# **CTA Assessment of Coronary Inflammation**

Subjects: Radiology, Nuclear Medicine & Medical Imaging Contributor: Jeremy Yuvaraj

Coronary computed tomography angiography (CTA) provides a means of mapping inflammatory changes to both epicardial adipose tissue (EAT) and pericoronary adipose tissue (PCAT) as independent markers of coronary risk.

atherosclerosis coronary artery disease computed tomography coronary angiography

coronary inflammation

adipose tissue

epicardial adipose tissue

pericoronary adipose tissue

# **1. Introduction**

In recent decades, atherosclerosis has become well recognised as a disease of chronic vascular inflammation. Where the excess accumulation of lipid in the arterial wall was thought to be the most prominent driver of plaque formation, a strong body of evidence highlights the critical effect of vascular inflammatory mechanisms in plaque formation and morphology, as well as their contribution to the onset of major coronary events. The poor localisation of traditional systemic biomarkers to the coronary vasculature has led to exploration and discovery of suitable alternative methods of quantifying inflammatory risk using non-invasive coronary computed tomography angiography (CTA) imaging.

### 2. Coronary Computed Tomography Angiography (CTA) Assessment of Cardiac Adipose Tissue as a Marker of Coronary Inflammation

#### 2.1. EAT Quantification

CT attenuation of adipose tissue reflects morphological derangements of adipocytes exposed to the effects of local vascular inflammation. Coronary CTA readily quantifies EAT volume and density as independent markers of adverse cardiometabolic risk each bearing associations with CAD (Figure 1) <sup>[1][2]</sup>. Increased EAT volume is a predictor of the presence of CAD, acute MI and 'high-risk' CAD phenotypes <sup>[2][3][4]</sup>. Likewise, EAT attenuation has been associated with CAD, although the nature of this association demonstrates significant heterogeneity. Factors such as coronary calcification <sup>[5][6][7][8][9]</sup>, coronary events <sup>[1][5]</sup> and statin therapy <sup>[10][11]</sup> all producing seemingly disparate effects on EAT attenuation. This may be owed to the anatomy of EAT as a depot, which inherently encompasses a wide range of adipocytes varying in proximity to the vessel wall and, accordingly, exposure to coronary inflammation.



**Figure 1.** Epicardial adipose tissue (EAT) shown in purple on axial view of coronary computed tomography angiography (CTA). EAT in patient without coronary artery disease (CAD) shown in left panel (**A**), and EAT in patient with CAD shown in right panel (**B**).

#### 2.2. Pericoronary Adipose Tissue and Fat Attenuation Index

Pericoronary adipose tissue (PCAT) resides directly adjacent to the coronary adventitia, and harbours the most profound exposure to inflammatory mediators that may arise from the vasculature. Accordingly, it is emerging as a prominent non-invasive metric of coronary inflammation. The current primary definition of PCAT on coronary CTA is adipose tissue residing within a volume that extends to an orthogonal distance equivalent to the diameter of the target vessel. PCAT is typically measured around select lesions, or in the proximal segments of the major coronary arteries, particularly the right coronary artery (RCA) due to the low number of side branches, abundance of adipose tissue and uniformity of luminal diameter from its ostial to distal segments [12][13] (Figure 2).

The pericoronary fat attenuation index (FAI) is an AI-driven quantitation of adipose tissue attenuation computationally adjusted for a range of additional factors, such as CT technical parameters and adipocyte morphology <sup>[12][13]</sup>. Increased pericoronary FAI has been associated with inflammatory mediators <sup>[12]</sup>, high-risk coronary plaque <sup>[14]</sup> and coronary event endpoints <sup>[13][15]</sup>. It also reflects the effects of anti-inflammatory therapy <sup>[16][17]</sup>.



**Figure 2.** Pericoronary adipose tissue (PCAT) shown in cross-sectional (**A**,**B**) and longitudinal (**C**,**D**) views of the right coronary artery (RCA) on coronary computed tomography angiography (CTA). PCAT in RCA without plaque represented in the left panels (**A**,**C**), and PCAT in RCA with calcified and non-calcified plaque represented in the right panels (**B**,**D**). Colour map describes spectrum of adipose tissue attenuation values in Hounsfield units (HU), ranging from –190 HU (yellow) to –30 HU (red), with higher attenuation values indicating inflammatory changes.

