coronary stenosis were significant. Both models were statistically significant, with Nagelkerke R^2 values of 0.429 and 0.808, respectively. Model 1 showed adequate fit (Hosmer-Lemeshow p=0.269), while Model 2 indicated imperfect calibration (p=0.011) (Table 3).

Predictor	Model 1 Adjusted OR	95% CI	P value	Model 2 Adjusted OR	95% CI	P value
PCAT attenuation, per 1-HU increase	1.240	1.200–1.282	<0.001	1.193	1.132–1.257	<0.001
Age, per year	1.042	1.025–1.058	<0.001	0.984	0.957–1.011	0.233
Male sex	1.431	1.055–1.941	0.021	1.015	0.615–1.676	0.954
BMI, per kg/m²	1.010	0.970–1.052	0.620	1.080	1.010–1.155	0.025
Hypertension	1.546	1.128–2.121	0.007	1.096	0.660–1.819	0.723
Diabetes mellitus	1.465	0.991–2.164	0.055	0.672	0.351–1.289	0.232
Dyslipidemia	1.503	1.088–2.075	0.013	1.107	0.658–1.862	0.703
Former smoker vs. never smoker	1.073	0.738–1.560	0.711	0.959	0.525–1.753	0.893
Current smoker vs. never smoker	1.398	0.965–2.025	0.077	1.624	0.882–2.990	0.119
Statin use	1.020	0.734–1.416	0.908	0.839	0.486–1.448	0.529
Maximum stenosis, per 1% increase	—	—	—	1.168	1.145–1.192	<0.001

TABLE 3: Association of PCATa with high-risk plaque: multivariable logistic regression

Model 1 was adjusted for age, sex, BMI, hypertension, diabetes mellitus, dyslipidemia, smoking status, and statin use. Model 2 included all Model 1 covariates with additional adjustment for maximum coronary stenosis. Reference categories were female sex, no hypertension, no diabetes mellitus, no dyslipidemia, never smoking, and no statin use. PCATa was analyzed per 1-HU increase; because PCAT values are negative, an increase represents a shift toward less-negative attenuation.

HRP: high-risk plaque; PCATa: pericoronary adipose tissue attenuation; HU: Hounsfield units; BMI: body mass index; OR: odds ratio; CI: confidence interval.

Model 1 and Model 2 were fitted using the same analytic cohort. Maximum coronary stenosis was coded as 0% for participants with no detectable coronary plaque. The final analytic sample for Model 2 was N = 1,482.

Mean PCATa demonstrated good discriminatory ability for identifying the study-defined higher patient-level HRP feature-burden category, with an AUC of 0.839 (95% CI, 0.816-0.862). At an exploratory threshold of −77.50 HU, sensitivity was 79.7% and specificity was 73.5% (Figure 5).

