heterogeneity when it comes to degree of hypertrophy and progression to heart failure. Underlying interplay of metabolism may explain this variability. We sought to determine if a gradient of metabolic remodelling exists across the spectrum of AS studying its relationship with stenosis severity and degree of hypertrophy.

Methods 74 asymptomatic AS participants, mild (n=18), moderate (n=38), severe (n=18); and 13 healthy controls underwent CMR imaging for cardiac function, energetics (PCr/ATP) and triglyceride content (MTG). Participants were divided into quartile groups of LV wall thickness (LVWT) and peak aortic valve gradient (AVG) to study relationship between cardiac metabolism and LV structure and function.

Results There was a stepwise deterioration in LV structure and function across both the LVWT and AVG quartile groups (p <0.05). PCr/ATP was reduced in Q2 (1.43 ± 0.13 vs 1.80 ± 0.14 in Q1 controls, p=0.05) with a further progressive decline (Q4, 1.39 ± 0.14, p=0.02). MTG was elevated in Q2 (1.52 ± 0.84% vs 1.25 ± 0.70% in Q1, p = 0.032). AS groups had impaired systolic longitudinal strain, which was related to reduction in PCr/ATP (R 0.219, p 0.03). PCr/ATP was also strongly associated with myocardial fibrosis (p 0.01).

Conclusion A gradient of myocardial energetic deficit and stenosis exists across the spectrum of AS, notably we show that these metabolic changes precede irreversible LV remodelling and subclinical dysfunction.

OP5 IMPACT OF DIABETES MELLITUS ON THE QUANTITATIVE ASSESSMENT OF CORONARY ATHEROSCLEROSIS IN SCOT-HEART TRIAL

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Objective To determine the natural history of coronary 18F-fluoride uptake over 12 months in patients with either advanced stable coronary artery disease or a recent myocardial infarction.

Methods Patients with established multivessel coronary artery disease and either stable disease or a recent acute myocardial infarction underwent coronary 18F-fluoride positron emission tomography and computed tomography angiography which was repeated at either 3, 6 or 12 months. Coronary 18F-fluoride uptake was assessed in each vessel by measuring the coronary microcalcification activity (CMA). Coronary calcification was quantified by measuring calcium score, mass, and volume.

Results Fifty-nine patients had stable coronary artery disease (median age 68 years, 93% male) and fifty-two patients had a recent myocardial infarction (median age 65 years, 83% male). Reflecting the greater burden of coronary artery disease, baseline CMA values were higher in those with stable coronary artery disease. Coronary 18F-fluoride uptake (CMA>0) was associated with higher baseline calcium scores (294 [116–483] versus 72 [8 -222] AU; P<0.001), and more rapid progression of coronary calcification scores (39 [10–82] versus 12 [1–36] AU/year; P<0.001), compared to the absence of uptake (CMA=0). Coronary 18F-fluoride uptake did not markedly alter over the course of 3, 6 or 12 months in patients with either stable coronary artery disease or a recent myocardial infarction.

Conclusion Coronary 18F-fluoride uptake is associated with severity and progression of coronary artery disease but does not undergo rapid dynamic change in patients with stable or unstable coronary artery disease.

OP4 TEMPORAL CHANGES IN CORONARY 18F-FLUORIDE PLAQUE UPTAKE IN PATIENTS WITH CORONARY ATHEROSCLEROSIS

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Objective To determine the natural history of coronary 18F-fluoride uptake over 12 months in patients with either advanced stable coronary artery disease or a recent myocardial infarction.

Methods Patients with established multivessel coronary artery disease and either stable disease or a recent acute myocardial infarction underwent coronary 18F-fluoride positron emission tomography and computed tomography angiography which was repeated at either 3, 6 or 12 months. Coronary 18F-fluoride uptake was assessed in each vessel by measuring the coronary microcalcification activity (CMA). Coronary calcification was quantified by measuring calcium score, mass, and volume.

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Conclusion Coronary 18F-fluoride uptake is associated with severity and progression of coronary artery disease but does not undergo rapid dynamic change in patients with stable or unstable coronary artery disease.