Original Article

Pericoronary adipose tissue attenuation in patients with acute aortic dissection based on coronary computed tomography angiography

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Background: Periaortic fat is associated with coronary disease. Thus, it was hypothesized that the inflammation associated with acute aortic dissection (AAD) spreads to pericoronary adipose tissue (PCAT) via thoracic periaortic fat. Pericoronary adipose tissue attenuation (PCATa) serves as a marker for inflammation of perivascular adipose tissue (PVAT). This study sought to examine PCATa in individuals diagnosed with AAD.

Methods: Consecutive patients with chest pain from May 2020 to September 2022 were prospectively enrolled in this study and underwent coronary computed tomography angiography (CCTA) and/or aorta computed tomography angiography (CTA). Based on the results of the CTA, the patients were divided into the following two groups: (I) the AAD group; and (II) the non-AAD group. PCATa of the right coronary angiography (RCA), left anterior descending (LAD), and left circumflex (LCx) was quantified for each patient using semi-automated software. The PCATa values were compared between the AAD and non-AAD patients according to the atherosclerosis of the coronary arteries. Similarly, the PCATa values of the AAD patients were compared between the preoperative and postoperative steady states.

Results: A total of 136 patients (42 female, 94 male; mean age: 63.3±11.9 years) were divided into the two groups according to the presence of aortic dissection on CTA. The RCA_{PCATa}, LAD_{PCATa}, and LCx_{PCATa} values were significantly higher in the AAD subjects than the non-AAD subjects, regardless of the presence or absence of atherosclerosis in the coronary arteries [–85.1±9.3 *vs.* –92.9±10.0 Hounsfield unit (HU); –83.2±7.4 *vs.* –89.9±9.1 HU; –77.5±8.4 *vs.* –85.6±7.9 HU, all P<0.001). The preoperative RCA_{PCATa}, LAD_{PCATa}, and LCx_{PCATa} values were higher in the AAD patients than the postoperative steady-state patients (–82.9±8.7 *vs.* –97.6±8.8 HU; –79.8±7.6 *vs.* –92.8±6.8 HU; –74.6±7.1 *vs.* –87.7±6.9 HU, all P<0.001). According to the multivariable logistic regression analysis, high RCA_{PCATa} and LAD_{PCATa} values were associated with AAD regardless of the degree of stenosis [odds ratio (OR) =0.014; 95% confidence interval (CI): 0.001–0.177; P=0.001 and OR =0.010; 95% CI: 0.001–0.189; P=0.002].

Conclusions: PCATa on computed tomography was increased in patients with AAD regardless of the presence or absence of coronary artery disease (CAD). This suggests that vascular inflammation is present in AAD independent of CAD. Further research should be conducted to investigate the potential of this imaging biomarker to predict AAD and monitor patients' responses to therapies for AAD.

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Keywords: Acute aortic dissection (AAD); coronary computed tomography angiography (CCTA); pericoronary adipose tissue (PCAT)

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Introduction

Acute aortic dissection (AAD) is a catastrophic event and a disease with high morbidity and mortality (1). Over the last 20 years, the disease incidence rate of aortic dissection has remained stable and has been estimated to be 7.7 per 100,000 person-years (2). The exact etiology of this disorder is unknown; however, some factors are thought to be possible causes, including high blood pressure, arteriosclerosis, smoking, aging, and genetics. Regardless of the etiological factor, patients may suffer from angiogenesis and inflammatory reactions (3).

Perivascular adipose tissue (PVAT) encompasses the adipose tissue surrounding the blood vessels, specifically the tissue in close proximity to the vascular wall. This adipose tissue envelops a wide array of systemic blood vessels, encompassing major arteries and veins, organ-specific vasculature, as well as microvessels within skeletal muscle (4). The distinct inflammatory attributes resulting from PVAT dysfunction have been documented across various vascular pathologies, such as atherosclerosis, hypertension, diabetes, aging, and obesity (5-7). Thus, it is important that methodologies be developed to assess the attributes of PVAT and evaluate its inflammatory state. Such endeavors would extend understandings of the initial phases of vascular pathologies and facilitate the formulation of novel therapeutic approaches centered on PVAT.

The density of pericoronary adipose tissue (PCAT) on coronary computed tomography angiography (CCTA) is an imaging biomarker for evaluating coronary arterial inflammation. Empirical evidence indicates that the inflammatory signaling of proinflammatory cytokines from the human arterial wall and their effects on lipolysis and adipogenesis will lead to the phenotypic alterations of PVAT (8). Consequently, these processes lead to modifications in adipocyte size and volume (9). The fat attenuation index (FAI) of CCTA can be used to detect changes in adipocyte dimensions and lipid and water composition within the tissue surrounding the coronary vasculature.

Given the potential presence of PVAT inflammation in patients with AAD, and the ability of PVAT to play both paracrine and endocrine roles in cardiovascular disease through the secretion of various adipocytokines (10), it was hypothesized that pericoronary adipose tissue attenuation (PCATa) would be increased in AAD patients. However, it was uncertain whether PCATa could serve as a sensitive imaging biomarker. Thus, this study sought to compare the features of PCATa in AAD patients and non-AAD patients. We present this article in accordance with the STROBE reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-23-253/rc).

Methods

Patients

Between May 2020 and September 2022, 136 consecutive patients with chest pain were included in the study. In total, 70 AAD patients were diagnosed by aorta computed tomography angiography (CTA) or triple-rule-out computed tomography angiography (TRO-CTA). In total, 66 non-AAD patients underwent TRO-CTA. The AAD patients confirmed by CTA scanning were scanned by CCTA the day after the CTA scan was finished.