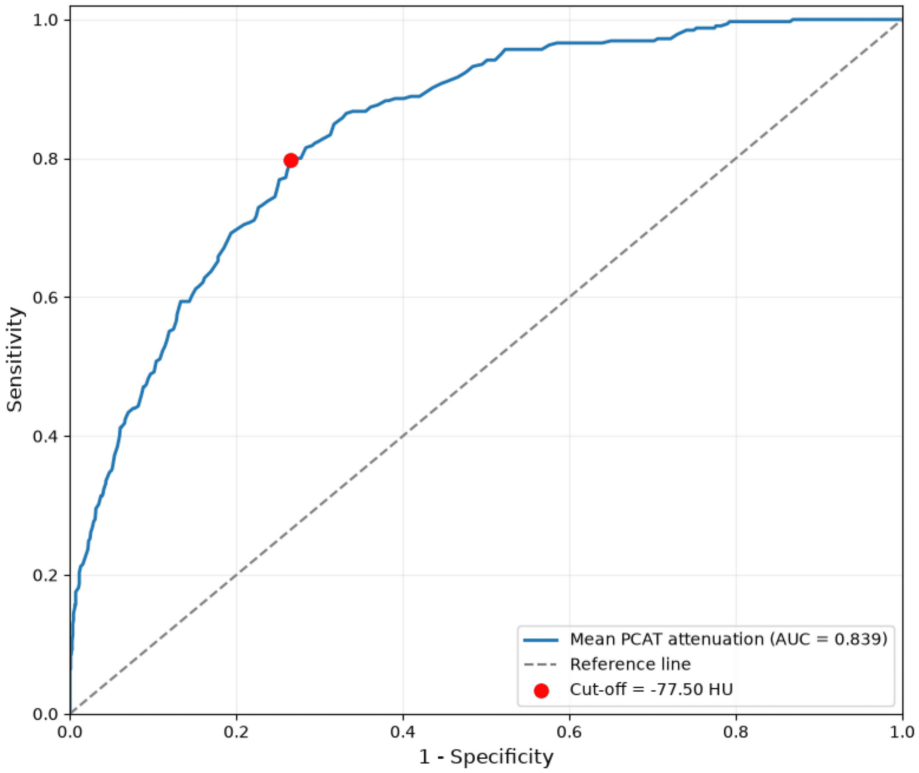


FIGURE 5: Receiver operating characteristic curve of mean PCATa for discrimination of the study-defined higher patient-level HRP feature-burden category

The area under the ROC curve (AUC) was 0.839 (95% CI, 0.816–0.862). An exploratory pericoronary adipose tissue attenuation (PCATa) threshold of −77.50 HU yielded 79.7% sensitivity (259/325) and 73.5% specificity (850/1,157).

HRP: high-risk plaque; HU: Hounsfield units.

Image credit: Created by the authors using Python version 3.14.7 (Python Software Foundation, Fredericksburg, VA, US) within the JupyterLab environment (version 4.6.4; (Computer software). GitHub. https://github.com/jupyterlab/jupyterlab).

As shown in Table 4, mean PCATa was significantly higher in participants with each HRP feature compared with those without the feature (all P<0.001).

High-risk plaque feature	Absent, mean ± SD (HU)	Present, mean ± SD (HU)	P value
Low-attenuation plaque	−80.756 ± 5.511	−73.674 ± 5.575	<0.001
Positive remodeling	−80.587 ± 5.507	−73.073 ± 5.666	<0.001
Spotty calcification	−80.398 ± 5.669	−73.141 ± 5.531	<0.001
Napkin-ring sign	−80.010 ± 5.784	−72.529 ± 6.035	<0.001

TABLE 4: Mean PCATa according to individual patient-level high-risk plaque features

Values are mean ± SD. P values were derived from independent-samples t tests. A two-sided P value <0.05 was considered statistically significant.
PCATa: pericoronary adipose tissue attenuation; HU: Hounsfield units.

PCATa was −73.674 ± 5.575 HU versus −80.756 ± 5.511 HU in participants with versus without low-attenuation plaque, −73.073 ± 5.666 HU versus −80.587 ± 5.507 HU for positive remodeling, −73.141 ± 5.531 HU versus −80.398 ± 5.669 HU for spotty calcification, and −72.529 ± 6.035 HU versus −80.010 ± 5.784 HU for

the napkin-ring sign. Overall, the results demonstrate a consistent association between the presence of individual HRP features and less negative (higher) PCATa.

Inter-reader agreement was excellent for HRP classification and PCATa in the 1,482 participants, as shown in Table 5.

Assessment	Agreement statistic	Estimate (95% CI)	P value
HRP classification	Cohen's κ	0.964	<0.001
PCATa	ICC, single measures	0.961 (0.956–0.964)	<0.001

TABLE 5: Inter-reader agreement for HRP classification and PCATa

Cohen's κ was used for categorical HRP classification. We calculated ICC using a two-way mixed-effects model with absolute agreement for single measurements. P values <0.05 were considered statistically significant.

HRP: high-risk plaque; PCATa: pericoronary adipose tissue attenuation; ICC: intraclass correlation coefficient; CI: confidence interval (N=1,482).

