Results Over a median follow-up of 8 years, 120 participants (13.1%) died. Overall, lower 3D LVEF, and higher 3D iEDV and iESV were associated with increased risk of all-cause mortality (table 1a 1b). However, sex modified the associations between volume indices and LVRI with all-cause mortality; hence we present sex-specific results in table 1. After multi-variable adjustment for age, ethnicity, systolic blood pressure, antihypertensive medications, cholesterol: HDL ratio, diabetes, body mass index, smoking and history of coronary heart disease, per SD change, decrease in LVRI and LVEF, and increase in volumes were independently associated with increased risk of mortality in men (figure 1, table 1a 1b). However, associations between volume indices, LVRI and, to a lesser extent, LVEF, and mortality were reversed in women (figure 1, table 1a 1b): fully adjusted hazard ratios (95% CI): EDV: men: 1.22 (1.0, 1.4), women: 0.55(0.28, 1.1), ESV: men: 1.31(1.10, 1.60), women: 0.59(0.34, 1.0), LVRI: men: 0.79(0.65, 0.98), women: 1.40(0.88, 2.2), LVEF: men: 0.81(0.67, 0.97), women: 1.31(0.69, 2.5). Although numbers of women are small, effect sizes indicated that lower volumes and higher LVRI were associated with higher risk of all-cause mortality in women.

Conclusion In the general population, 3DE-derived LVEF, volumes and remodelling predict the long-term risk of all-mortality, independent of clinical confounders and cardiovascular risk factors strongly in men. However the direction of association for volumes and remodelling measures is reversed in women suggesting that sex-differences in cardiac structure and function seem to be associated differently with the risk of all-cause mortality.

Conflict of Interest N/A

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18F-SODIUM FLUORIDE POSITRON EMISSION TOMOGRAPHY PREDICTS PROGRESSION OF CORONARY CALCIFICATION

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Introduction Combined positron emission tomography and computed tomography (PET-CT) using the radiotracer 18F-sodium fluoride (18F-NaF) to detect microcalcification provides imaging of both coronary artery anatomy and disease activity simultaneously. While recent studies have suggested that 18F-NaF activity may help identify high-risk coronary atherosclerosis, the role of 18F-NaF uptake in predicting progression of coronary atherosclerosis is unknown. In this study, we aimed to investigate the relationship between baseline coronary arterial 18F-NaF activity and the subsequent progression of coronary arterial calcification in patients with clinically stable coronary artery disease.

Methods Patients with clinically stable, multivessel coronary artery disease underwent combined 18F-NaF PET-CT and CT coronary calcium scoring at baseline with repeat CT coronary calcium scoring at one year. Coronary arterial PET uptake was analysed qualitatively and semi-quantitatively in diseased vessels by measuring maximum tissue-to-background ratio (TBRmax) – defined as the maximum standardised uptake value in a plaque divided by the mean blood pool activity

Abstract 4 Figure 1

Abstract 4 Table 1 Progression of coronary calcification in patients with and without increased ¹⁸F-NaF activity at baseline

PROGRESSION OF CALCIFICATION AT ONE YEAR	All patients (n=183)	PET positive (n=116)	PET negative (N=67)	P value
Change in Agatston Score (AU)	70[22-143]	98[39-166]	35[7-93]	<0.001
Change in Calcium Volume (mm ³)	60[16-121]	82[29-149]	33[3-82]	<0.001
Change in Calcium Mass (mg)	14[4-35]	22[7-42]	6[1-21]	<0.001
Change in Average Density (mg/mm ³)	0.01[-0.002-0.02]	0.01[0.001-0.02]	0.01[-0.004-0.12]	0.78

measured in the right atrium. Coronary calcification was quantified by measuring calcium mass (mg), volume (mm³), average calcium density (mg/mm³) and total Agatston score (AU).