To be eligible for inclusion in this study, the patients had to meet the following inclusion criteria: (I) be aged over 30 years; (II) present with acute chest pain; and (III) have a clinical suspicion of AAD. Patients were excluded from the study if they met any of the following exclusion criteria: (I) had contraindications to contrast-enhanced computed tomography (CT); (II) were below the age of 30 years; and/or (III) had acute chest pain that was attributed to alternative causes, such as pulmonary embolism, acute myocardial infarction, or chest infection (*Figure 1*).

Of the 70 AAD patients, there were 12 Stanford A and 58 Stanford B patients. All the patients had successful stent implantations or ascending aorta replacements. Among the AAD patients with atherosclerosis of the coronary artery, 16 underwent second CCTA scans six months after the stent

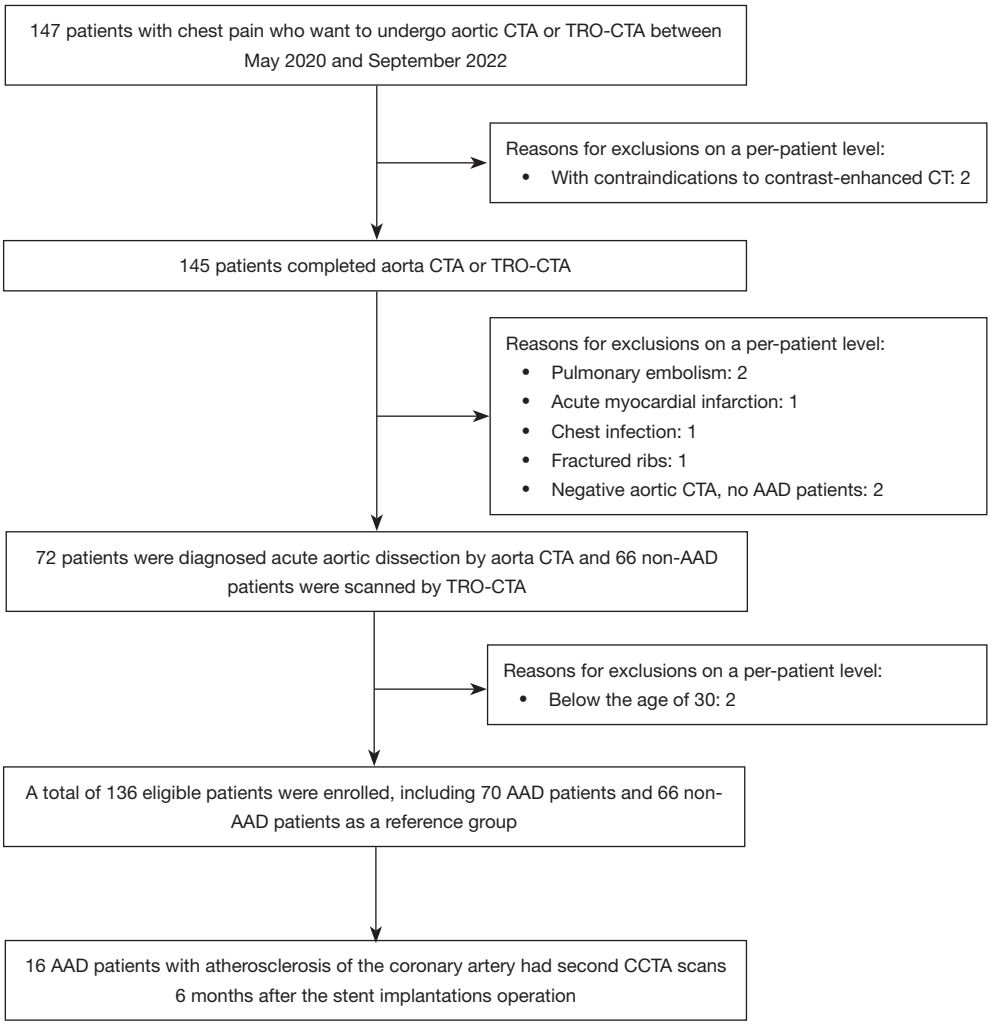

Figure 1 Flowchart displaying the number of patients enrolled in the study. CTA, computed tomography angiography; TRO-CTA, triple rule-out computed tomography angiography; CT, computed tomography; AAD, acute aortic dissection; CCTA, coronary computed tomography angiography.

implantation operation.

This was a prospective, cross-sectional study. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and was approved by the Medical Ethics Committee of Chongqing General Hospital. Informed consent was obtained from all patients.

CTA acquisition

All the patients were scanned on a dual-layer spectral detector CT scanner (IQon, Philips Healthcare, Best, The Netherlands). The automatic bolus tracking method was used in the scans. The region of interest (ROI) was located at the center of the thoracic aorta at the bronchial bifurcation level. The ROI size was 8 mm^2. When the predetermined signal attenuation threshold reached 100 Hounsfield units (HUs), the scans were initiated automatically. Contrast media (Visipaque Iodixanol 370; GE Healthcare, Ireland) was injected intravenously through the antecubital vein using an 18-gauge catheter dual-tube high pressure syringe (Ulrich REF XD 2051) with a fixed 4 mL/s flow rate. Next, 30 mL of saline was injected at the same injection rate. Contrast media was used on an individual basis according to body weight. The total amount of contrast media was

calculated as follows: total amount of contrast media = patient weight × 0.8 mL/kg.

