# Pericoronary adipose tissue density is increased in patients with recent spontaneous coronary dissection

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## Abstract

### Objective

Inflammatory activity is one of the potential mechanisms of spontaneous coronary artery dissection (SCAD). Recently the pericoronary adipose tissue attenuation (PCAT) derived from computed tomography angiography (CTA) has been established as a method for measuring vascular inflammation. We aimed to characterize the pancoronary and vessel-specific PCAT in patients with and without recent SCAD.

### Methods

The study comprised SCAD patients referred to a tertiary center between 2017 and 2022 who underwent CTA and were compared with individuals with no prior SCAD. PCAT was analyzed on end-diastolic CTA reconstructions along proximal 40 mm of all major coronary vessels as well as the SCAD-related vessel. We analyzed 48 patients with recent SCAD (median 6.1 [3.5-14.9] months since SCAD, 95.8% female) and 48 patients in the non-SCAD group.

### Results

Pancoronary PCAT was higher in patients with SCAD compared with non-SCAD (-80.6±7.9 vs. -85.3 HU ±6.1, P=0.002). Vessel-specific PCAT in patients with SCAD compared with non-SCAD was higher both for the RCA (-80.9±9.5 vs. -87.1±6.9 HU, P=0.001) and the LCA (-80.3±7.8 vs. -83.4±7.2 HU, P=0.04). In patients with SCAD, PCAT of SCAD-related vessel was not significantly different from averaged PCAT of unaffected vessels (-81.2±9.2 vs. -80.6±7.6, P=0.74). There was no association between PCAT and the interval from SCAD to CTA.

### Conclusions

Patients with recent SCAD have higher pericoronary adipose tissue attenuation compared with non-SCAD patients suggesting an increased perivascular inflammatory activity. This association is not restricted to the dissected vessel.

**Keywords**: spontaneous coronary artery dissection, inflammation, computed tomography, pericoronary adipose tissue

## Key Messages

### What is already known about this subject?

PCAT (pericoronary adipose tissue attenuation derived from computed tomography) is an emerging marker of atherosclerotic plaque vulnerability. Non-atherosclerotic spontaneous coronary artery dissection (SCAD) has distinct pathophysiology which involves the vessel wall and perivascular space.

### What are the new findings?

- PCAT assessment is feasible in patients with recent SCAD
- Patients with recent SCAD have higher PCAT compared with individuals with no history of SCAD
- PCAT is elevated not only in the SCAD-related, but also in remote coronary vessels
- No apparent association of PCAT and time since SCAD was observed

### How might these results change the focus of research or clinical practice?

Consistently higher PCAT may suggest increased inflammatory activity of perivascular space in patients with recent SCAD. Whether it is a patient-specific feature of vulnerability or a persistent sequelae of the acute coronary event remains to be determined.

## Background

Spontaneous coronary artery dissection (SCAD) is increasingly being recognized as a significant cause of myocardial infarction, especially in young and middle-aged women.(1) Its pathophysiology is consistent with formation of an intramural haematoma within the tunica media of a coronary artery causing compression of its true lumen, ultimately leading to blood flow restriction or obstruction. While the underlying mechanisms of hematoma formation are not well understood, autopsy studies in fatal cases of SCAD-related myocardial infarction have shown increased perivascular inflammation. (2)

Recently pericoronary adipose tissue attenuation (PCAT) has been established as a non-invasive and reproducible imaging method for detection and quantification of perivascular inflammation in the coronary vasculature.(3) In atherosclerotic coronary artery disease higher PCAT was associated with a markedly increased risk of future myocardial infarction above and beyond risk scores, the coronary calcium burden and the presence of adverse plaque morphology.(4,5) To what extent this parameter could be useful in patients with primarily non-atherosclerotic coronary pathologies, such as those with recent SCAD, remains undetermined. In this study we therefore aimed to characterize PCAT in SCAD patients and compare it with non-SCAD individuals.

## Methods

*Population*

This is a retrospective observational study based on a countrywide prospective registry of patients with recent SCAD evaluated at the National Institute of Cardiology between 2017 and 2022. At our institution patients with angiographically confirmed SCAD undergo routine follow-up CTA and vascular CT for evaluation of the coronary vessel healing process and extracoronary vascular abnormalities. We identified consecutive SCAD patients with good CTA quality and compared them with non-SCAD patients of similar age, sex and risk factor profile without obstructive coronary artery disease who underwent CTA within the same timespan, using the same CT scanner and acquisition protocol (Figure 1). Studies were conducted with the approval of the National Institute of Cardiology research ethics committee (decision IK-NPIA-0021-63/1724/18), in accordance with the Declaration of Helsinki and with the written informed consent of each participant.

*Angiographic analysis*

All angiograms acquired at the acute phase of SCAD were retrospectively analyzed by two experienced interventional cardiologists (JKa and RW). Dissections were classified according to the classification proposed by Saw et al.(6) and modified by authors of the ESC-ACCA position paper (1)

into 5 categories: 1-arterial wall contrast staining with multiple radiolucent lumens; 2a-diffuse lumen narrowing bordered by normal artery segments; 2b- diffuse lumen narrowing extending to the tip of the artery; 3-focal or tubular stenosis mimicking atherosclerosis and 4-total occlusion of a distal vessel. Multivessel SCAD was defined as simultaneous dissections in more than one artery, without continuity and multisegment SCAD as one dissection extending into two or more coronary segments. Proximal and distal reference diameters were measured using quantitative coronary angiography unless dissection extended to the end of the vessel. SCAD was defined as distal when its proximal part was located in the distal segment of the left anterior descending artery (LAD), distal segment of the left circumflex artery (LCX), obtuse marginal branch or distal branch of the right coronary artery (RCA) – posterior descending or posterolateral.

*CTA analysis*

The scans were acquired using a dual source 2x192 slice scanner (SOMATOM® Force, Siemens Healthineers, Forchheim, Germany) after sublingual administration of glyceryl trinitrate (0.4 mg). In patients with heart rate >80/min 5-10 mg of intravenous metoprolol was administered. A 50–80 ml bolus of Iomeprol (Iomeron 400, Bracco, Milan, Italy) was injected intravenously at 4.5 ml/s followed by a retrospectively gated or prospectively electrocardiogram-triggered acquisition protocol with 192 × 0.6 mm collimation and 70–100 kV tube voltage, adjusted according to BMI. Coronary datasets were reconstructed in mid-diastole (60–70%) and end-systole (40–50% of the R-R interval) with 0.6-mm section thickness and 0.4-mm increment.

Initially CTA datasets were analyzed in a standard fashion by an experienced operator using SyngoVia image analysis software to elucidate the coronary anatomy, patency of coronary vessels, as well as presence of coronary plaque. Diastolic reconstructions were preferentially used unless end-systolic reconstructions provided higher image quality. Pericoronary adipose tissue attenuation (PCAT) was analysed using AutoPlaque® v2.5 (Cedars Sinai Medical Center, Los Angeles, USA) according to previously described methodology.(5,7) Three-dimensional layers within radial distance from the outer coronary wall equal in thickness to the average diameter of the vessel were automatically constructed. Within such cylindrical volumes of interest voxels with CT attenuation from −190 to −30 HU were considered as adipose tissue. Pericoronary adipose tissue density was defined as the mean attenuation within such volumes of interest after automatic exclusion of adjacent myocardium and branches (Figure 2). We aimed to analyze proximal 40 mm segments of all major coronary vessels (RCA, LAD and LCX) of a diameter > 2mm in all study participants. Additionally, if a SCAD was restricted to a major (>2mm) secondary branch (ie. diagonal, marginal, posterior descending etc.) this vessel was additionally analyzed. In case a stent was placed as an initial treatment of SCAD, the analysis was performed proximally or distally to the stent, whichever provided the possibility of including 40 mm of vessel > 2mm diameter.

