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Association between spontaneous internal carotid artery dissection and perivascular adipose tissue attenuation on computed tomography angiography

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Abstract

Background: Spontaneous cervical artery dissection (sCAD) is a leading cause of ischemic stroke in young patients. Studies using high-resolution magnetic resonance imaging and positron emission tomography have suggested vessel wall inflammation to be a pathogenic factor in sCAD. Computed tomography (CT) attenuation of perivascular adipose tissue (PVAT) is an established non-invasive imaging biomarker of inflammation in coronary arteries, with higher attenuation values reflecting a greater degree of vascular inflammation.

Objectives: We evaluate the CT attenuation of PVAT surrounding the internal carotid artery ($PVAT_{carotid}$) with and without spontaneous dissection.

Methods: Single-center prospective observational study of 56 consecutive patients with CT-verified spontaneous dissection of the internal carotid artery (ICA). Of these patients, six underwent follow-up computed tomography angiography (CTA). Twenty-two patients who underwent CTA for acute neurological symptoms but did not have dissection formed the control group. Using semi-automated research software, $PVAT_{carotid}$ was measured as the mean Hounsfield unit (HU) attenuation of adipose tissue within a defined volume of interest surrounding the ICA.

Results: $PVAT_{carotid}$ was significantly higher around dissected ICA compared with non-dissected contralateral ICA in the same patients (-58.7 ± 10.2 vs -68.9 ± 8.1 HU, $p < 0.0001$) and ICA of patients without dissection (-58.7 ± 10.2 vs -69.3 ± 9.3 HU, $p < 0.0001$). After a median follow-up of 89 days, there was a significant reduction in $PVAT_{carotid}$

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around dissected ICA (−57.5 ± 13.4 to −74.3 ± 10.5 HU, $p < 0.05$), while no change was observed around non-dissected contralateral ICA (−71.0 ± 4.4 to −74.1 ± 4.1 HU, $p = 0.19$). ICA dissection was an independent predictor of $PVAT_{carotid}$ following multivariable adjustment for age and the presence of ICA occlusion.

Conclusion: $PVAT_{carotid}$ is elevated in the presence of sCAD and may decrease following the acute event.

Keywords
Computed tomography angiography, internal carotid artery, spontaneous dissection, perivascular adipose tissue

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Background

Spontaneous cervical artery dissection (sCAD) is one of the leading causes of ischemic stroke in people aged 50 years or younger.[1] Most sCAD occurs in the internal carotid artery (ICA), more than 2 cm above the bifurcation of the common carotid artery.[2] The etiology of sCAD is multifactorial and remains poorly understood, with possible constitutional factors being connective tissue disorders and genetic predisposition.[3] However, such chronic conditions do not account for all the clinical features of sCAD, and several studies have suggested infection as a potential trigger.[4,5] Consistent with this, reports have demonstrated elevated serum markers of inflammation in patients with sCAD.[6,7] Furthermore, radiological evidence of both systemic and localized inflammatory arteriopathy has been demonstrated in patients with sCAD using high-resolution magnetic resonance imaging (hrMRI) and position emission tomography–computed tomography (PET-CT).[8,9]

Assessment of the computed tomography (CT) attenuation of pericoronary adipose tissue is an established non-invasive imaging biomarker of coronary inflammation, which has been shown to predict coronary plaque progression and cardiac mortality.[10,11] Increased CT attenuation of perivascular adipose tissue (PVAT) associates with histopathologic markers of inflammation, including increased pro-inflammatory cytokines and macrophage activation.[12] Perivascular fat stranding has also been observed on coronary CT angiograms in patients with spontaneous coronary artery dissections.[13] While many studies have demonstrated the promise of CT imaging for the assessment of pericoronary adipose tissue inflammation, the clinical utility of carotid artery PVAT measurements remains unknown. Hence, we aimed to evaluate CT attenuation of PVAT surrounding the ICA ($PVAT_{carotid}$) with and without spontaneous dissection.