Agreement for HRP classification was extremely high (Cohen's κ=0.964, P<0.001), and agreement for PCATa was excellent (ICC=0.961, 95% CI 0.956-0.964, P<0.001), indicating high consistency between the two readers.

Discussion

Association between PCATa and patient-level HRP feature burden

This study demonstrated a consistent association between PCATa and HRP characteristics on CCTA. Higher PCATa was positively correlated with the total number of recorded patient-level HRP features (Spearman's ρ=0.498, P<0.001), indicating an association between PCATa and greater cumulative feature burden. This result aligns with published findings of increased PCATa in HRP patients and in HRP patients who later developed acute coronary syndrome (ACS) [13]. PCATa also increased progressively with the total number of patient-level HRP features, supporting a graded relationship rather than a simple binary distinction between patients with and without HRP. This finding is consistent with previous CCTA evidence demonstrating higher PCATa with an increasing number of plaques exhibiting high-risk features per patient [14]. The two-model analysis further supports the robustness of the association between PCATa and higher patient-level HRP feature burden. Model 1, adjusted for conventional cardiovascular risk factors, including age, sex, BMI, hypertension, diabetes mellitus, dyslipidemia, smoking status, and statin use, demonstrated that each 1-HU increase in PCATa was associated with 24.0% higher adjusted odds of higher patient-level HRP feature burden (adjusted OR, 1.240; 95% CI, 1.200-1.282; P<0.001). This association remained significant, although slightly attenuated, in Model 2 after additional adjustment for maximum coronary stenosis (adjusted OR, 1.193; 95% CI, 1.132-1.257; P<0.001). These findings are consistent with previous CCTA studies reporting associations between PCATa, HRP features, and adverse cardiovascular outcomes, even after adjustment for plaque characteristics, stenosis severity, and plaque burden [13,15]. These results together suggest potential for the use of PCATa to supply data beyond traditional assessments of coronary disease that can be related to plaque vulnerability. However, because of the cross-sectional design of the study, the present results are more to be viewed as an independent association, not necessarily as causal or incremental prognostic data.

The direction of the age association changed after additional adjustment for maximum coronary stenosis. Age was positively associated with higher patient-level HRP feature burden in Model 1 (adjusted OR, 1.042; P<0.001), whereas the association was no longer statistically significant in Model 2 (adjusted OR, 0.984; P=0.233). This change should be interpreted as a difference in the conditional association after adjustment rather than evidence of a protective effect of older age. Previous CCTA data have demonstrated that older age is associated with greater overall coronary plaque burden and differences in plaque composition, while the prevalence of qualitative HRP features does not necessarily increase in parallel with age [16]. Importantly, the relationship between PCATa and HRP was still statistically significant once accounting for the maximum stenosis.

This study is consistent with growing evidence that PCATa represents biological events associated with coronary atherosclerosis and plaque vulnerability. PCAT is contiguous with the coronary arterial wall and may be sensitive to inflammatory signals from the vascular tissue beneath the wall [5]. Inflammatory activation may change the cellular and lipid makeup of perivascular adipose tissue, which is associated with less negative CT attenuation. Therefore, increased PCATa could potentially be an imaging correlate of the vascular inflammatory environment of the coronary artery [5]. Prior studies have also linked increased PCATa to adverse plaque features, supporting the biological plausibility of the present results [10,17]. Our study extends this evidence by demonstrating that the association between PCATa and plaque vulnerability

is evident both for a composite HRP phenotype and across individual HRP characteristics.

PCATa and the spectrum of HRP features

We also observed consistent associations across all four predefined HRP characteristics. PCATa was significantly elevated in participants with low-attenuation plaque, positive remodeling, spotty calcification, and the napkin-ring sign, with all comparisons having a P<0.001. Previous studies have shown that PCATa is associated with HRP, but the strength of association varies by plaque phenotype [9,11]. This finding matters because these features represent different morphological expressions of plaque vulnerability. The consistent direction of association suggests that elevated PCATa may reflect a broader pericoronary environment associated with multiple manifestations of plaque vulnerability, rather than a single morphological characteristic. Recent CCTA studies support this interpretation, showing higher PCATa in patients with multiple HRP features and higher PCATa with increasing HRP burden [14,18]. The progressive increase in PCATa observed from participants with no HRP features to those with four features in the present study further extends this evidence by demonstrating a graded relationship with cumulative HRP-feature burden.