*Outcomes*

The primary outcome was the pancoronary PCAT defined as averaged PCAT in the RCA, LAD and LCX. Secondary analyses included vessel-specific PCAT of the RCA and LCA (averaged from LAD and LCX) as well as the SCAD-specific PCAT.

*Statistical analysis*

Categorical variables were presented as counts and percentages and compared with Fisher's exact test wherever appropriate. Continuous variables were assessed for normality using the Kolmogorow-Smirnov test. Variables with normal distribution were presented as means with standard deviations and compared with Student's T-test. Paired test was used for comparison of PCAT in dissected and non-dissected vessels. Variables with non-normal distribution were compared with the Mann-Whitney test. Three-group comparison was performed using the ANOVA test. Correlation of two continuous variables was analyzed with Pearson's test. P value of less than 0.05 was considered significant. Calculations were performed using the SPSS software v.22 (IBM corp., Armonk, USA).

*Patient and Public Involvement*

Patients and Public were not directly involved in the trial design. However, we carefully assessed the burden of the trial interventions on patients. We intend to disseminate the main results to trial participants and will seek patient and public involvement in the development of an appropriate method of dissemination.

**Results**

The final cohort comprised 48 SCAD survivors (95.8% female, mean age 45.3 ±9.4 years, median time since acute event 6.1 [3.5-14.9] months), and 48 patients without prior SCAD (91.7% female, mean age 43.5 ±8.1 years). The most prevalent indication for CTA in the control group was evaluation of stable chest pain (50%), followed by the workup of arrythmia (20.8%) and planned heart surgery (14.6%). The groups were not statistically different in terms of clinical characteristics (Table 1). However, there were differences in laboratory findings between patients with SCAD compared with the control group including lower haemoglobin (13.1 vs. 14.1 g/dL, P=0.001), C-reactive protein (0.1 vs. 0.3 mg/dL, P=0.03) and low-density lipoprotein cholesterol concentration (64 vs. 128 mg/dL, P<0.001). Moreover, patients with SCAD had lower prevalence of right-dominant coronary anatomy compared with the control group (72.9 vs. 91.9%, P=0.03).

The most prevalent vessel affected by SCAD was the LAD (45.8%). The majority of dissections were angiographic type 2 (64.6%) and multisegment (56.3%) and 14.6% affected more than one vessel (Table 2). Most SCAD patients had no sign of atherosclerosis by coronary angiography (91.7%).

Among of SCAD patients 14.6% had evidence of coronary plaque on CTA, similar to the control group (29.2%, p=0.08), while none of the individuals had >50% coronary stenosis.
Pancoronary PCAT was significantly higher in patients with SCAD compared with non-SCAD group (-80.5±7.9 vs. -85.2 HU ±6.1, P=0.002). In the vessel-specific analysis PCAT was higher both for the RCA (-80.9±9.5 vs. -87.1±6.9 HU, P=0.001) and the LCA (-80.3 ±7.8 vs. -83.5 ±7.2 HU, P=0.04) in patients with SCAD compared with non-SCAD respectively (Figure 3, Supplementary Table 1). In patients with SCAD, PCAT of the dissected vessel was not significantly different from averaged PCAT of unaffected vessels (-81.2±9.2 vs. -80.6±7.6, P=0.43). Time interval from SCAD to CTA did not correlate with the pancoronary PCAT (r= -0.08, P=0.61), as well as vessel-specific PCAT of the RCA (r= -0.04; P=0.81) or LCA (r= -0.12; P=0.44) (Figure 4). When time from SCAD was divided into tertiles, no significant differences in pancoronary and vessel-specific PCAT were observed between the first and third tertile (Supplementary Table 2). The differences in PCAT between SCAD and non-SCAD groups persisted in the subgroup analysis stratified by the scanner tube voltage (Supplementary Table 3). Patients with proximal and distal dissection had similar PCAT (Supplementary Table 4).

**Discussion**

The main findings of our study are as follows: 1) assessment of PCAT is feasible not only in patients with atherosclerosis, but also in non-atherosclerotic vascular pathologies, such as SCAD; 2) pancoronary, as well as vessel-specific PCAT is significantly higher in patients with recent SCAD compared with clinically similar patients without prior SCAD (Figure 5); 3) there is no difference of PCAT of the SCAD-related vessel compared with other vessels in patients with prior SCAD; 4) PCAT does not correlate with time since SCAD.

To the best of our knowledge our study is the largest report on the utility of non-invasive quantitative assessment of PCAT in patients with spontaneous coronary artery dissection. In recent years the awareness of SCAD as a significant cause of acute coronary syndrome and sudden cardiac death occurring mostly in young and middle-aged women substantially increased.(8) Accumulation of evidence from multiple retrospective registries contributed to improved understanding of the disease, including identification of risk factors (eg. pregnancy and postpartum period, fibromuscular dysplasia), specific triggers (including extreme physical or emotional stress or Valsalva-type activities).(9) Particular focus has been dedicated to unraveling the pathophysiological mechanisms leading to the disruption of the vessel wall. Currently it seems most plausible that the dissection is initiated from the adventitial space through traversing microvessels.(10) Multiple histopathology-based case reports suggested significant cellular infiltration of the adventitial space around coronary vessels in SCAD.(11) Given that SCAD is a recurrent condition in 10-22% of cases there is an unmet

clinical need for a repeatable, quantitative and non-invasive method for measuring disease activity and efficacy of potential secondary prevention therapies.(9)

Pericoronary fat has recently gained considerable attention. Numerous studies have shown that the fatty tissue closely surrounding blood vessels is involved in a complex bidirectional interplay with adjacent tissue and the vessel wall.(12) It has been suggested, that inflammation of the vascular wall triggers the release of cytokines, which diffuse into the perivascular space, suppress the differentiation of adipocytes and decrease intracellular lipid production.(13) Effectively, adipocytes become smaller and perivascular space expands, shifting the overall tissue balance from the lipidic towards more aqueous phase.(7) Such changes are detected by CTA as increased CT attenuation, and can be analyzed in a semi-automated way with dedicated software to be finally expressed as PCAT.(12) In patients with coronary atherosclerosis higher PCAT is a marker of vulnerable plaques, largely independent of established CTA-derived high-risk plaque features (5,14,15). Individuals with increased PCAT around proximal coronary segments had higher long-term-risk of adverse cardiac events and mortality. (4,16)

The evidence regarding the utility of PCAT in non-atherosclerotic vascular disorders is scarce. Wall et al. evaluated PCAT in patients with Takayasu arteritis reporting higher values in comparison with patients with stable CAD and correlation of higher PCAT with clinical, biochemical, and positron emission tomography imaging markers disease activity.(17) The first report of pericoronary fat changes associated with SCAD was published by Hedgire et al. who described 11 cases of "fat stranding" (increased attenuation of fat issue), of which nine had underlying atherosclerotic plaques and two had evidence of SCAD.(18) Unlike their study, our report focused specifically on patients with confirmed SCAD, applied consistent quantitative methodology of PCAT assessment and utilized a well-matched control group. Recently Yuvaraj et al. studied PCAT and epicardial fat attenuation in 11 patients with SCAD and 27 in the non-SCAD control group and concluded that the PCAT and epicardial fat attenuation was not significantly different between both groups.(19) We hypothesize that the reason for the discrepancy compared to our study could be the smaller sample size and suboptimally selected control group (imbalance in the proportion of female and male subjects, no data on the extent of atherosclerosis and inflammatory status).

