remodelling, low-attenuation plaque, spotty calcification or napkin-ring sign. The association with future incidence of major adverse cardiac events (cardiac mortality or non-fatal myocardial infarction) was assessed using Cox regression models (adjusted for age, sex, epicardial fat volume and coronary artery disease ≥50% stenosis).

**Results** 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 of 5.6 years (25th-75th percentile: 4.0–7.0 years) 91 MACE were recorded. Patients with both HRP features and high FAI (FAI+/HRP+) had a 6.3-fold higher adjusted risk of MACE compared to those with neither of these risk features (HRP-/FAI-). Furthermore, patients without HRP features but with high FAI (HRP-/FAI+) had a 4.9-fold higher adjusted risk of MACE compared to the reference (HRP-/FAI-) group.

**Conclusion** FAI is a stronger predictor of cardiac mortality than high-risk plaques, and there is additive predictive value between plaque morphology and coronary inflammatory burden. There is need for tools to provide comprehensive risk assessment based on CCTA, by extracting, weighting and interpreting all available information from these scans.

**OP4** PERIVASCULAR FAT ATTENUATION INDEX MAPPING AROUND THE RIGHT AND LEFT CORONARY ARTERY INDEPENDENTLY PREDICT CARDIAC MORTALITY

**Introduction** Coronary inflammation induces spatial changes in perivascular adipose tissue (PVAT) composition, which can now be detected as spatial changes in PVAT attenuation quantified by the perivascular Fat Attenuation Index (FAI) on coronary computed tomography angiography (CCTA). We assessed the ability of perivascular FAI mapping around the right and left coronary territories to independently stratify future cardiac risk.

**Methods** This was a post-hoc analysis of the CRISP-CT (Cardiovascular Risk Prediction using Computed Tomography) study that included 3912 patients with an average age of 55.7 years (standard deviation [SD]: 13.7 years). Perivascular FAI mapping was performed around the proximal right (RCA) and left anterior descending (LAD) coronary arteries. FAI was calculated based on the weighted average attenuation of PVAT using the CaRi-HEART algorithm, as previously described. The independent association with future incidence of cardiac death was assessed in adjusted Cox regression models.

**Results** Over a median follow-up period of 5.6 years, 74 cardiac deaths were recorded. The cross-sectional association between perivascular FAI around the RCA and LAD at baseline was found to be moderate (R=0.36, P<0.001). Interestingly, both higher peri-RCA and peri-LAD FAI values were independently associated with a higher risk of cardiac mortality, following multivariable adjustment for age, sex, hypertension, hyperlipidemia, diabetes mellitus, smoking, epicardial adipose tissue volume and coronary artery disease (≥50% stenosis) (adjusted Hazard Ratio [95% confidence interval] of 1.70 [1.23–2.35], P=0.001 & 1.55 [1.12–2.14], P=0.008 per 1 SD increments, respectively)

**Conclusion** Perivascular FAI around the coronary arteries is characterised by anatomical/ regional variability, and its values are affected by the coronary segment interrogated. Non-invasive characterization of coronary inflammation using CCTA-derived FAI should include a comprehensive assessment of both coronary arteries in order to better characterize the inflammatory residual risk of each patient.

**OP5** PERICORONARY ADIPOSE TISSUE DENSITY IS greater in TAKAYASU ARTERITIS THAN ATHEROSCLEROSIS AND IS ASSOCIATED WITH CORONARY ARTERIAL INFLAMMATION MEASURED BY 68GA-DOTATATE PET

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**Introduction** Pericoronary adipose tissue (PCAT) density is associated with vascular inflammation, but its nature is not fully understood. We compared PCAT density with clinical and molecular imaging markers of inflammation.

**Methods** PCAT density was quantified in patients with Takayasu arteritis (TAK), coronary artery disease (CAD), and age and gender-matched healthy controls from cardiac CT images using semi-automated software (AutoPlaque). In TAK patients, PCAT density was also compared to the Indian Takayasu Clinical Activity Score (ITAS). In CAD patients, PCAT density was compared to maximum tissue-to-blood ratio (TBRmax) from motion-corrected 68Ga-DOTATATE PET, using image registration software (FusionQuant), and aortic 18F-fluorodeoxyglucose (FDG) PET. Imaging was acquired during clinical care or prior research. 68Ga-DOTATATE is an experimental marker of vascular inflammation that binds macrophage somatostatin receptor-2.

**Results** 60 patients were included (TAK, n=20; CAD, n=20; healthy, n=20). Mean PCAT density varied significantly among the three groups (TAK: -74.00 ±SD 11.92 Hounsfield unit [HU]; CAD: -80.39 ±SD 10.9 HU; healthy controls: -83.85 ±SD 10.07 HU; p<0.0001). C-reactive protein was greater in TAK than CAD patients (TAK: 25.2 ±SD 16.1 mg/L; CAD: 2.5 ±SD 1.73 mg/L, p=0.04). PCAT density was significantly associated with ITAS (r=0.61, p=0.004) in TAK patients, and coronary 68Ga-DOTATATE TBRmax (rho=0.31, p<0.001) in CAD patients. No significant patient-level confounders were identified. PCAT density was not statistically associated with aortic 18F-FDG in CAD patients, or subcutaneous (pre-sternal) adipose tissue density in either disease group.

**Conclusion** PCAT density could be a useful, non-PET marker of coronary arterial inflammation and disease activity in both TAK and CAD patients.