Patients and methods

Patients

Consecutive adult patients diagnosed with sCAD between 2011 and 2018 admitted to Clínica Alemana de Santiago (teaching not-for-profit private hospital in Chile) were prospectively included. Dissection of the ICA was confirmed by cervicocranial CTA, magnetic resonance angiography (MRA), or digital subtraction angiography (DSA) according to standard imaging criteria: a mural hematoma, aneurysmal dilation, long tapering stenosis, intimal flap, double lumen, or occlusion >2 cm above the carotid bifurcation revealing an aneurysmal dilation or long tapering stenosis after recanalization.[14] The local protocol includes cervical MRA or CTA as the first test, and in case of doubts, a confirmation with a second imaging (MRA, CTA, or DSA) was performed. Of these patients, only those with an ICA dissection and available CTA were included in this study. Patients with poor CTA image quality and who did not consent to research were also excluded. The date of dissection was estimated from the first appearance of one or more of the following symptoms or signs: acute cervical pain; local symptoms such as cervical swelling or Horner syndrome; and clinical features of cerebral ischemia. Patients with a history of related trauma were excluded. The study was approved by the local Human Research Ethics Committee (reference no. 2018-01, Scientific Ethics Committee of the Faculty of Medicine, Universidad del Desarrollo, Chile), and all patients in the sCAD cohort provided written informed consent.

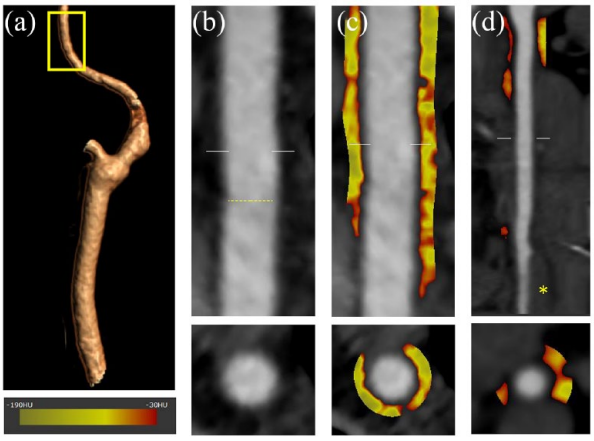
The control group consisted of patients who underwent neck CTA between 2018 and 2021 for acute neurological symptoms, which subsequently ruled out sCAD and atherosclerotic disease in the large arteries.

Clinical data analysis

Medical history, clinical characteristics, and late clinical outcomes were collected in a prospective database. Ischemic stroke was defined as a permanent neurological dysfunction caused by focal brain, spinal, or retinal infarction. Cardiovascular risk factors such as the presence of diabetes mellitus (hemoglobin $A1_c > 6.5\%$ or on diabetic medication), hypertension, smoking, hyperlipidemia, and coronary artery disease were also recorded.

All patients with sCAD were treated with antiplatelet agents (aspirin and/or clopidogrel) as per local protocol.

Figure 1. Representative computed tomography of a patient with spontaneous coronary artery dissection: (a) Three-dimensional reconstruction of the common and internal carotid arteries. (b) Curved planar reformation and cross-sectional view of a normal ICA. (c) CT attenuation measurement of perivascular adipose tissue of a normal ICA. (d) $PVAT_{carotid}$ measurement of a dissected ICA. Yellow asterisk denotes a section of mural hematoma.



Patients were assessed by their treating physician on follow-up, and outcomes, including the recurrence of ICA dissection and neurologic disability based on the modified Rankin scale, were recorded.

Imaging technique

The cervicocranial CTA studies were performed on one of the institution's CT scanners, including SOMATOM definition AS+, sensation 16, biograph 64 (Siemens Healthcare, Forchheim, Germany), and Aquilion ONE (Toshiba Medical Systems, Otawara, Japan). For the Siemens system, collimation was 128×0.6 mm, rotation time 330 ms, and tube voltage was between 100 and 140 kV. For the Aquilion ONE system, collimation was 0.5×80 mm, rotation time 500 ms, and tube voltage was between 100 and 120 kV. For each study, approximately 70 mL of non-ionic iodinated contrast was administered (iohexol 350, Omnipaque; GE Healthcare, NJ, USA). Follow-up imaging was performed within the same institution if clinically indicated.