While our study clearly shows higher CTA-derived PCAT in patients with SCAD, the underlying cause of this finding remains to be determined. Substantial time between SCAD and CTA and low levels of circulating biomarkers (C-reactive protein and white blood cell count) as well as higher statin use at the time of CTA in SCAD survivors, suggest that increased PCAT may result from non-inflammatory phenomena. One possibility is higher density of adventitial vasa vasorum in SCAD patients, which was previously reported in an optical coherence tomography study.(20) Whether those

microvessels play a causative role in the development of dissection or develop as a result of vessel injury is unknown. Secondly, dissections with residual intramural haematoma may result in elevated PCAT values due to contrast staining of the vessel wall. Finally, late post-injury changes in perivascular tissue composition including fibrosis may possibly lead to higher PCAT. However, uniform distribution of increased PCAT among SCAD-and non-SCAD territories does not seem to support this hypothesis.

Interestingly, we did not observe any relationship between PCAT and the interval from dissection to CT imaging (Figure 4 and Figure 6). Similarly, studies of patients with atherosclerosis failed to consistently show the dynamic changes of pericoronary fat attenuation with time since acute event (5,13). While Antonopoulos et al. have shown that perivascular fat attenuation index (FAI) around the acute myocardial infarction culprit lesion and the proximal right coronary artery decreased with time since infarction, Oikonomou et al. reported unchanged patient-level perivascular fat radiomic profile up to 6 months after myocardial infarction.(7,15) On the other hand, initiation of statin treatment led to significant decrease in perivascular fat attenuation index assessed around non-calcified and mixed, but not in calcified plaques in serially imaged patients with coronary atheroma.(21) Similarly, in patients with psoriasis, biological anti-inflammatory treatment reduced perivascular fat attenuation index at 1 year follow-up.(22) These observations may suggest that increased pericoronary inflammation in SCAD is distinct from the disease in coronary artery disease and autoimmune disorders.

Finally, in patients with recent SCAD PCAT quantified around the SCAD-related artery was not significantly different from PCAT measured around non-affected coronary vessels. This suggests that in SCAD coronary inflammation affects the entire coronary vasculature rather than being limited to the dissected artery. This observation has two potential implications. It might suggest that the active inflammatory process does not occur as a result of the dissection (an inflammatory response), but instead contributes (promotes) the acute event. Furthermore the fact that in SCAD survivors PCAT is similar across the coronary vasculature suggests that sampling a single vessel (i.e. RCA as in seminal PCAT literature) shall suffice for gaining insight into the patient's inflammatory status.(7) However, this observation should be interpreted with caution as a recent histopathology analysis has shown a relationship of the extent of inflammatory infiltrate in SCAD victims with time from symptom onset, so the answer is not yet definitive.(2)

Limitations

As for every single-center registry analysis we cannot rule out selection bias. The measured PCAT in SCAD patients does not need to be scanner agnostic and hence further multicenter studies are necessary to confirm our findings. Since our study lacks serial CTA assessment and there was

substantial variability in the interval between SCAD and CTA, the absence of correlation of PCAT with the time from the event to CTA should be interpreted with caution.

## Conclusions

Pericoronary adipose tissue attenuation is higher in patients with recent SCAD compared with individuals without prior SCAD. This association is not restricted to the dissected vessel, nor reflected in routine serum inflammatory analysis and persists over time. It suggests a global and sustained increase of pericoronary inflammatory activity in SCAD survivors.


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**Conflicts of Interest**: Nothing to Disclose.

**Data availability**: The data underlying this article will be shared on reasonable request to the corresponding author.

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Table 1. Baseline characteristics of the SCAD and non-SCAD control population

| | **SCAD (N=48)** | **non-SCAD (N=48)** | **P-Value** |
|---|---|---|---|
| Age, y | 45.3 ±9.4 | 43.5 ±8.1 | 0.34 |
| Female sex | 46 (95.8) | 44 (91.7) | 0.68 |
| Diabetes mellitus | 4 (8.3) | 1 (2.1) | 0.36 |
| Current smoking | 5 (10.4) | 7 (14.6) | 0.76 |
| Prior smoking | 2 (4.2) | 4 (8.3) | 0.68 |
| Hypertension | 22 (45.8) | 20 (41.7) | 0.84 |
| Left ventricular dysfunction | 7 (14.6) | 2 (4.2) | 0.16 |
| Hypothyroidism | 6 (12.5) | 8 (16.7) | 0.17 |
| Hyperthyroidism | 1 (2.1) | 1 (2.1) | 1.0 |
| Coronary plaque on CTA | 7 (14.6) | 14 (29.2) | 0.14 |
| >50% luminal stenosis on CTA | 0 | 0 | 1.0 |
| Laboratory findings | | | |
| White blood cell count, $10^3$/ μL | 6.5 (5.1-7.7) | 6.7 (5.5-7.9) | 0.07 |
| Haemoglobin, g/dL | 13.1 (12.3-13.6) | 14.1 (13.3-15.0) | 0.001 |
| C-reactive protein, mg/dL | 0.1 (0.1-0.2) | 0.3 (0.1-0.5) | 0.03 |
| Creatinine, mg/dL | 0.77 (0.67-0.83) | 0.74 (0.68-0.87) | 0.29 |
| LDL-cholesterol, mg/dL | 64 (53-83) | 128 (110-160) | <0.001 |
| Right dominance | 35 (72.9) | 44 (91.9) | 0.03 |
| Indication for CTA | | | |
| Follow-up after SCAD | 48 (100) | 0 | |
| Chest pain evaluation | 0 | 24 (50) | |
| Planned heart surgery | 0 | 7 (14.6) | |
| Left ventricular dysfunction | 0 | 3 (6.3) | |
| Arrythmia evaluation | 0 | 10 (20.8) | |
| Other | 0 | 4 (8.3) | |
| Scanner tube voltage, kV | 85 (80-90) | 85 (80-90) | 0.12 |

SCAD=spontaneous coronary artery dissection; Left ventricular dysfunction = LVEF <50%; LDL=low-denisity lipoprotein; RCA=right coronary artery; LAD/D=left anterior descending artery/diagonal branch; LCX/OM=left circumflex artery/obtuse marginal branch. Variables compared with Student's T-test are reported as means and standard deviations while variables compared with Mann-Whitney test are reported as medians and interquartile ranges.

Table 2. Clinical, angiographic and procedural characteristics of the SCAD population

| Clinical characteristics | |
|---|---|
| Age at SCAD, years | 43.6 ± 9.4 |
| Time between SCAD and CTA, mo. | 6.1 (3.5-14.9) |
| Pregnancy-related SCAD | 1 (2.1) |
| Prior SCAD | 3 (6.3) |
| - of which treated with PCI | 2 (4.2) |
| Angiographic and procedural characteristics | |
| SCAD-involved vessel | |
| Right coronary artery | 13 (27.1) |
| Left anterior descending artery/diagonal | 22 (45.8) |
| Left circumflex artery/marginal | 18 (37.5) |
| Left main coronary artery | 4 (8.3) |
| Multisegment SCAD | 27 (56.3) |
| Multivessel SCAD | 7 (14.6) |
| Distal SCAD | 29 (60.4) |
| SCAD angiographic classification | |
| Type 1 | 9 (18.8) |
| Type 2a | 21 (43.8) |
| Type 2b | 10 (20.8) |
| Type 3 | 3 (6.3) |
| Type 4 | 6 (12.5) |
| Initial TIMI flow | |
| 3 | 30 (63.8) |
| 2 | 7 (14.9) |
| 1 | 4 (8.5) |
| 0 | 6 (12.8) |
| Final TIMI flow | |
| 3 | 40 (83.3) |
| 2 | 5 (10.6) |
| 1 | 2 (4.3) |
| 0 | 1 (2.1) |
| Dissection length >20 mm | 39 (81.3) |
| Proximal reference lumen diameter, mm | 3.0 (2.5-3.5) |
| Distal reference lumen diameter, mm | 2.0 (1.5-3.0) |
| Atherosclerosis extent | |
| None | 44 (91.7) |
| <50% luminal stenosis | 4 (8.3) |
| >=50% luminal stenosis | 0 |
| Intravascular imaging | 3 (6.3) |
| Acute treatment of SCAD | |
| Conservative | 32 (66.7) |
| Balloon angioplasty | 3 (6.3) |
| Stent implantation | 12 (25.0) |
| Maximum stent diameter, mm | 3.0 (2.3-3.5) |
| Total stent length, mm | 41 (22-90.8) |
| Number of stents | 2 (1-3) |
| GpIIbIIIa inhibitor used | 6 (12.5) |
| Residual dissection post-stent | 9 (18.8) |
| Propagation of hematoma | 3 (6.3) |
| Coronary artery bypass grafting | 1 (2.1) |

SCAD=spontaneous coronary artery dissection; CTA-computed tomography angiography; PCI-percutaneous coronary intervention; TIMI-Thrombolysis In Myocardial Infarction

**Figure legends**

Figure 1. Study design and flowchart.