The scan parameters were as follows: tube voltage: 120 kVp; tube current automatic exposure control: dose right index =13; field of view: 250 mm; tube rotation time: 0.27 s; detector collimation: 64 mm × 0.625 mm; slice thickness: 0.9 mm; increment: 0.45 mm; and matrix: 512×512.

The patients diagnosed with AAD by a cardiac surgeon had CTA scans performed from the arcus aortae to the external iliac artery. The scanning range of the CCTA was from the lower edge of the heart to the tracheal eminence. The scanning range of the TRO-CTA was from the lung apices to the diaphragm. In CCTA and TRO-CTA, electrocardiogram-gated acquisitions were used. Before the CCTA and TRO-CTA examinations, a 25–50 mg of a β-receptor blocker (metoprolol succinate sustained-release tablets, AstraZeneca, Sweden) was orally administered to reduce and stabilize the heart rate of the patients with a heart rate >75 bpm.

PCATa analysis

Cardiovascular radiologists with more than 10 years of experience performed the measurements. The CT measurement of the PCAT was fully automated. In all the patients, PCATa was measured using a dedicated workstation (Coronary Artery Analysis 3.0.0.1, DeepWise version, China). An area of PCAT was defined as adipose tissue located within radial distances equal to the diameter of the coronary artery (8,11). An attenuation measurement of the adipose tissue was taken on all voxels with attenuation values between –190 and –30 HU, and the PCATa value was automatically calculated as the average CT for PCATa. As the PCAT was sampled with multiplanar reconstructions, a fully automated manner was used to track the lumen and the inner and outer vessel wall border in the pre-identified segment of interest. In relation to overall length, the mean CT PCATa values of the left anterior descending (LAD), the left circumflex artery (LCx), and the right coronary angiography (RCA) were recorded in an automated manner.

Statistical analysis

SPSS 26.0 statistical software was used. The measurement data are expressed as the mean ± standard deviation. The categorical variables are expressed as frequencies and percentages. The Shapiro-Wilk test was used to test the normal distribution the continuous data. Levene's test was used to assess variance homogeneity. If the data were homogeneous and normally distributed, an independent sample *t*-test was used to compare the groups. The Chi-squared test was used to assess the categorical variables. A P value <0.05 was considered statistically significant. To ascertain the association between the CCTA-derived imaging parameters and AAD, multivariable logistic regression analysis was conducted, with adjustment variables, including age, sex, dysarteriotony, dyslipidaemia, and current smoking status.

Results

Clinical characteristics

From May 2020 to September 2022, 136 patients (42 female, 94 male; mean age: 63.3±11.9 years) suffering from acute chest pain were enrolled in the study and divided into two groups according to the presence of AAD on CCTA. Of these patients 70 patients (20 women, 50 men; mean age, 61.3±12.3 years; range, 34–93 years) had AAD, and 66 patients (22 women, 44 men; mean age, 65.4±11.2 years; range, 42–89 years) did not have AAD. The AAD patients were predominantly male, were smokers and had hyperlipidaemia. The RCA_{PCATa}, LAD_{PCATa} and LCx_{PCATa} values were significantly higher in the AAD patients than the non-AAD subjects (–85.1±9.3 *vs.* –92.9±10.0 HU; –83.2±7.4 *vs.* –89.9±9.1 HU; –77.5±8.4 *vs.* –85.6±7.9 HU, all P<0.001) (*Table 1*).

Comparison of CT parameters stratified by AAD and non-AAD

Regardless of whether the coronary arteries were normal with or without obstructive stenosis, the RCA_{PCATa}, LAD_{PCATa} and LCx_{PCATa} values were significantly higher in the AAD group than the non-ADD group (normal coronary artery: –87.1±8.8 *vs.* –94.7±9.9 HU, P=0.002; –85.5±7.6 *vs.* –92.1±8.6 HU, P=0.002; –80.8±8.1 *vs.* –89.6±7.0 HU, P<0.001, respectively; without obstructive stenosis: –84.5±11.4 *vs.* –93.3±12.1 HU, P=0.03; –83.4±6.8 *vs.* –89.6±9.1 HU, P=0.04; –76.9±8.2 *vs.* –82.7±7.6 HU, P=0.04, respectively, *Figure 2*; with obstructive stenosis: –83.0±7.8 *vs.* –89.1±7.3 HU, P=0.01; –80.2±6.8 *vs.* –86.1±9.3 HU, P=0.03; –73.9±7.6 *vs.* –80.8±6.2 HU, P=0.004, *Figure 3*).

For the obstructive stenosis group, non-calcified plaque,

Table 1 Demographic data

Baseline characteristics	Total (n=136)	Patients with AAD (n=70)	Patients without AAD (n=66)	P value
Age (years)	63.3±11.9	61.3±12.3	65.4±11.2	0.46
Male	94 (69.1)	50 (71.4)	44 (66.7)	0.55
Other risk factors				
Dysarteriotony	94 (69.1)	43 (61.4)	51 (77.3)	0.04
Dyslipidemia	73 (53.7)	40 (57.1)	33 (50.0)	0.13
Current smoker	40 (29.4)	15 (21.4)	25 (37.9)	0.02
CACS	68 (50.0)	36 (51.4)	32 (48.5)	0.73
RCA_{PCATa} (HU)	−88.9±10.4	−85.1±9.3	−92.9±10.0	<0.001
LAD_{PCATa} (HU)	−86.4±8.9	−83.2±7.4	−89.9±9.1	<0.001
LCx_{PCATa} (HU)	−81.5±9.1	−77.5±8.4	−85.6±7.9	<0.001

Data are presented as the mean ± standard deviation or number (percentage). AAD, acute aortic dissection; CACS, coronary artery calcium scoring; RCA, right coronary angiography; PCATa, pericoronary adipose tissue attenuation; LAD, left anterior descending; LCx, left circumflex.