Incremental information beyond coronary stenosis and diagnostic performance

The independent association between PCATa and HRP after adjustment for maximum coronary stenosis is also noteworthy. Maximum stenosis was also strongly associated with HRP, but the association of PCATa and HRP remained significant in the multivariable model. This finding supports the concept that anatomical luminal narrowing and plaque vulnerability may capture partially distinct aspects of coronary disease, although the present study did not evaluate incremental prognostic value. Similarly, previous CCTA studies have shown that HRP carries prognostic information independent of obstructive stenosis, as HRP is independently associated with coronary events after adjustment for stenosis [19,20]. Furthermore, HRP has been demonstrated to be a significant independent risk factor for events in patients with both HRP and obstructive CAD, and HRP alone can provide prognostic information similar to obstructive CAD [20]. These results support complementary evaluation of plaque morphology and luminal stenosis on CCTA, especially if HRP features can identify clinically relevant plaque vulnerability not fully appreciated by luminal narrowing. In this context, PCATa may provide complementary information regarding the biological environment surrounding the coronary artery, rather than simply reflecting luminal disease severity [21].

The diagnostic analysis also confirms the possible utility of PCATa as an additional imaging marker. PCATa demonstrated good discrimination for the study-defined higher patient-level HRP feature-burden category, with an AUC of 0.839 (95% CI, 0.816-0.862). An exploratory threshold of −77.50 HU yielded 79.7% sensitivity and 73.5% specificity. Previous HRP studies using CCTA have also shown the discriminatory performance of PCATa, with thresholds reported to vary by imaging technique and study methodology [17]. Although these results suggest effective discriminative ability, the threshold determined in the present study should not be assumed to be a universal clinical threshold because PCATa measurements vary methodologically and technically. PCATa depends on CT acquisition parameters, scanner characteristics, and reconstruction techniques [22]. Therefore, this threshold needs external validation across institutions, patient populations, and CT platforms before it can be widely adopted for routine clinical decision-making [21].

Reproducibility and clinical implications

The results are methodologically justifiable because of strong inter-reader reproducibility. Agreement was very high for HRP classification (Cohen's κ=0.964) and PCATa (ICC=0.961; 95% CI, 0.956-0.964). This is especially important because quantitative imaging biomarkers must be measured consistently and reproducibly to be reliably used in clinical practice [23]. The use of a predefined RCA segment and blinded independent image interpretation may have contributed to the high reproducibility observed in the present study [24].

Nevertheless, interpret the clinical implications of these findings cautiously. The present study shows an association between plaque vulnerability and the imaging phenotype of PCATa, but not that raised PCATa values lead to plaque instability or a future cardiovascular event. Furthermore, while PCATa remained independently associated with HRP even after adjusting for several conventional risk factors, residual confounding by unmeasured inflammatory, metabolic, or lifestyle factors remains possible. The significant Hosmer-Lemeshow test also indicates some evidence of model miscalibration, despite the strong overall model performance, further supporting the need for external validation.

Overall, the results indicate that PCATa could offer complementary biological information with traditional CCTA parameters of coronary stenosis and plaque morphology. Rather than replacing established HRP characteristics, PCATa may potentially enhance characterization of the pericoronary environment and identify patients with a more adverse plaque phenotype. Future multicenter, long-term studies that include cardiovascular outcomes are needed to determine whether PCATa provides incremental prognostic information beyond the known clinical and CCTA parameters.