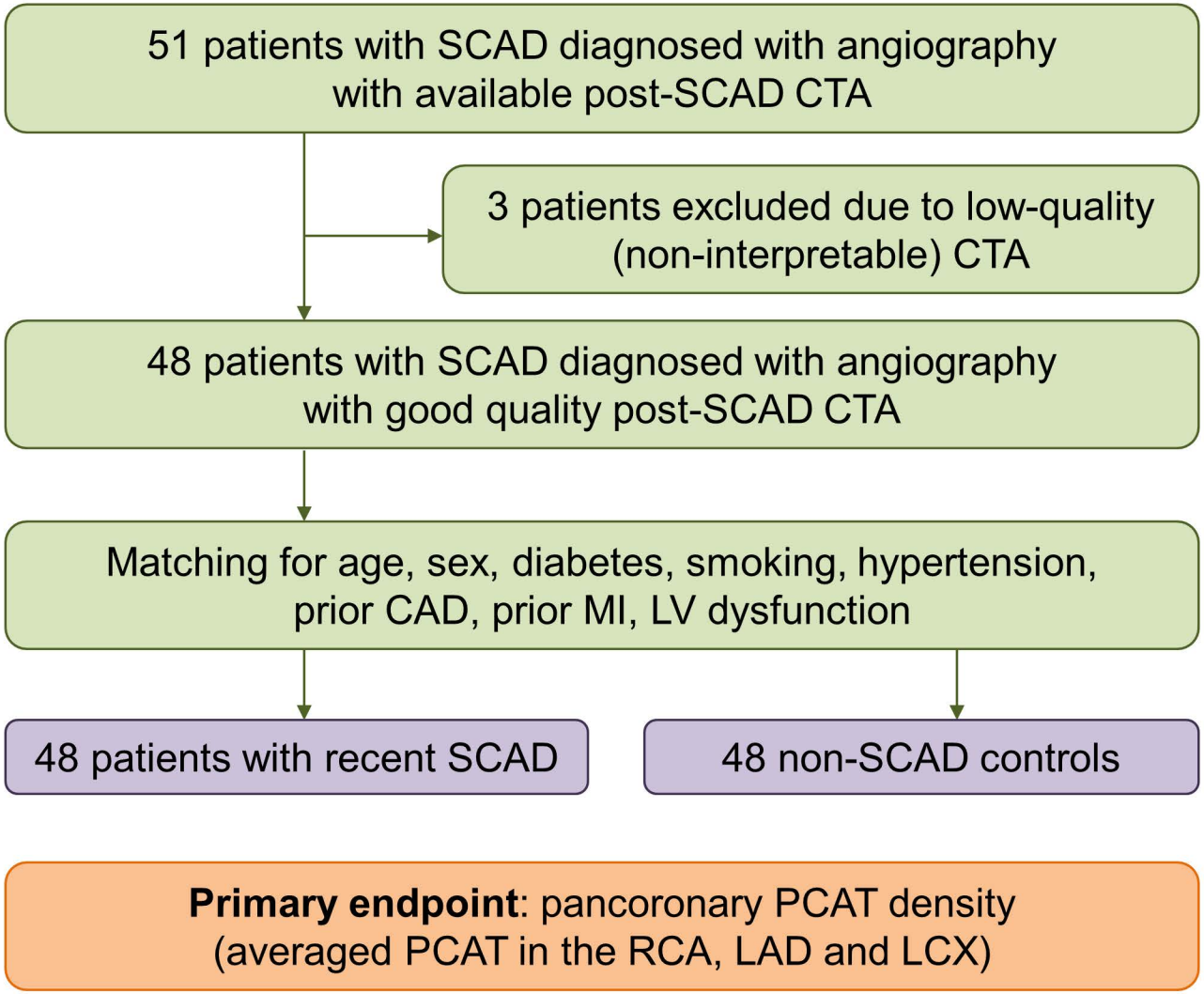
Figure 2. Representative cases of pericoronary adipose tissue attenuation (PCAT) in a patient with high (a-c) and low (d-f) PCAT. a-curved multiplanar reconstruction of right coronary artery (RCA) in a patient with prior SCAD of ramus intermedius showing high PCAT; b-short axis view of RCA in the same patient; c-straight multiplanar reconstruction of RCA in the same patient; d- curved multiplanar reconstruction of RCA in a patient with hypertension and history of smoking showing low PCAT; b-short axis view of RCA in the same patient; c-straight multiplanar reconstruction of RCA in the same patient.

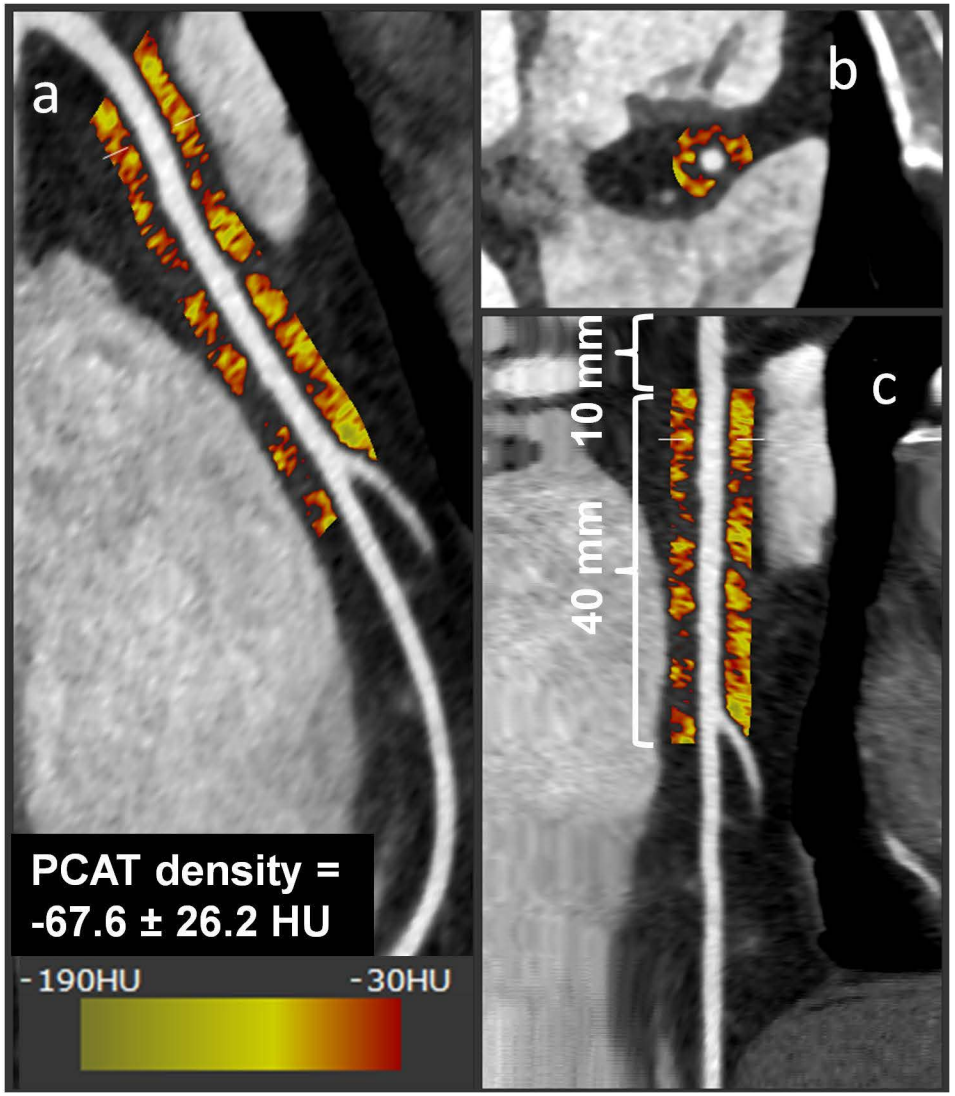
Figure 3. Pericoronary adipose tissue attenuation (PCAT) in patients with and without recent SCAD. LCA - left coronary artery. RCA - right coronary artery. Boxes represent medians and interquartile range.