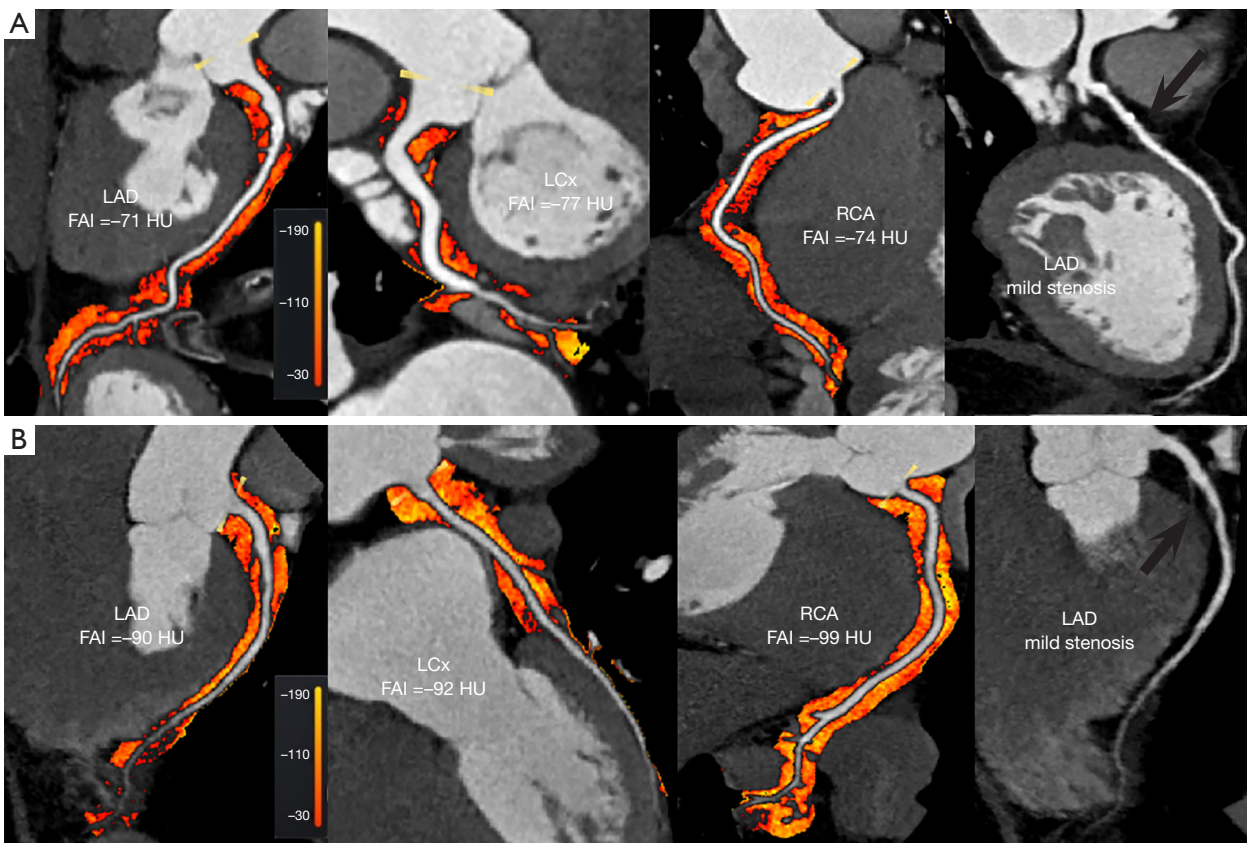

Figure 2 Representative case of AAD and non-AAD patients without obstructive stenosis. (A) CCTA of a 65-year-old female with AAD revealed calcified plaque and mild stenosis at the proximal LAD. (B) CCTA of a 66-year-old male without AAD revealed calcified plaque and mild stenosis at the middle LAD. The LAD, LCx, RCA, and PCATa values were higher in the AAD patients than the non-AAD patients. LAD, left anterior descending; FAI, fat attenuation index; LCx, left circumflex; RCA, right coronary angiography; AAD, acute aortic dissection; non-AAD, no acute aortic dissection; CCTA, coronary computed tomography angiography.