Imaging data analysis

Evaluation of the CT attenuation of the $PVAT_{carotid}$ was performed post hoc. We adapted an established approach used in the coronary arteries.[12] An experienced independent reader (K.C.) who was blinded to the patient clinical data analyzed all common and ICA segments using semi-automated research software (Autoplaque, version 2.5; Cedars-Sinai Medical Center, Los Angeles, CA, USA). The carotid CT angiographic data set was exported to the workstation in Digital Imaging and Communications in Medicine (DICOM) format. With Autoplaque, the observer used multiplanar reformatting to generate a long-axis view of the carotid artery and manually defined the proximal and distal limits of the arterial segment of interest. A circular region of interest was placed in the aortic arch to define the "normal blood pool," as well as 10–12 control points in the arterial lumen to define the luminal centerline. The common carotid artery was segmented from its origin at the aortic arch to its termination at the carotid bifurcation. For the non-dissected ICA, we analyzed the entire length from its origin at the carotid bifurcation up until its entry through the carotid canal. For the dissected ICA, we analyzed the segment starting from its origin at the bifurcation until the start of dissection. The dissected segment was not included in the analysis due to the inability to consistently delineate the outer vessel wall for every patient. PVAT was defined as all voxels with CT attenuation between −190 and −30 Hounsfield units (HUs) located within a radial distance from the outer carotid artery vessel wall equal to the average diameter of the vessel (Figure 1); the average diameter of the common carotid artery in the whole cohort was 6 and 4 mm for the ICA. The PVAT-CT attenuation of the ICA ($PVAT_{carotid}$) was defined as the average CT attenuation within this defined volume of interest.

Statistical analysis

We performed both *within-subject* and *between-subject* analyses. Within subjects who had unilateral ICA dissections, we compared $PVAT_{carotid}$ around the dissected ICA to the contralateral non-dissected ICA. Paired *t*-tests were used to assess the mean and maximum difference in $PVAT_{carotid}$ between left- and right-sided ICAs in the same patient. Between-subject comparisons examined the $PVAT_{carotid}$ around dissected ICAs versus non-dissected ICAs in the control group. Values are given as mean ± SD. Groups were compared using the independent *t*-test for the comparison of continuous variables and the Fisher exact test for categorical variables. To examine the association of clinical and imaging characteristics with $PVAT_{carotid}$ in the entire cohort, we used linear mixed models with a random intercept to account for the within-patient clustering of vessels. To assess the predictive value of $PVAT_{carotid}$ for the presence of ICA dissection, we used mixed-effects logistic regression with adjustment for clinically relevant variables. All analyses were performed using SPSS Statistics Software (v26; IBM Corporation, Armonk, NY, USA). A two-sided *p* value of <0.05 was considered significant. All $PVAT_{carotid}$ measurements are reported in HU.

Results

Patient characteristics

A total of 56 patients with spontaneous ICA dissection and 22 control patients were included in the study (Table 1). For patients with ICA dissection, average age was 46 years and 54% were male. Fifty-two percent (n = 29) of these patients had their CTA scan within 24 h of their symptom onset, and 39% of these patients demonstrated stroke secondary to the ICA dissection. Of the patients with unilateral ICA dissections, 41% occurred on the left, 46% occurred on the right, and the remaining 13% had bilateral dissections. For patients in the control group, their average age was 40 years and 45% were male. Most of these patients (63%) had their CTA scan within 24 h of symptom onset, and 95% of these patients demonstrated concurrent stroke. The following imaging findings were described: 39 had mural hematoma, 15 pseudoaneurysms, 14 vessel occlusions, 4 intimal flap, and 1 double lumen.

For patients in the control group, average age was 40 years and 45% were male. Similarly, most of these patients (63%) had their CTA scan within 24 h of symptom onset, and 21 (95%) of these patients demonstrated concurrent stroke: 14 patients had patent foramen ovale, 1 patient had lacunar stroke, 1 patient had an associated cerebral venous thrombosis, and 5 patient's stroke etiology were undetermined after complete study.

Within-subject $PVAT_{carotid}$ comparison for unilateral ICA dissection

For patients with unilateral ICA dissections, we performed a within-subjects analysis comparing the $PVAT_{carotid}$ differences between the dissected ICA with contralateral non-dissected ICA. We found significantly elevated $PVAT_{carotid}$ around the dissected ICA versus the non-dissected ICA (mean HU difference: 10.7, $p < 0.001$) (Table 2). There was no significant difference with the $PVAT_{carotid}$ surrounding the common carotid arteries on the dissected side versus the non-dissected side (mean HU difference: 0.08, $p = 0.6$).