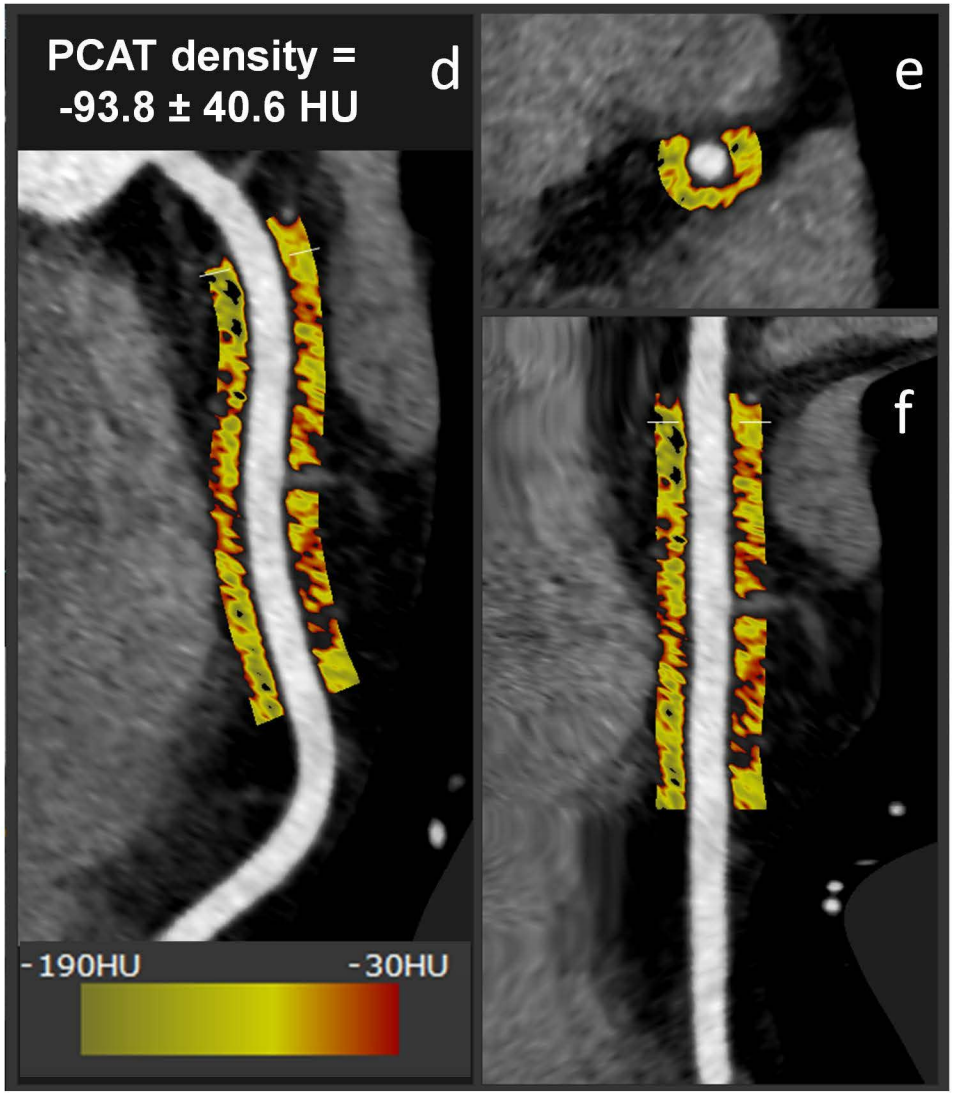
Figure 4. Pericoronary adipose tissue attenuation (PCAT) of the entire coronary vasculature (A), left coronary artery (B) and right coronary artery (C) in patients with recent SCAD according to the time since acute event. R - Pearson's correlation coefficient. Solid line - linear regression. Dotted lines - 95% confidence interval.

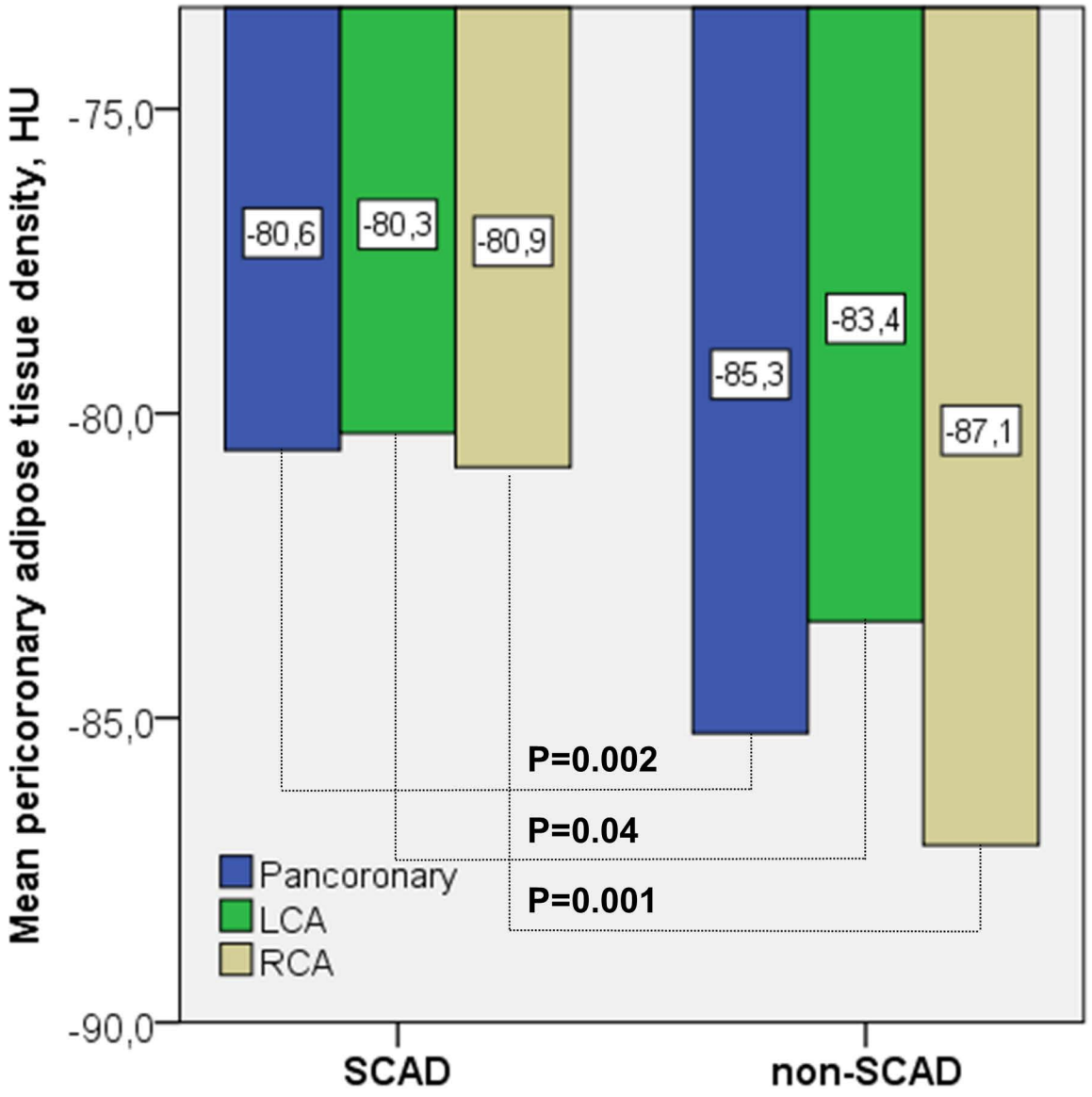
Figure 5. Summary of the study design and main findings.