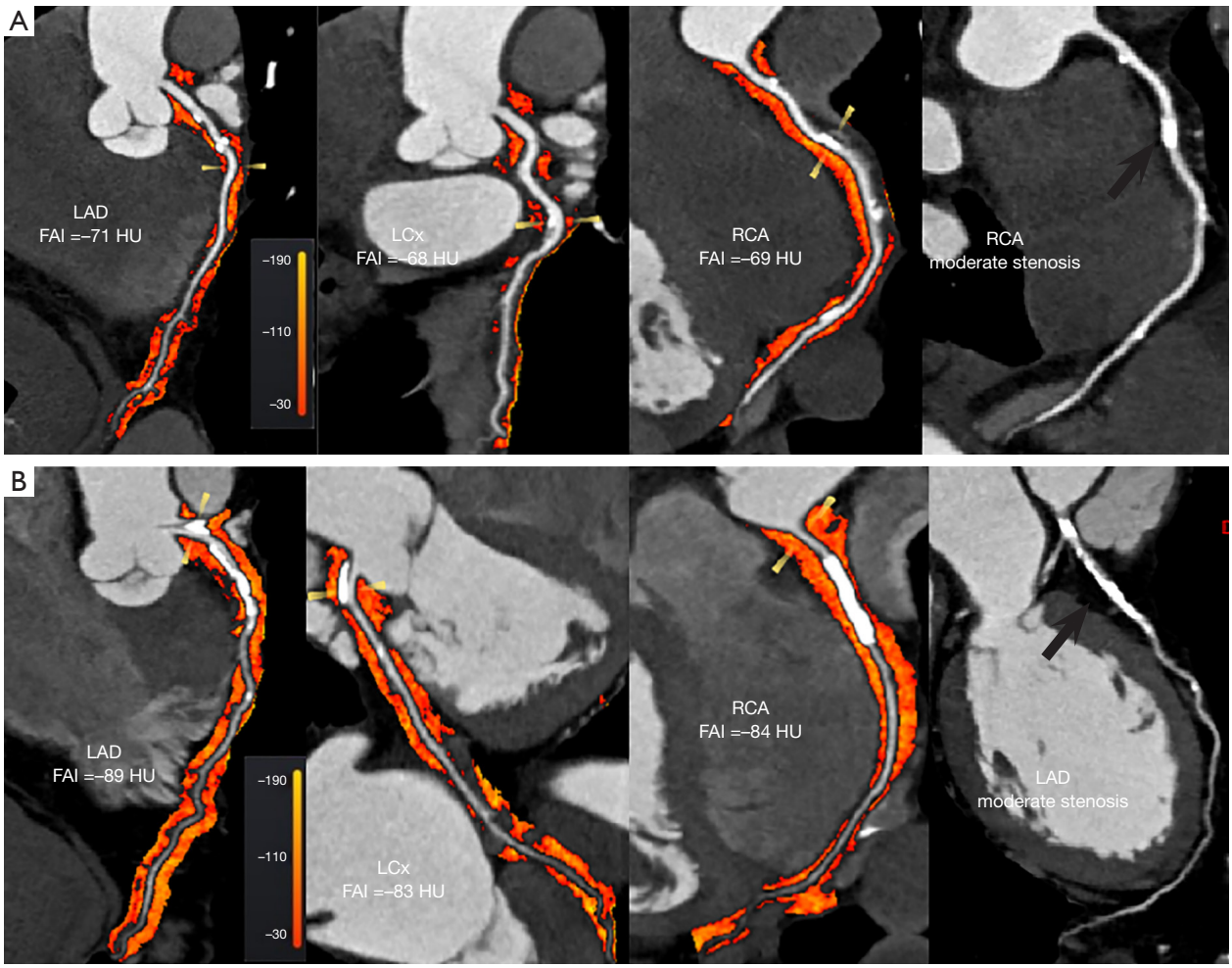

Figure 3 Representative images of AAD and non-AAD patients with obstructive stenosis. (A) CCTA of a 60-year-old male with AAD revealed calcified plaque and mild stenosis at the proximal RCA; (B) CCTA of a 62-year-old male without AAD revealed calcified plaque and mild stenosis at middle LAD. The AAD patients had higher PCATa values for LAD, LCx, and RCA than the non-AAD patients. LAD, left anterior descending; FAI, fat attenuation index; LCx, left circumflex; RCA, right coronary angiography; AAD, acute aortic dissection; non-AAD, no acute aortic dissection; CCTA, coronary computed tomography angiography; PCATa, pericoronary adipose tissue attenuation.

calcified plaque, and mixed plaque were more commonly present in the AAD patients than the non-AAD patients (82.6% *vs.* 72.2%, P=0.43; 78.3% *vs.* 66.7%, P=0.41; 82.6% *vs.* 72.2%, P=0.43), but there were no significant differences between the AAD patients and non-AAD patients in terms of non-calcified plaque, calcified plaque, and mixed plaque. In the non-obstructive stenosis group, the AAD patients showed a significantly higher prevalence of mixed plaque than the non-AAD patients (94.4% *vs.* 60.0%, P=0.01). However, the incidence of non- and calcified plaque did not differ between the two groups (*Table 2*).

Comparison of CT parameters stratified by AAD pre- and post-operation

For the AAD patients with atherosclerosis of the coronary artery, the RCA_{PCATa}, LAD_{PCATa} and LCx_{PCATa} values were significantly higher preoperatively than postoperatively (–82.9±8.7 *vs.* –97.6±8.8 HU; –79.8±7.6 *vs.* –92.8±6.8 HU; –74.6±7.1 *vs.* –87.7±6.9 HU, all P<0.001) (*Table 3*, *Figure 4*).

