Perivascular fat attenuation index stratifies the cardiac risk associated with high-risk plaque features on coronary computed tomography angiography

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Background: Qualitative high-risk plaque (HRP) features detected on coronary computed tomography angiography (CCTA) are associated with increased risk of adverse cardiac events. Coronary inflammation is a key determinant of plaque progression and instability and can now be captured on routine CCTA as inflammation-induced changes in perivascular adipose tissue composition, detectable by the perivascular Fat Attenuation Index (FAI).

Purpose: To explore the ability of perivascular FAI phenotyping to stratify the cardiac risk associated with the presence of adverse plaque morphology on routine CCTA.

Methods: This was a post-hoc analysis of the CRISP-CT (Cardiovascular RISk Prediction using Computed Tomography) study, which involved 3912 patients (mean age 55.7±13.7 years, 41.1% females) undergoing clinically-indicated CCTA in two centres (Erlangen, Germany & Cleveland, USA). Perivascular FAI mapping was performed around the proximal 10-50 mm of the right coronary artery and defined as the weighted mean attenuation of the perivascular adipose tissue, as previously validated. HRP features were defined as the presence of =1 of the following: positive remodelling, low-attenuation plaque, spotty calcification or napkin-ring sign (A). Cox regression models (adjusted for age, sex, epicardial fat volume and coronary artery disease [=50% stenosis]) were used to explore the association between FAI, HRP, and future major adverse cardiac events (MACE: defined as the composite of cardiac mortality and non-fatal myocardial infarction).

Results: At baseline the prevalence of HRP and high FAI (=70.1 Hounsfield Units, as previously validated) was 23.6% (n=923) and 24.3% (n=952) respectively. Over a median follow-up period of 5.6 years (25th-75th percentile: 4.0-7.0 years) there were 91 confirmed MACE. Patients with both HRP features (HRP+) and high FAI (FAI+) had a 6.3-fold (P<0.001) higher adjusted risk of MACE compared to individuals with neither of these risk features (HRP-/FAI-) (B). Furthermore, patients without HRP features but with high FAI (HRP-/FAI+) had a 4.9-fold (P<0.001) higher adjusted risk of MACE compared to the reference (HRP-/FAI-) group. However, among patients with low FAI, there was no significant difference in the prospective risk of MACE between HRP+ and HRP- patients (P=0.87).

Conclusion: FAI is associated with an increased risk of adverse events in both patients with and without high-risk plaques, highlighting coronary inflammation as a major determinant of plaque vulnerability, independent of adverse plaque morphology. Non-invasive characterization of coronary inflammation using CCTA-derived FAI
can improve risk stratification by supplementing the traditional anatomical assessment of the coronary vasculature with a functional marker of disease activity.