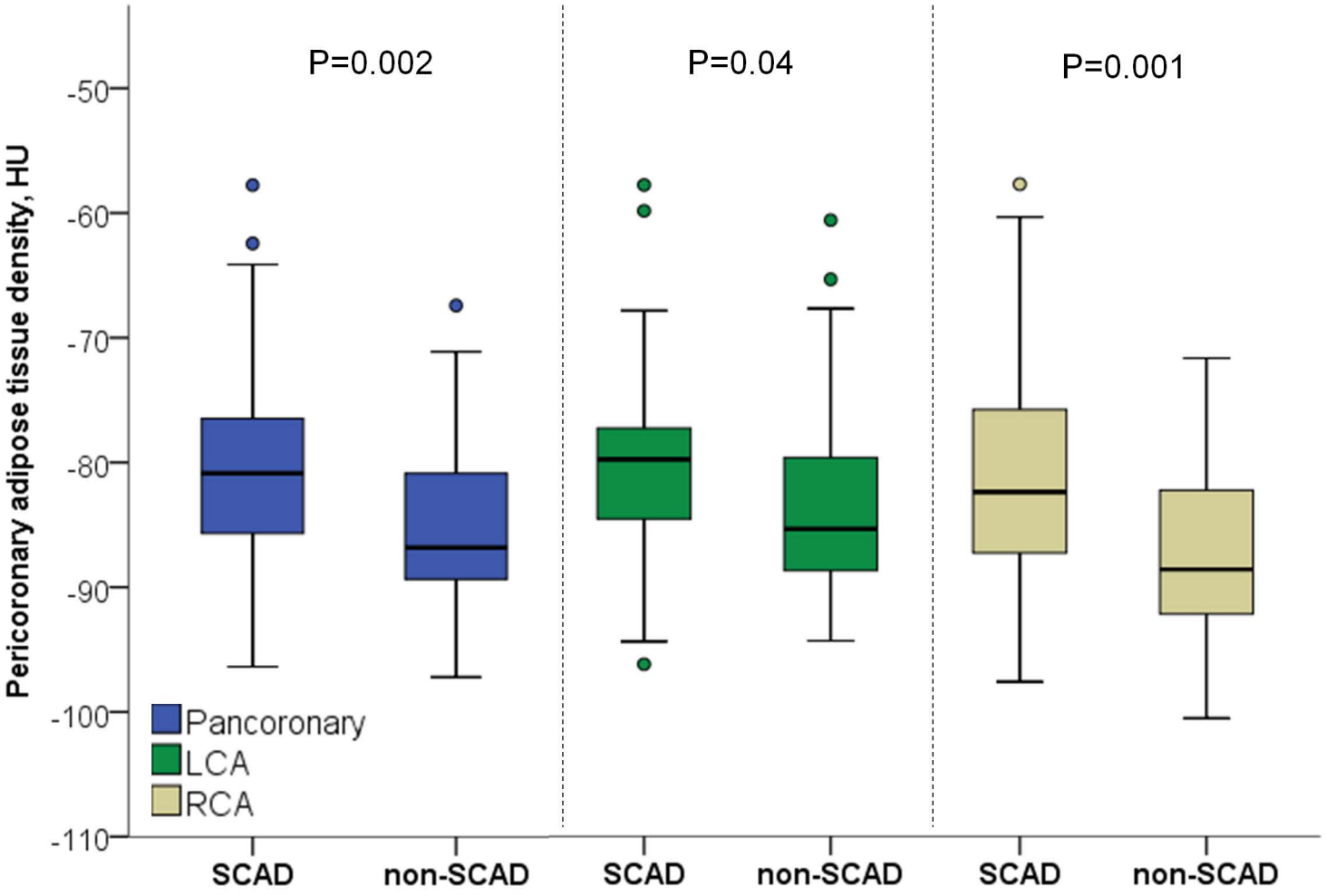
Figure 6. A patient in their 40s presenting with acute multivessel SCAD in the RCA and first obtuse marginal branch (OM1) diagnosed initially with CTA, confirmed with angiography who underwent stent implantation to the RCA. a-angiographic view of the type 1 mid-RCA SCAD and type 2a SCAD of right ventricular branches; b-angiographic view of the left coronary artery showing type 1 SCAD of OM1; c-curved multiplanar reconstruction of the RCA showing extensive hematoma (d-dotted lines show approximate vessel borders); e-curved multiplanar reconstruction of the RCA showing high PCAT; f-1-year follow-up angiogram of the RCA showing patent stent in the middle segment and healed dissection in the proximal segment; g-7-year CTA (5-mm maximum intensity projection) showing patent stent and healed dissections in the RCA; h-curved multiplanar reconstruction showing patent stent and healed dissections in the RCA; i-curved multiplanar reconstruction showing persistently high PCAT.


51 patients with SCAD diagnosed with angiography with available post-SCAD CTA
3 patients excluded due to low-quality (non-interpretable) CTA
48 patients with SCAD diagnosed with angiography with good quality post-SCAD CTA
Matching for age, sex, diabetes, smoking, hypertension, prior CAD, prior MI, LV dysfunction
48 patients with recent SCAD
48 non-SCAD controls
Primary endpoint: pancoronary PCAT density (averaged PCAT in the RCA, LAD and LCX)