Table 2 Comparison of CT parameters stratified by AAD and non-AAD patients with obstructive stenosis, without obstructive stenosis, and with no atherosclerosis of the coronary artery

Baseline characteristics	With obstructive stenosis (n=41)			Without obstructive stenosis (n=33)			Normal coronary artery (n=62)		
	Patient with AAD (n=23)	Patient without AAD (n=18)	P value	Patient with AAD (n=18)	Patient without AAD (n=15)	P value	Patient with AAD (n=29)	Patient without AAD (n=33)	P value
Age (years)	71.7±10.7	70.8±8.9	0.92	57.0±10.0	64.9±11.1	0.06	55.9±10.0	62.8±11.6	0.01
Male	15 (65.2)	11 (61.1)	0.79	12 (66.7)	13 (86.7)	0.31	23 (79.3)	20 (60.6)	0.11
Dysarteriotony	14 (60.8)	15 (93.3)	0.12	13 (72.2)	11 (73.3)	0.94	16 (55.2)	25 (75.8)	0.09
Dyslipidemia	14 (60.8)	10 (55.6)	0.74	11 (61.1)	9 (60.0)	0.95	18 (62.1)	14 (42.4)	0.07
Current smoking	6 (26.1)	6 (33.3)	0.62	3 (16.7)	7 (46.7)	0.06	6 (20.7)	12 (36.4)	0.11
Non-calcified plaque	19 (82.6)	13 (72.2)	0.43	8 (44.4)	9 (60.0)	0.38			
Mixed plaque	19 (82.6)	13 (72.2)	0.43	17 (94.4)	9 (60.0)	0.01			
Calcified plaque	18 (78.3)	12 (66.7)	0.41	9 (50.0)	5 (33.3)	0.35			
RCA_{PCATa} (HU)	−83.0±7.8	−89.1±7.3	0.01	−84.5±11.4	−93.3±12.1	0.03	−87.1±8.8	−94.7±9.9	0.002
LAD_{PCATa} (HU)	−80.2±6.8	−86.1±9.3	0.03	−83.4±6.8	−89.6±9.1	0.04	−85.5±7.6	−92.1±8.6	0.002
LCx_{PCATa} (HU)	−73.9±7.6	−80.8±6.2	0.004	−76.9±8.2	−82.7±7.6	0.04	−80.8±8.1	−89.6±7.0	<0.001

Data are presented as the mean ± standard deviation or number (percentage). CT, computed tomography; RCA, right coronary angiography; LAD, left anterior descending; LCx, left circumflex; PCATa, pericoronary adipose tissue attenuation; AAD, acute aortic dissection.

Table 3 Comparison of CT parameters in AAD patients pre- and post-operation

Indicators	Pre-operation (n=16)	Post-operation (n=16)	P value
RCA_{PCATa} (HU)	−82.9±8.7	−97.6±8.8	<0.001
LAD_{PCATa} (HU)	−79.8±7.6	−92.8±6.8	<0.001
LCx_{PCATa} (HU)	−74.6±7.1	−87.7±6.9	<0.001

Data are presented as the mean ± standard deviation. CT, computed tomography; RCA, right coronary angiography; LAD, left anterior descending; LCx, left circumflex; PCATa, pericoronary adipose tissue attenuation; AAD, acute aortic dissection.

Multivariable analysis of the relationship between CCTA-based imaging parameters and AAD

After adjusting for all the other imaging parameters using multivariate logistic regression, high RCA_{PCATa} and LAD_{PCATa} values were associated with AAD regardless of the degree of stenosis [odds ratio (OR) =0.014; 95% confidence interval (CI): 0.001–0.177; P=0.001 and OR =0.010; 95% CI: 0.001–0.189; P=0.002], while the LCx_{PCATa} value was not found to be associated with AAD (OR =0.159; 95% CI: 0.012–2.127; P=0.16) (*Table 4*).

Discussion

The primary finding of this study was that the RCA_{PCATa} and LAD_{PCATa} values were significantly more elevated in AAD patients than non-AAD patients, irrespective of the presence of coronary artery stenosis. Thus, an increase in PCATa could potentially serve as an initial imaging biomarker for identifying AAD patients, regardless of the existence of obstructive stenosis.

PVAT encompasses the adipose tissue surrounding the blood vessels positioned in close proximity to the vascular wall. It envelops a vast majority of the systemic blood vessels, comprising major arteries and veins, as well as vasculature specific to organs and microvessels within skeletal muscle. PVAT is adjacent to the adventitial layer of the vascular wall separating structure or layer in large vessels, as in small vessels. It is considered an additional