#### 2.3. 'Crude' PCAT Attenuation and CAD

Numerous observational studies aside from those listed above have together cultivated a mounting level of evidence into the nature of coronary inflammation shown through PCAT attenuation, albeit without the adjustments afforded by the algorithm that characterises FAI. A range of methodologies have been previously employed to study and classify PCAT on CT. While these studies are informative, a key limitation is the absence of a clear demarcation between pericoronary and 'non-pericoronary' fat, and thus the influence of 'non-perivascular' fractions of EAT in analysis cannot be dismissed. Many recent studies, however, have adopted the standardised approach described previously <sup>[12][13]</sup> and while these studies may assess the 'unadjusted' or 'crude' form of PCAT

attenuation, the employment of a consistent methodology has been conducive to the generation of results that are more readily reproducible and cross-verifiable <sup>[18]</sup>.

PCAT attenuation studies have shown that coronary inflammatory changes may occur incrementally with the burden of CAD and coronary events <sup>[19]</sup>. PCAT attenuation is also increased around high-risk coronary plaque <sup>[20]</sup> <sup>[21]</sup> and in male patients <sup>[22][23][24][25]</sup>.

#### 2.4. PCAT Assessment of Haemodynamically-Significant Lesions

It is important to note that high-risk coronary plaque phenotypes are often non-obstructive in nature, and thus differential pathophysiological mechanisms may be pertinent to plaque morphology compared to plaque-related stenosis. Nevertheless, several studies comparing PCAT attenuation with measures of coronary flow and myocardial ischaemia denote the potential role of vascular inflammation in coronary stenosis <sup>[26][27][28]</sup>.

#### 2.5. PCAT Assessment in Non-Atherosclerotic Disease States

Therefore, both adjusted and 'crude' PCAT assessment have demonstrated distinctly increased coronary inflammation in patients with developing high-risk lesions or major coronary events, collectively providing further validation for the potential clinical utility of this technique. In addition to these studies on orthodox coronary atherosclerosis, PCAT attenuation has been explored in a number other coronary and inflammatory disease states, including vasospastic angina <sup>[29][30]</sup>, atherosclerotic intraplaque cholesterol crystals <sup>[31]</sup>, and spontaneous coronary artery dissection <sup>[32]</sup>.

#### 2.6. Technical Parameter Influence in PCAT Assessment

A number of observational studies have evaluated the impact of scan parameters, particularly contrast enhancement, on PCAT attenuation. Given the nature of coronary CTA as one of the first-line imaging modalities with wide usage in assessment of suspected CAD, PCAT attenuation studies have thus far analysed primarily contrast-enhanced CT acquisitions in both retrospective and prospective cohorts. The current literature demonstrates PCAT attenuation is increased in contrast-enhanced scans compared to non-contrast scans <sup>[33]</sup>. This is an important consideration as the LAD has reportedly higher PCAT attenuation at the ostium than the distal vessel, which corresponds to decreasing luminal diameter and contrast volume <sup>[34][35]</sup>.

## **3. Current Limitations and Future Directions**

Current knowledge on PCAT attenuation as an imaging biomarker of inflammation is, therefore, ever-expanding, but is not without some limitations. Differences in PCAT attenuation owed to epicardial and overall adiposity <sup>[33]</sup>, as well as due to sex, call for future studies to adjust for these potential patient-specific confounders. While the impact of specific scan parameters on PCAT attenuation has been increasingly evaluated, validation of 'crude' PCAT assessment across different scanners has yet to be explored. As discussed, PCAT attenuation has been assessed previously using a range of methodologies, and this has been to a large extent accounted for via the use of

validated per-patient and per-lesion forms of assessment. However, there is some heterogeneity as to the degree to which plaque within the RCA may affect the per-patient assessment of PCAT in this vessel [19][24]. Moreover, the present definition of PCAT (adipose tissue within a distance equivalent to the vessel diameter) includes an inherently larger volume of adipose tissue in the RCA than in its counterpart vessels. The consistent luminal diameter of the RCA ensures that adipocytes within up to approximately 2-3 mm from the vessel wall are considered PCAT throughout the vessel's passage; conversely, the LAD and LCx are encased in less adipose tissue overall, and decreases in luminal diameter mean a progressively decreasing volume of adipose tissue volume in the mid-to-distal segments of these vessels falls within the classification of PCAT. In theory, the broader inclusion of adipose tissue around the RCA indicates PCAT assessment in this vessel may include adipocytes that are distinctly less 'inflamed', while PCAT in the LAD or LCx would consist only of adipocytes that are most proximal to the vessel wall. In this regard, alternative methodologies consistent with the theoretical framework outlined previously would be worthy of consideration. For example, a recent study <sup>[25]</sup> evaluated per-patient PCAT attenuation as the mean attenuation of adipose tissue within a region of 1 mm thickness around all three coronary vessels, and thus a consistent volume of PCAT being assessed for each vessel was ensured. Nevertheless, extensive validation and a wealth of literature highlights the utility of the current methods of PCAT assessment at the per-patient and per-lesion level  $\left[\frac{12}{13}\right]$ .

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