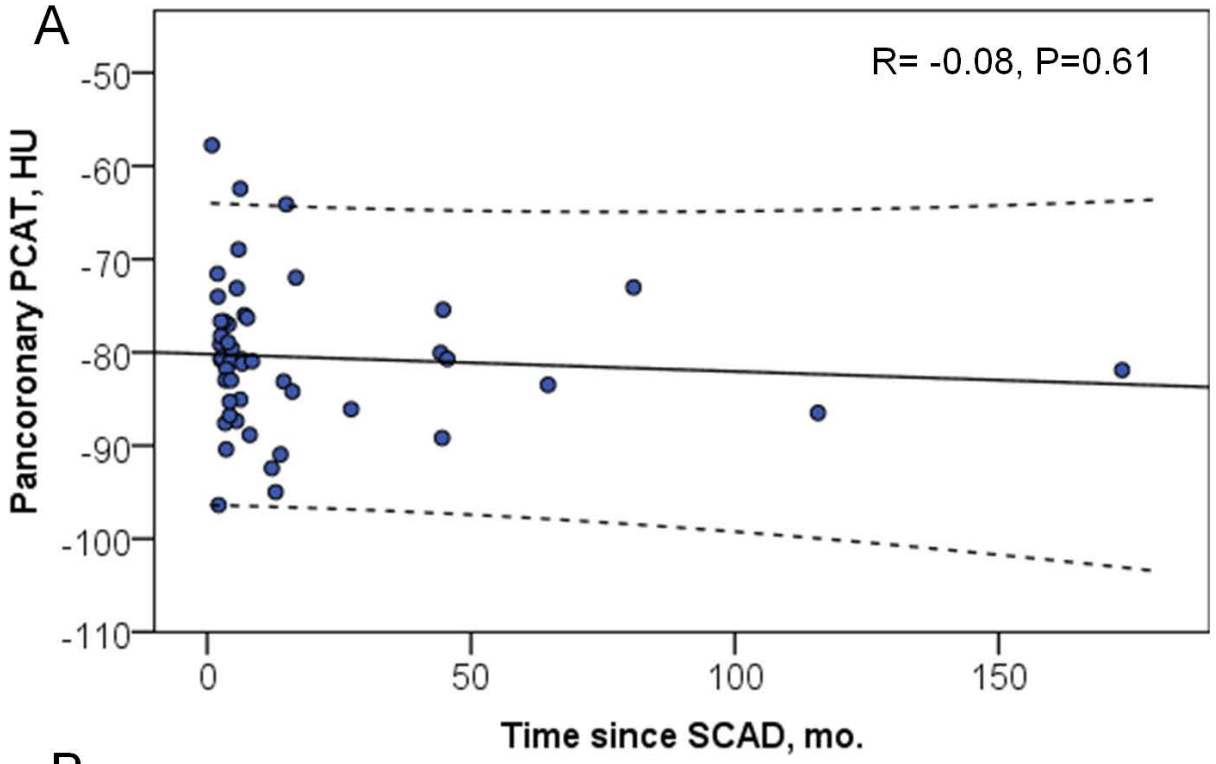

a
b
c
10 mm
40 mm
PCAT density =
-67.6 ± 26.2 HU
-190HU
-30HU


PCAT density =
-93.8 ± 40.6 HU
d
e
f
-190HU
-30HU

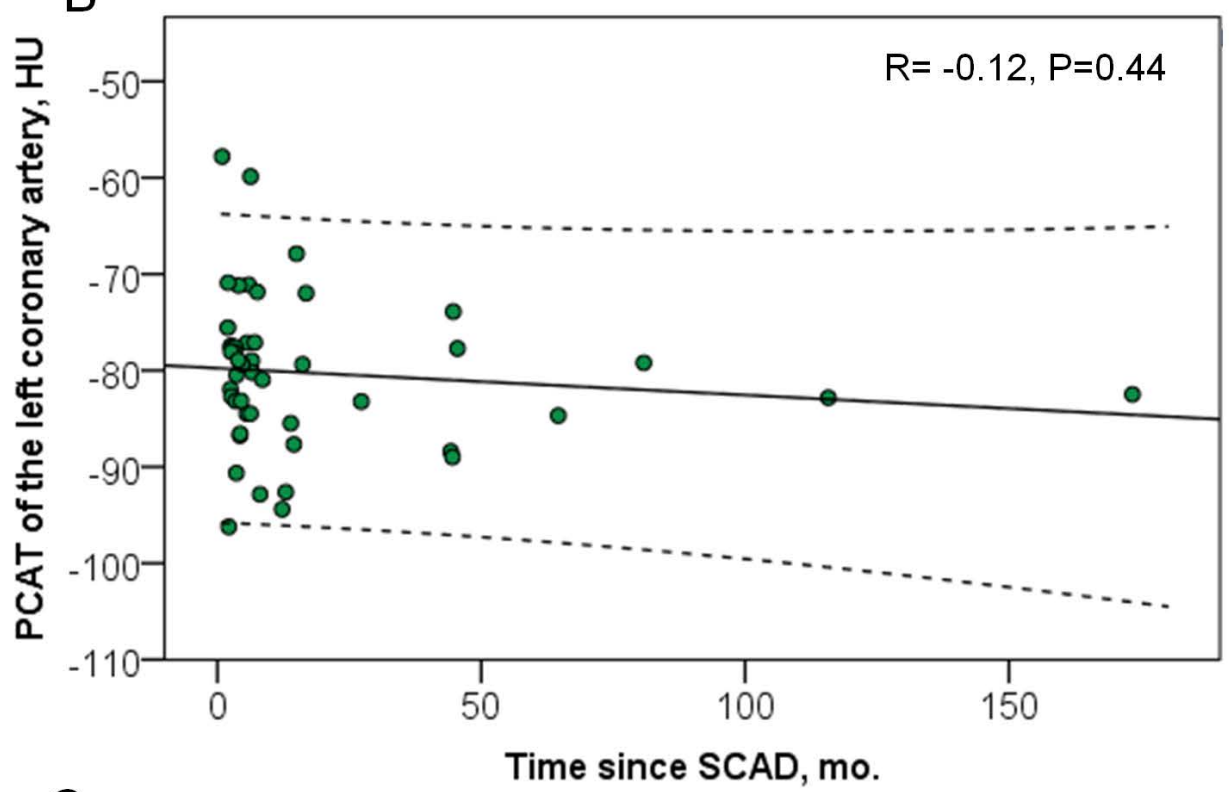

Mean pericoronary adipose tissue density, HU
-75,0
-80,0
-85,0
-90,0
-80,6
-80,3
-80,9
-85,3
-83,4
-87,1
P=0.002
P=0.04
P=0.001
Pancoronary
LCA
RCA
SCAD
non-SCAD


P=0.002
P=0.04
P=0.001
Pericoronary adipose tissue density, HU
-50
-60
-70
-80
-90
-100
-110
Pancoronary
LCA
RCA
SCAD
non-SCAD
SCAD
non-SCAD
SCAD
non-SCAD

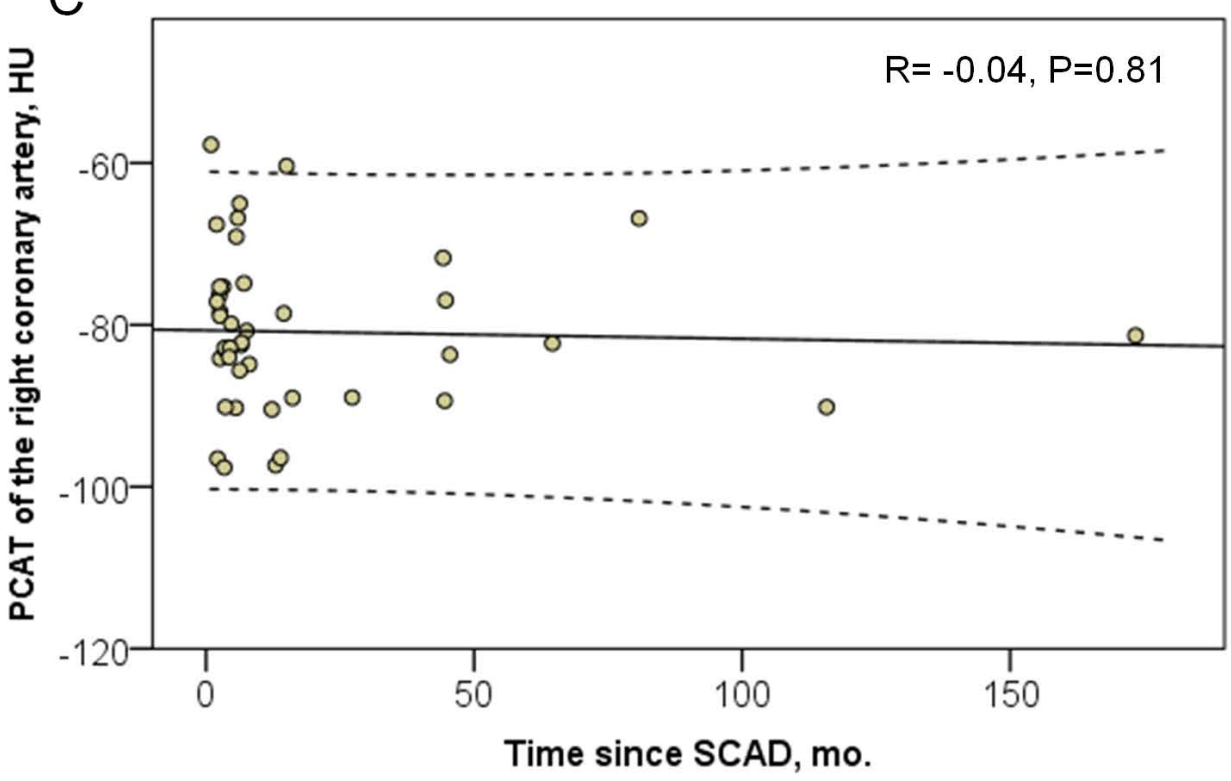
A


R= -0.08, P=0.61
Pancoronary PCAT, HU
-50
-60
-70
-80
-90
-100
-110
0
50
100
150
Time since SCAD, mo.