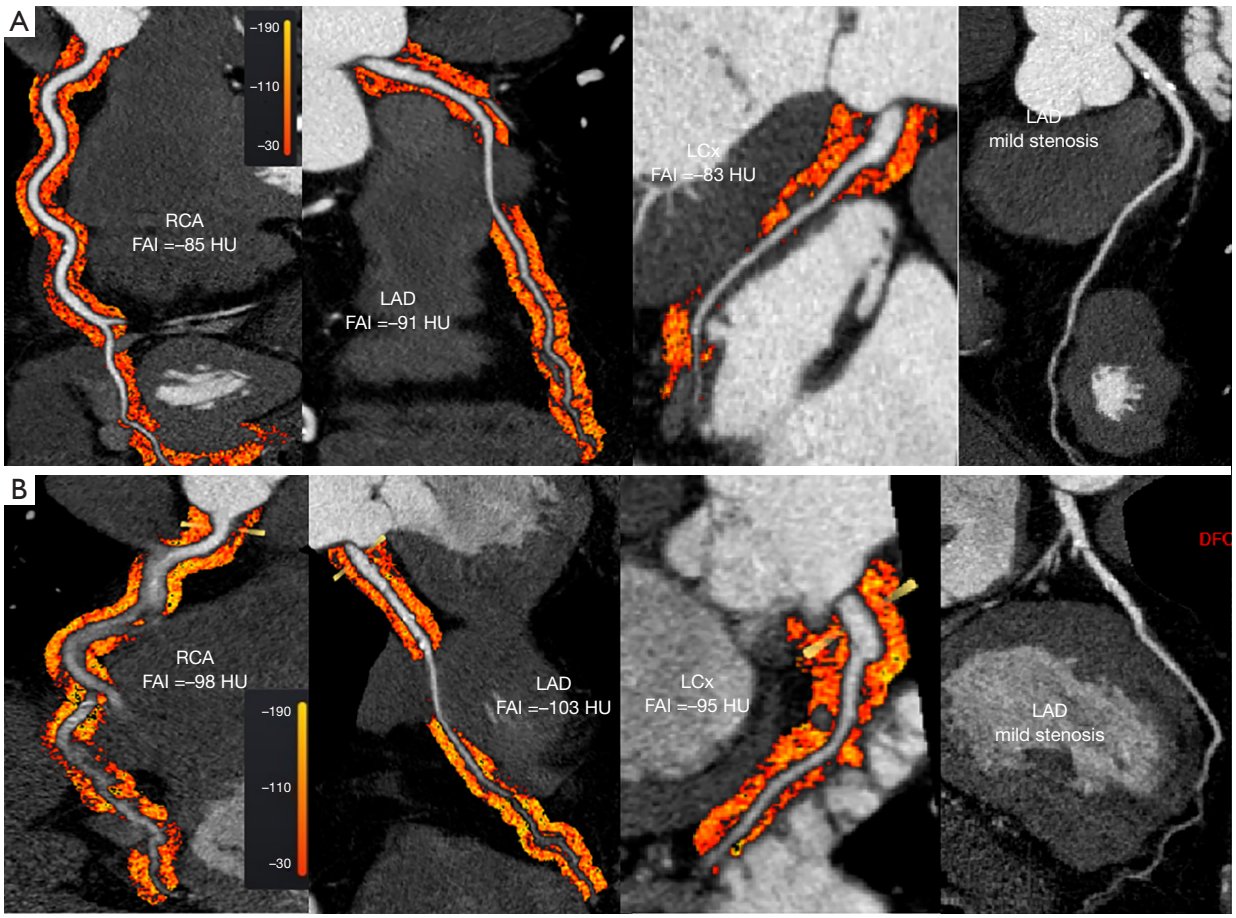

Figure 4 Representative case of AAD patients pre- and post-operation. (A) A 53-year-old female with AAD had calcified plaque and mild stenosis at her proximal LAD prior to surgery. (B) At 6 months post-surgery, the same patient showed calcified plaque and mild stenosis at the proximal LAD. The PCATa values for LAD, LCx, and RCA were higher in the preoperative period than the 6-month postoperative period. RCA, right coronary angiography; FAI, fat attenuation index; LAD, left anterior descending; LCx, left circumflex; AAD, acute aortic dissection; non-AAD, no acute aortic dissection; PCATa, pericoronary adipose tissue attenuation.

Table 4 Multivariable analysis for the relationship between CCTA-based imaging parameters and acute aortic dissection

Indicators	Univariate analysis			Multivariate analysis		
	OR	95% CI	P value	OR	95% CI	P value
Age (years)	0.800	0.386–1.658	0.54	0.574	0.153–2.827	0.65
Male	2.579	1.275–5.215	0.008	0.114	0.029–0.445	0.002
Dysarteriotony	2.135	1.008–4.521	0.04	4.902	1.403–17.130	0.01
Dyslipidemia	0.591	0.298–1.171	0.13	2.176	0.754–6.282	0.15
Current smoking	2.383	1.120–5.070	0.02	0.320	0.177–0.877	0.02
RCA_{PCATa} (HU)	7.250	3.022–17.395	<0.001	0.014	0.001–0.177	0.001
LAD_{PCATa} (HU)	2.319	1.165–4.614	0.01	0.010	0.001–0.189	0.002
LCx_{PCATa} (HU)	4.950	2.287–10.717	<0.001	0.159	0.012–2.127	0.16

CCTA, coronary computed tomography angiography; OR, odds ratio; CI, confidence interval; RCA, right coronary angiography; PCATa, pericoronary adipose tissue attenuation; LAD, left anterior descending; LCx, left circumflex.

layer of the vessel wall itself, as it can integrate into the vascular wall. Recent studies have reported that PVAT has some similarity to the endothelium in the modulation of vascular function (12-14). In a healthy state, PVAT secretes vasoprotective adipokines (e.g., adiponectin and omentin-1), which promote vasodilatation, and exert anti-inflammatory, antifibrotic, and antioxidant effects. PVAT directly induces inflammation of the adjacent arteries when it is dysfunctional or excessive (6). Vascular inflammation is a key feature in atherogenesis (15). PVAT inflammation may induce atherosclerosis as per the "out-side-to-inside" theory of vascular inflammation (16). PVAT and atherosclerotic lesions augment each other's proinflammatory state by paracrine signaling, which results in a vicious cycle.

The PVAT around coronary arteries is part of the epicardial adipose tissue (EAT) compartment. EAT is adipose tissue that is situated entirely within the pericardial sac (17). The adipose tissue located in the mediastinum, outside the pericardial sac, is commonly known as intrathoracic adipose tissue. This includes the adipose tissue surrounding both the descending and ascending aorta. Previous research has shown that the association between EAT volume and coronary artery disease (CAD) is weaker than the association between intrathoracic adipose tissue volume and CAD in patients with cardiovascular disease (18). The Framingham study reported a consistent effect size for both total mediastinal adipose tissue (both intrathoracic and epicardial) and EAT in CAD (19). Additionally, an examination of postmortem samples revealed a correlation between the expression of adipokines and cytokines in PVAT surrounding the aorta and coronary arteries, and the presence of atherosclerosis (20). Consequently, measurements of adipose tissue surrounding the coronary arteries could be used as predictive indicators of atherosclerosis (21,22).