Results One hundred and eighty-three patients who underwent baseline and repeat imaging at one year were included in the study (81% male, median age 66). Of these participants, 116 (63%) had evidence of increased 18F-NaF activity in at least one vessel. Patients with increased 18F-NaF uptake had a higher total calcium score (524[242–1091] AU), volume (491[247–984], mm³) mass (99[46–212] mg) and average calcium density (0.20[0.18–0.23] mg/mm³) at baseline compared to patients without increased uptake (136[55–361] AU, 131[54–343] mm³, 24[11–69] mg, 0.18[0.16–0.20] mg/mm³; $P < 0.001$ for all), and demonstrated greater progression of coronary calcification (table 1). In patients with an increase in calcium score at one year ($n = 160$), there was a moderate correlation between baseline TBRmax and change in total calcium score ($R = 0.45$, $R^2 = 0.20$; $p < 0.001$) and change in total calcium mass ($R = 0.50$, $R^2 = 0.25$; $p < 0.001$) at one year (figure 1). There was no correlation between baseline TBRmax and change in average calcium density ($p = 0.43$).

Conclusion Coronary PET-CT using 18F-NaF identifies patients with a higher calcification burden and predicts progression of coronary arterial calcification at one year.

Conflict of Interest none

5 INTER INDIVIDUAL VARIATIONS IN LDL-C AND CAROTID PLAQUE LIPID CONTENT WITH STATIN AND THE IMPACT OF PLAQUE BURDEN ON PLAQUE LIPID REDUCTION

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Introduction Little is known about the variations in atherosclerotic lipid content when juxtaposed to changes LDL-c in response to statin treatment. T2 mapping is a recently-validated magnetic resonance imaging (MRI) technique that can precisely quantify carotid plaque lipid content and its reduction following initiation of high-intensity statin treatment.¹

Abstract 5 Figure 1

Abstract 5 Figure 2

Moreover, this highly-sensitive technique may add insights into the potential differential response of carotid plaque lipid content in ‘normal’ versus ‘abnormal’ carotid arteries.

Methods Statin-naïve patients presenting with acute myocardial infarction who underwent paired carotid MRI coupled with venous blood sampling were included. Using a modified classification of American Heart Association (AHA) of carotid vessel wall, 2 slices of each individual corresponding to type I and II were categorised into the ‘normal’ group while other slices corresponding to type III onward of AHA classification were combined together as the ‘abnormal’ group.

Results 552 patients were included in this study. Mean age was 63 ± 13 years and 75% were male. The median EF was 50% (40–55). 21% of patients were classified as reduced EF, 41% as mrEF, and 38% with preserved EF. There were significant differences across three subgroups (reduced EF, mrEF, and preserved EF respectively) in age (66 ± 13 , 62 ± 13 , 62 ± 12 , $P = 0.017$), troponin value [4606 (1589–10000), 2819 (1059–6347), 902 (329–2869), ($P < 0.001$)] and proportion of patients with normal renal function (69%, 82%, 88%, $P < 0.001$) and anterior STEMI (70%, 45%, 20%, $P < 0.001$). There was a stepwise increase in the primary endpoint according to the EF category 6.2%, 18.5%, 34.8% $P < 0.001$ (Figure: Primary endpoint in STEMI patients stratified according to their baseline ejection fraction); hazard ratio (HR) for mrEF versus preserved EF 3.25 (95% CI 1.74 to 6.05), $P < 0.001$, and HR for mrEF versus reduced EF 0.48 (95% CI 0.31 to 0.74, $P = 0.001$). The difference was derived from each of the primary components i.e. death (3.3%, 10.1%, 17.4%, $P < 0.001$), re-admission with heart failure (2.9%, 6.2%, 13%, $P = 0.002$), and ventricular arrhythmia/ ICD implantation (0%, 4.8%, 12.2%, $P < 0.001$). There were differential independent predictors of primary endpoint between mrEF and preserved EF (Table 18&2). Normal kidney function was associated with better clinical outcomes in both EF categories.

When the cohort was stratified according to the structure of carotid arteries, the group with ‘normal’ carotid slices had no significant changes in carotid lipid% compared to baseline [3.1% (0.5 – 7.1) vs. 3.7% (0.8 – 6.6), $P = 0.086$]. On the other hand, there was a significant reduction in carotid lipid% in the ‘abnormal’ group when compared to baseline [9.7% (4.1 – 18.4) vs. 6.2% (2.3 – 12.8), $P < 0.001$].

Similarly, there was no significant changes in carotid vessel wall volume after three months of statin treatment in the ‘normal’ group [33.0 mm³ (28.5 – 40.0) vs. 33.3 mm³ (28.8 – 38.6), $P = 0.61$]. In contrast, a significant reduction in carotid vessel wall volume was detected between baseline and