Limitations

Several limitations should be considered. First, this retrospective cross-sectional design does not allow determination of temporal or causal relationships between PCATa and HRP. These data do not allow conclusions about whether elevated PCATa increases plaque vulnerability or results from an inflammatory response to underlying plaque disease. Second, this was a single-center study of patients undergoing clinically indicated CCTA. Although the study used consecutive sampling, the population may not represent asymptomatic individuals, lower-risk populations, or patients managed in other healthcare settings. Therefore, HRP prevalence and PCATa discriminative performance for the study-defined HRP outcome may vary in other populations. These results may have been influenced by referral and spectrum effects, given that the cohort comprised patients referred for clinically indicated CCTA. The results may not directly apply to populations undergoing asymptomatic screening or people who have an extremely low pretest probability of CAD.

Third, we assessed PCATa in a specific, proximal RCA segment rather than across multiple coronary vessels. This method allowed standardization of measures, but it may not have captured spatial heterogeneity of pericoronary inflammation. Furthermore, attenuation values can vary with scanner technology, X-ray tube voltage, contrast enhancement, reconstruction algorithm, and segmentation method, making it difficult to compare absolute HU values across centers directly. Although the same institutional CCTA acquisition and reconstruction protocol was used throughout the study period, individual patient-level CT acquisition and reconstruction parameters were not independently extracted from DICOM metadata or compared between the study groups. Tube voltage was adjusted according to patient body habitus; therefore, some technical variability may have remained. Consequently, differences in CT acquisition parameters cannot be completely excluded as a potential source of variability in PCATa measurements. In addition, we measured PCATa at the proximal RCA at the patient level, whereas we assessed HRP features throughout the coronary tree. Thus, this analysis did not establish a relationship between local PCATa and the plaque vulnerable site. Patient-level RCA PCAT may not fully reflect perivascular inflammatory disease in other coronary territories. Fourth, this study lacked prospective clinical outcomes and independent validation of the ROC-derived −77.50-HU threshold. Specifically, subsequent ACS events were not assessed. Therefore, we could not compare the incidence of ACS between patients with HRP and those with different PCATa levels. Consequently, the prognostic value of PCATa for predicting future ACS remains undetermined. Thus, the identified cutoff should be considered exploratory, not a clinical threshold. Renal function parameters, including serum creatinine and estimated glomerular filtration rate (eGFR), were not available in the study dataset and therefore could not be included in the multivariable analyses. Given the established relationship between impaired renal function and cardiovascular disease, residual confounding related to renal dysfunction cannot be excluded. Lastly, although the multivariable analyses adjusted for several important cardiovascular risk factors, baseline characteristics differed between patients with and without HRP. Residual confounding due to unmeasured or unavailable clinical and biological variables cannot be excluded. Furthermore, despite the relatively large sample size, the single-center nature of the study may limit the generalizability of the findings.

Future directions

Future studies should validate these findings prospectively across multiple centers and CT platforms using standardized PCAT measurement protocols. Future longitudinal studies will need to establish whether PCATa is independent of plaque burden, stenosis severity, and established HRP characteristics in predicting major adverse cardiovascular events. Lesion-specific and multi-vessel PCAT measurements may also clarify whether localized perivascular inflammation relates more strongly to individual vulnerable plaques than to patient-level measurements. Further evidence of its biological and prognostic importance may emerge by integrating PCATa with quantitative plaque burden, AI-based CCTA analysis, and intravascular imaging.

Conclusions

Among adults undergoing clinically indicated CCTA, higher PCATa was associated with a greater patient-level burden of recorded HRP features and remained independently associated with the study-defined higher feature-burden category after adjustment for conventional cardiovascular risk factors and maximum coronary stenosis. PCATa also demonstrated good discrimination for this study-defined outcome and excellent inter-reader reproducibility. These findings suggest that PCATa may provide complementary information regarding coronary plaque characteristics. However, because plaque-specific co-localisation of high-risk features was not available, the study does not establish a CAD-RADS 2.0 same-plaque HRP classification. The cross-sectional design, single-centre setting, patient-level RCA measurement, and absence of external and prognostic validation also preclude causal or prognostic inference. Further multicenter prospective studies with plaque-level assessment are needed to establish its clinical and prognostic value.

Additional Information

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All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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