Previous research examined whether the FAI of PVAT method could be used to measure changes in adipocyte lipid content around human coronary arteries (23). The FAI of PVAT not only detects coronary inflammation early but also identifies changes in coronary inflammation. It is important that inflamed coronary arteries be detected in treatment. Patients with high PCATa values but without CAD have an increased cardiovascular risk and require early primary preventative measures. PCATa may also identify inflamed vessels, and patients with inflamed vessels require more intense medical therapy. Previous research has shown that PCATa may be able to trace the reaction of the coronaries to systemic anti-inflammatory therapies, which should be investigated further in future randomized studies (22). However, there are relatively few studies about PCATa of the coronaries to systemic anti-inflammatory effects.

In this study, the PCATa values were higher in atherosclerosis patients with AAD than atherosclerosis patients without AAD. This finding suggests that the mutual promotion of AAD and atherosclerosis leads to an increased state of inflammation in thoracic PVAT and EAT, which may contribute to inflammation in the vessel wall through paracrine signaling (11). Our study also showed that PCATa values are low in patients with AAD concomitant with atherosclerosis after systemic anti-inflammatory therapies. This may be the cause by which PCATa can trace the reaction of the coronaries to systemic anti-inflammatory therapies (23).

High vessel pressure, low shear stress, and a turbulent flow pattern have been shown to be related to acute type A and B aortic dissection (24). In this study, we found that the prevalence of abnormal blood pressure was high, reaching 61.4%. Pathologically, hypertension is related to the activation of the renin-angiotensin-aldosterone system and increased vascular oxidative stress. The PVAT and PVAT/adventitial border are the primary sites of the initial inflammation in hypertension (25-27). PVAT may play a key role in modulating perivascular inflammation in hypertension, and researchers have shown that almost all components of the renin-angiotensin-aldosterone system, except renin, are expressed in PVAT (7,28). During the course of hypertension, there is a notable accumulation of immune cells primarily in the PVAT surrounding major blood vessels, including the aorta and renal arteries (29). There is growing evidence that the involvement of EAT may contribute to the development of hypertension (24,30). Thus, we speculate that the two above-mentioned elements may be able to generate the high PCATa of AAD.

In terms of the etiological factors of AAD, multiple studies have shown that it is likely that a constellation of factors (e.g., athero-arteriolosclerosis, hypertension, bicuspid aortic valve, idiopathic aortic root dilation, and senescence) actively participate in the weakening of the aortic wall (7,31-36). Degenerative lesions of the aortic wall affect muscles and elasticity, which can weaken the aorta; generally speaking, they affect elastic tissue in the young, and smooth muscle tissue in the elderly (3). A clinicopathologic study of 513 patients revealed that the most common pathologic histologic finding of the ascending aorta is medial degeneration in older patients (37). A slight to marked inflammatory reaction has been observed in both degenerative and inflammatory

diseases of the thoracic aorta (3). Our study showed that PCATa is also higher in AAD patients with normal coronary arteries. As mentioned above, we know that multiple factors that cause the weakening of the aortic wall are associated with inflammation. Based on our study, PCAT can be considered a marker of systemic perivascular inflammation. All of these factors may account for the high PCATa of AAD patients with normal coronary arteries.

The findings of the present study have a number of clinical implications. First, PCATa is higher in AAD patients, independent of existing atherosclerosis of the coronary artery. RCA_{PCATa} and LAD_{PCATa} values were found to be sensitive parameters for capturing perivascular inflammation in AAD patients. It may serve as an imaging biomarker to identify underlying inflammation of the vasculature in AAD patients. In addition, RCA_{PCATa} and LAD_{PCATa} values were sensitive parameters for the reaction to systemic anti-inflammatory therapies in AAD patients. It may serve as an imaging biomarker to identify the condition of inflammation reduction after systemic anti-inflammatory therapies in AAD concomitant atherosclerosis patients. Third, RCA and LAD are the preferred arteries for PCATa analysis.

Despite its promising results, the current study had several limitations. First, in this prospective study, the data were collected cross-sectionally, which means that only assumptions about possible etiological relationships can be made. Second, due to the sample size, multicenter and large-sample studies need to be conducted to verify our conclusions. Finally, the study data were obtained in routine medical care. However, laboratory inspection data on markers of dysfunction of fat surrounding coronary arteries, such as plasminogen activator inhibitor-1, were not recorded. The periaortic fat tissue is in contact with the aortic wall that can correspond to the level of periaortic inflammation. Thus, there might be a correlation between PCATa and periaortic fat tissue attenuation. More studies need to be conducted to explain it.

Conclusions

PCATa on CT was increased in patients with AAD regardless of the presence or absence of CAD. This suggests that vascular inflammation is present in AAD independent of CAD. Further research should be conducted to investigate the potential of this imaging biomarker to predict AAD and monitor patient responses to therapies for AAD.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://qims.amegroups.com/article/view/10.21037/qims-23-253/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-23-253/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and approved by the Medical Ethics Committee of Chongqing General Hospital. Informed consent was obtained from all patients.

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