Between-subject $PVAT_{carotid}$ comparison of dissected and non-dissected ICA

In the between-subjects analysis of all patients with ICA dissection, we found that the dissected ICA had a significantly

Table 1. Patient demographics.

	ICA dissection, n = 56	Control, n = 22	p
Clinical characteristics			
Age, years	46 (42–54)	40 (36–47)	0.09
Male	30 (54%)	10 (45%)	0.62
Hypertension	12 (21%)	3 (14%)	0.54
Diabetes mellitus	0	1 (5%)	0.28
Hypercholesterolemia	13 (23%)	2 (9%)	0.21
Smoking	17 (30%)	8 (36%)	0.6
Coronary artery disease	0	0	NS
Prior carotid artery dissection	3 (5%)	0	0.55
Days between symptom onset to initial CT scan	1 (0–5.5)	1 (0–1)	NS
ICA diameter, mm	4 (4–5)	4 (3.25–4)	NS
Concurrent stroke	22 (39%)	21 (95%)	<0.001
Affected vascular territory			
Left MCA	9 (16%)	7 (32%)	0.13
Right MCA	13 (23%)	7 (32%)	0.56
Left PCA	0	1 (5%)	0.28
Basilar	0	3 (14%)	0.02

(Continued)

Table 1. (Continued)

	ICA dissection, n = 56	Control, n = 22	p
Etiology of stroke			
Ischemic	22 (39%)	20 (91%)	<0.001
Venous infarction	0	1 (5%)	NS
ICA dissection side			
Left	23 (41%)	—	
Right	26 (46%)	—	
Bilateral	7 (13%)	—	
Dissection features			
Pseudoaneurysm	15 (29%)	—	
Vessel occlusion	14 (25%)	—	
Intimal flap	4 (7%)	—	
Double lumen	1 (2%)	—	
Mural hematoma	39 (70%)	—	
CT acquisition parameters			
CT Tube voltage			
100 kV	6 (11%)	0	0.17
120 kV	36 (64%)	22 (100%)	<0.001
140 kV	14 (25%)	0	<0.01
CT scanner vendor			
Siemens Healthcare	51 (91%)	21 (95%)	NS
Toshiba Medical Systems	5 (9%)	1 (5%)	NS

Data are median (IQR), and count (n) and percentage (%).
ICA: internal carotid artery; CT: computed tomography; MCA: middle cerebral territory; PCA: posterior cerebral artery; IQR: interquartile range.

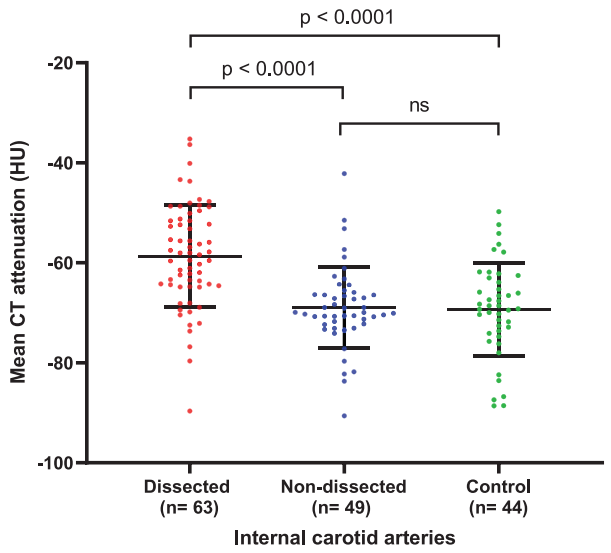
Table 2. Within-subject analysis of patients with unilateral ICA dissection.