B

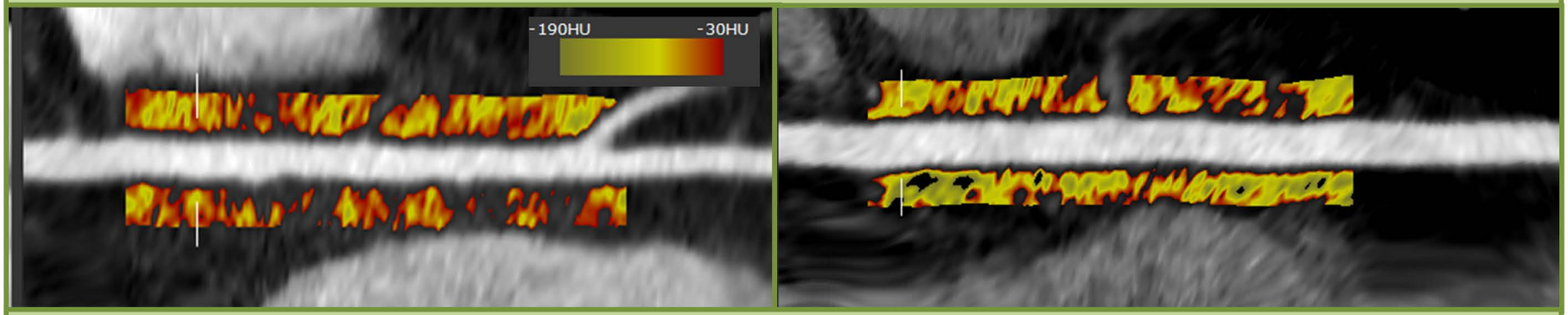

R= -0.12, P=0.44
PCAT of the left coronary artery, HU
-50
-60
-70
-80
-90
-100
-110
0
50
100
150
Time since SCAD, mo.

C


R= -0.04, P=0.81
PCAT of the right coronary artery, HU
-60
-80
-100
-120
0
50
100
150
Time since SCAD, mo.

| 48 patients with recent spontaneous coronary artery dissection (SCAD) | 48 non-SCAD patients matched for clinical characteristics |
|---|---|

CTA-derived pericoronary adipose tissue attenuation (PCAT) of three major coronary vessels and the SCAD-related vessel

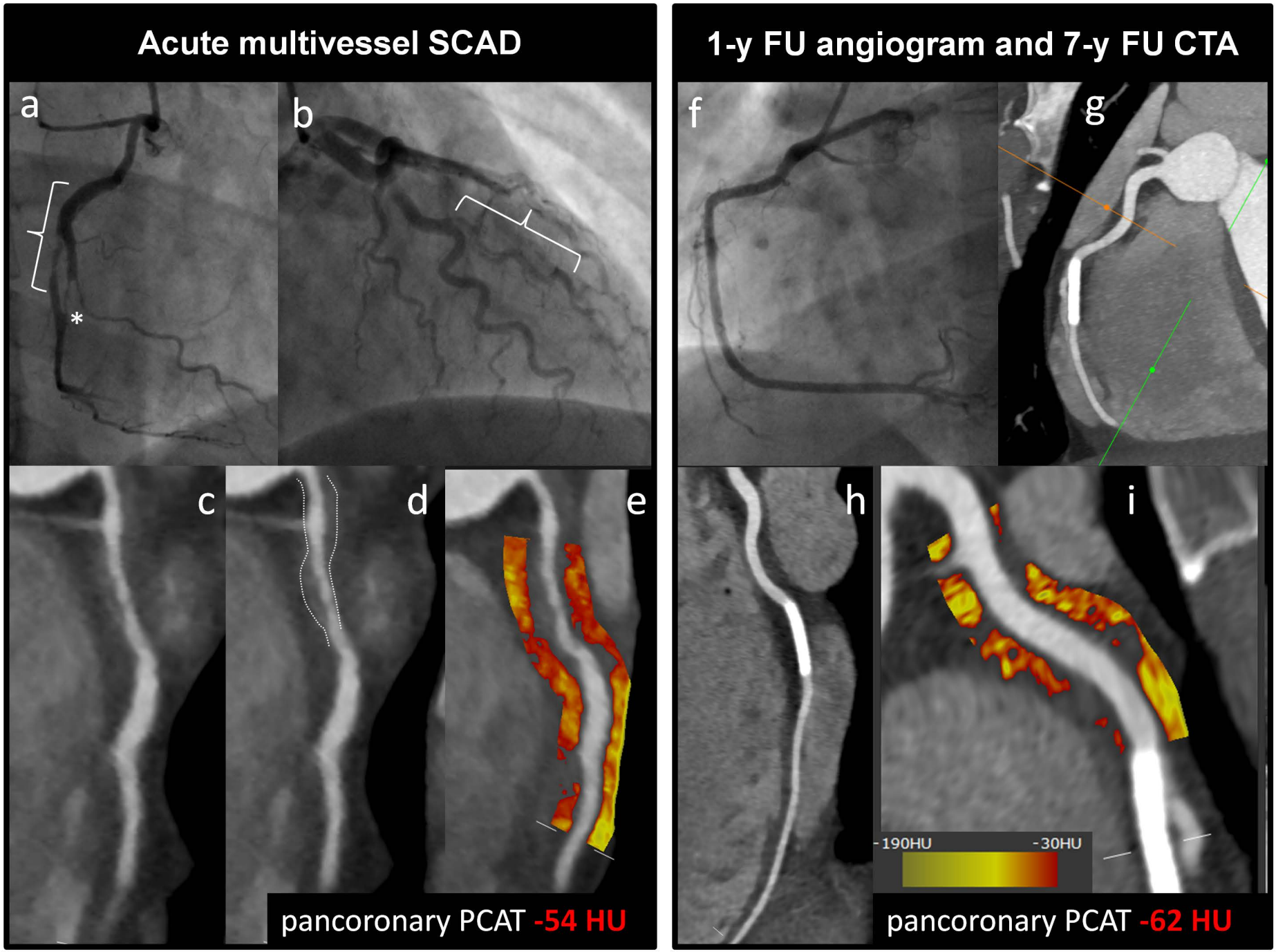



Pancoronary PCAT

| -80.5 HU | P=0.002 | -85.2 HU |
|---|---|---|

**Conclusion**

Patients with recent SCAD have higher pericoronary adipose tissue attenuation compared with non-SCAD population suggesting increased perivascular inflammatory activity.


Acute multivessel SCAD
a
b
*
c
d
e
pancoronary PCAT -54 HU
1-y FU angiogram and 7-y FU CTA
f
g
h
i
-190HU
-30HU
pancoronary PCAT -62 HU