PVATcarotid (HU)	Dissected side	Non-dissected side	p
Common carotid artery	−74.8 ± 9.1	−74.8 ± 8.4	0.60
ICA	−58.0 ± 10.0	−68.9 ± 8.0	<0.001

Data are mean (SD).
ICA: internal carotid artery; PVAT, perivascular adipose tissue; HU, Hounsfield unit.

higher $PVAT_{carotid}$ compared with the non-dissected ICA (−58.7 ± 10.2 vs −68.9 ± 8.1 HU, $p < 0.0001$; Figure 2). The dissected ICA also showed a significantly higher $PVAT_{carotid}$ in comparison with the control group patients who did not have ICA dissection (−58.7 ± 10.2 vs −69.3 ± 9.3 HU, $p < 0.0001$). Furthermore, the non-dissected ICA demonstrated no significant difference compared with the control group patients (−68.9 ± 8.1 vs −69.3 ± 9.3 HU, $p = 0.33$).

Figure 2. Dot plot showing the between-subject comparison of $PVAT_{carotid}$ of dissected and non-dissected ICA of patients with spontaneous dissection, as well as ICA of patients without dissection. Line indicate means (SD).



Follow-up post-ICA dissection

Patients with sCAD were followed-up at a median interval of 108 days (interquartile range (IQR): 49–189 days) after their initial CTA imaging (Table 3). A majority (n = 46, 82%) of the patients who had sCAD maintained a good functional outcome (modified Rankin score of 0–2); death occurred in 3 (5%) patients and a recurrent ICA dissection in 1 (2%) patient. Patients in the control group were followed-up at a median interval of 204 days (IQR: 183–220 days), with most of the patients (n = 18, 82%) maintaining a good functional outcome.

Of patients with ICA dissection, 6 (11%) underwent clinically indicated follow-up CT carotid angiograms at a median interval of 89 days (IQR: 78–107). One patient (17%) had bilateral ICA dissection and the remainder (83%) had unilateral ICA dissection. For the dissected ICA, the follow-up scans demonstrated a significant reduction in their $PVAT_{carotid}$ compared to the baseline scan (Figure 3(a)). For the non-dissected ICA, there was no significant difference in $PVAT_{carotid}$ between baseline and follow-up scans (Figure 3(b)).

Multivariable analysis on determinants of $PVAT_{carotid}$ and ICA dissection

Mixed-effect multivariable linear regression analysis showed ICA dissection to be independently associated with $PVAT_{carotid}$ (β coefficient: 10.80, $p < 0.001$), following adjustment for clinical and imaging characteristics (Table 4). Conversely, in mixed-effect multivariable logistic regression analysis adjusted for age, gender, and risk factors, an increasing $PVAT_{carotid}$ (β coefficient: 0.125, $p < 0.001$) and stroke in the ipsilateral cerebral territory (β coefficient: −3.05, $p = 0.003$) were associated with ICA dissection (Table 5).

Discussion

Using clinically routine CTA, we investigated the relationship between $PVAT_{carotid}$ of ICA with and without spontaneous dissection. We found that $PVAT_{carotid}$ is significantly elevated with the presence of spontaneous dissection before and after adjusting for known confounding factors. On

Table 3. Follow-up of patients with spontaneous ICA dissection and the control group.

	ICA dissection, n = 56	Control, n = 22	*p*
Days between initial CT imaging and final follow-up assessment	108 (49–189)	204 (183–220)	<0.0001
Outcomes			
Modified Rankin Scale			
0–2	46 (82%)	18 (82%)	0.18
3–6	7 (13%)	4 (18%)	0.18
Death	3 (5%)	0	0.55
Recurrent ICA dissection	1 (2%)	—	NS
Recurrent stroke	2 (4%)	0	NS

Data are median (IQR), and count (n) and percentages (%).
ICA: internal carotid artery; CT: computed tomography; IQR: interquartile range.

Figure 3. Follow-up comparison of $PVAT_{carotid}$ of dissected (a) and non-dissected (b) ICA of patients with spontaneous dissection.

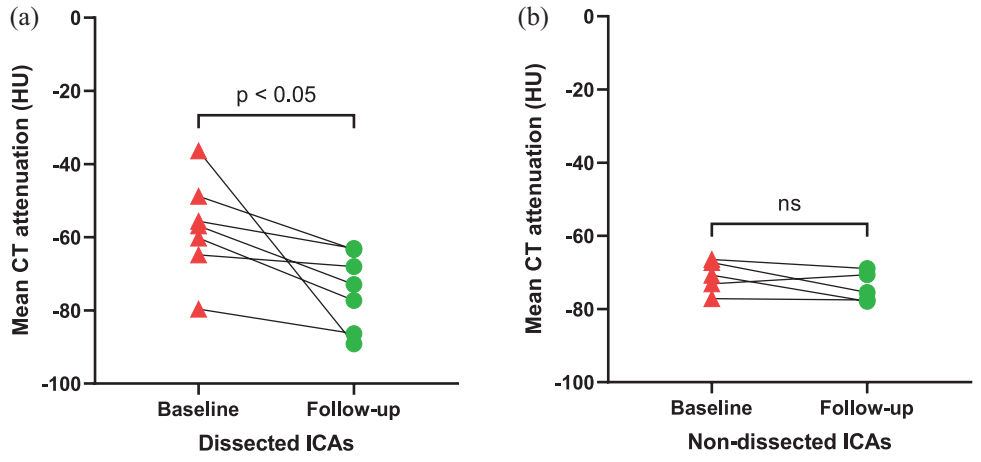


Table 4. Linear mixed model assessing the association of clinical and imaging characteristics with $PVAT_{carotid}$ in the entire cohort.

	β coefficient[a]	95% CI, lower	95% CI, upper	*p* value
Constant[b]	−79.77	−132.41	−27.13	**<0.01**
Age (y)	−0.13	−0.47	0.21	0.44
Male gender	−1.80	−7.42	3.83	0.53
Hypertension	−1.72	−8.39	4.95	0.61
Current smoking	−0.81	−6.82	5.21	0.79
History of prior ICA dissection	−4.02	−18.12	10.14	0.57
Stroke in the ipsilateral cerebral territory	−1.38	−7.67	4.91	0.66
ICA diameter (cm)	−0.067	−3.60	1.70	0.48
Days between symptom onset and CT scan	−0.063	−0.31	0.15	0.50
CT tube voltage (kV)	0.02	−0.20	0.24	0.86
Dissection of the ICA	10.80	6.44	15.17	**<0.001**
Dissection involving bilateral ICA	−2.58	−10.89	5.74	0.54
CT angiogram dissection features				
Pseudoaneurysm	−4.55	−11.01	1.91	0.16
Intimal flap	−3.02	−13.94	7.91	0.58
Double lumen	−4.83	−26.23	16.56	0.65
Vessel occlusion	3.25	−4.32	10.83	0.40

PVAT, perivascular adipose tissue; CI: confidence interval; ICA: internal carotid artery; CT: computed tomography; HU, Hounsfield unit.
Bold face *p* value indicates statistical significance.
[a]Dependent variable: PVAT attenuation (HU).
[b]Denotes a 47-year-old male with no ICA dissection.

follow-up imaging, we observed a significant reduction in $PVAT_{carotid}$ of the dissected ICA, while no significant differences with the non-dissected ICA. Given the recent data supporting the association between inflammation with pericoronary and periaortic adipose tissue attenuation,[12,15,16] our findings suggest there is increased inflammation in the

Table 5. Mixed-effect logistic regression model assessing predictors of the presence of ICA dissection in the entire cohort.

	β coefficient	95% CI, lower	95% CI, upper	*p* value
Age	0.009	−0.057	0.074	0.79
Male gender	0.152	−0.907	1.211	0.78
Hypertension	−0.475	−1.445	0.494	0.33
Dyslipidaemia	−0.085	−0.993	0.823	0.85
Current smoking	−0.102	−1.200	0.995	0.85
Concurrent stroke	−0.052	−0.871	0.767	0.90
Stroke in the ipsilateral cerebral territory	−3.053	−5.068	−1.037	**0.003**
$PVAT_{carotid}$	0.125	0.061	0.188	**<0.001**

ICA: internal carotid artery; CI: confidence interval; PVAT, perivascular adipose tissue.
Bold face *p* value indicates statistical significance.

adipose tissue surrounding dissected ICA. Based on our findings, the relatively lower attenuation of the contralateral non-dissected ICA indicates the inflammation to be localized and likely independent of any generalized systemic process. The observation that the elevated $PVAT_{carotid}$ attenuation resolved within weeks in our subset of patients who had serial CTA imaging suggests the transient nature of the underlying pathological process.

Our findings are consistent with a previous observational study using hrMRI and PET-CT in patients with sCAD.[8] Among 37 patients with sCAD, the majority of patients had either increased [^{18}F]-fluorodeoxyglucose accumulation (on PET-CT) or perivascular contrast enhancement (on hrMRI) at the site of dissection. Consistent with our follow-up imaging, a repeat hrMRI 3–6 months later showed complete resolution of contrast enhancement and perivascular oedema. The utility of CT-derived pericarotid adipose tissue attenuation was first investigated in patients with carotid atherosclerotic disease, showing that the attenuation of PVAT surrounding ICA ipsilateral to a stroke or transient ischemic attack is significantly higher compared to ICA that is asymptomatic.[17] Elevated PVAT attenuation was also found to be significantly associated with the presence of intraplaque hemorrhage (IPH) in the carotid artery.[18] IPH is a vulnerable plaque feature associated with plaque progression, rupture, and increased risk of cerebrovascular ischemic events.[19]

The vascular wall interacts with the surrounding adipose tissue in a complex and likely bidirectional manner.[20] Histological studies have shown that exposure of PVAT to pro-inflammatory cytokines suppresses the differentiation of pre-adipocytes while triggering their proliferation, resulting in numerous smaller adipocytes with fewer intracellular lipid droplets.[12] This creates a gradient of differing PVAT density with a lipid-rich/less-aqueous phase adjacent to a non-diseased vessel to a lipid-poor/more-aqueous phase adjacent to an inflamed artery. Routine CT employs an HU scale of attenuation (reduction in signal), which can be used as a non-invasive measure of adipose tissue characteristics.[21] The link between biopsy-proven PVAT inflammation and CT attenuation was demonstrated in coronary arteries in a landmark study by Antonopoulos et al.[12] This surrogate measure of coronary inflammation has been shown to predict plaque progression and cardiac mortality in patients undergoing cardiac CT and to differentiate stages of coronary artery disease.[10,11,22]

To our knowledge, this is the first observational study of $PVAT_{carotid}$ in patients with spontaneous ICA dissection. While markers of systemic inflammation including serum C-reactive protein and leucocytosis have been associated with sCAD, it provided a poor association with local vascular biological processes.[6] Existing imaging techniques such as hrMRI and PET-CT are limited by cost, availability, and complex imaging protocols. Quantification of $PVAT_{carotid}$ does not require extra protocol within routine carotid CTA and may represent a dynamic imaging biomarker of vascular inflammation. Adapting similar techniques from studies in the coronary arteries, we utilized semi-automated software for $PVAT_{carotid}$ assessment which is highly reproducible and minimizes observer bias.[23] The robustness of this approach will facilitate future prospective studies across institutions and different CT scanning hardware. Furthermore, it is important to note that there are currently no studies on a direct relationship between $PVAT_{carotid}$ and inflammation; future studies with histology or PET imaging are needed to validate this association. If confirmed, our findings lend support to the pathogenic role of inflammation in spontaneous ICA dissection and the ability to assess such inflammation using routine CT angiogram. However, it remains uncertain whether the perivascular

inflammation is a cause of spontaneous ICA dissection or a result of the inflammatory response caused by tissue damage secondary to vessel disruption.

Several limitations of our study should be noted. First, this is an observational study from a single center, and hence our findings need to be validated in a large multicentre cohort. Second, it was not possible for the assessment of $PVAT_{carotid}$ to be blinded to the ICA pathology as it is visible on CT. Third, CTA scans were performed using different scanners and acquisition parameters, which may have affected the HU attenuation values of $PVAT_{carotid}$ and limited between-subject comparisons. The influence of different CTA image acquisition parameters and CT scanners on PVAT quantification should be investigated in future studies. Finally, the sample size of this study does not allow a valid interpretation of the clinical, therapeutic, and prognostic relevance of $PVAT_{carotid}$, and we do not have other local biomarkers or histologic validation of inflammation in the $PVAT_{carotid}$.

Conclusion

Our findings lend support to the hypothesis that inflammation is a pathologic factor in ICA spontaneous dissection and can be quantified noninvasively on routine CTA imaging. Furthermore, imaging and histopathological investigations are necessary to examine the association between $PVAT_{carotid}$ and inflammation.

Data availability

Unpublished data are available upon reasonable request by a qualifying investigator.

Declaration of conflicting interests

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