

$\pm 1$  vs  $-20\% \pm 2$ ,  $p=0.02$ ). There were significant negative correlations between CFVR and basal septal ( $r = -0.7$ ,  $p=0.003$ ), mid-septal ( $r = -0.5$ ,  $p=0.037$ ) and global T1 times ( $r = -0.6$ ,  $p=0.012$ ) – figure 1. Basal septal T1 times were significantly elevated in subjects with CMD – median 1309ms (IQR 1301-1313) vs 1292ms (IQR 1281-1295),  $p=0.028$ . There was a trend towards increased mid-septal and global T1 times in the CMD group – figure 2 and table 2. There was no difference in T2 times between the groups.

**Conclusions** This is the first study to show that, similar to HCM, increased myocardial fibrosis is associated with reduced CFVR in ESRD. Although causation cannot be demonstrated in this study, it raises the fascinating question of whether myocardial fibrosis is ‘the chicken or the egg’ in this inverse relationship. Further mechanistic studies are needed to confirm this association and to determine which is the primary pathology. It is likely that the interplay between myocardial fibrosis and CMD contributes to the significant cardiac mortality seen in ESRD.

**Conflict of Interest** None to declare

160

#### THE IMPACT OF EXERCISE AND DIET INTERVENTION ON LEFT ATRIAL FUNCTION IN TYPE 2 DIABETES: RESULTS FROM A RANDOMISED STUDY

<sup>1</sup>Aseel Alfuhied, <sup>1</sup>Gaurav Gulsin, <sup>1</sup>Emer Brady, <sup>2</sup>Kelly S Parke, <sup>1</sup>Lavanya Athithan, <sup>2</sup>Joseph Henson, <sup>2</sup>Emma Redman, <sup>3</sup>Thomas Yates, <sup>3</sup>Melanie Davies, <sup>1</sup>Anna-Marie Marsh, <sup>1</sup>Gerry McCann, <sup>1</sup>Anvesha Singh. <sup>1</sup>University of Leicester, Leicester, UK; <sup>2</sup>NIHR Leicester Biomedical Research Centre, University of Leicester; <sup>3</sup>University Hospitals of Leicester NHS Trust

10.1136/heartjnl-2021-BCS.157

**Introduction** The effects of low-energy diet or exercise on cardiovascular function in younger adults with type 2 diabetes (DIASTOLIC) study showed a significant improvement in left ventricle peak early diastolic strain rate in response to a 12-week programme of aerobic exercise in patients with type-2 diabetes (T2D). The impact of exercise and a low-calorie meal replacement plan (MRP) on left atrial (LA) function in T2D has not yet been explored. We investigated the effect of lifestyle intervention on LA volumetric and strain parameters by cardiac magnetic resonance (CMR) imaging.

**Methods** The DIASTOLIC study was a prospective, randomised, open-label, blind endpoint trial. Obese participants with T2D (aged 18–65 years) were randomized to a 12-week intervention of: aerobic exercise training or low energy ( $\approx 810$  kcal/day) MRP. CMR was performed at baseline and week-12. Images were analysed using Medis v3.1. LA strain and strain rate (LAS/SR) were assessed using Feature Tracking (QStrain v2.0), corresponding to LA reservoir (LAS/SR-r), conduit (LAS/SR-cd), and booster pump (LAS/SR-bp) using 4- and 2-chamber standard steady-state free precession cine images, and average values calculated. LA volumes (LAV) were measured on 4 a d 2-chamber cine images and LA emptying fraction (LAEF) was calculated using biplane area-length method (QMass v8.1) for total, passive and active EF.

**Results** 45 participants with T2D completed the trial and had analysable LA cine images (22 exercise and 23 MRP). There were no significant changes in the standard assessment of LA volumetric function (LAV/LAEF) measured on CMR or in LV filling pressure (E/e') measured on echocardiography, in either group. In the MRP group, there were significant reductions in BMI (4.8 kg/m<sup>2</sup>), mean systolic blood pressure (SBP)

(13mmHg), and a significant increase in LAS-r and LAS-bp ( $29.9 \pm 7.0$  to  $32.3 \pm 7.0$ ,  $p=0.036$  and  $14.6 \pm 5.3$  to  $17.2 \pm 3.7$ ,  $p=0.034$ ) (see Table). The exercise arm showed a small reduction in BMI (0.8kg/m<sup>2</sup>), no significant change in BP and a significant improvement in LASR-bp ( $-1.19 \pm 0.3$  to  $-1.32 \pm 0.4$ ;  $p=0.041$ ).

**Conclusion** A low-calorie MRP led to significant weight loss and improved SBP, with associated improvement in LA reservoir and contractile function on CMR strain assessment. LAS could detect early LA reverse remodelling post-lifestyle intervention in young adults with T2D despite no change in volumetric measurements.

**Conflict of Interest** None

161

#### 18F-SODIUM FLUORIDE POSITRON EMISSION TOMOGRAPHY IN ACUTE AORTIC SYNDROME

<sup>1</sup>Maaz Syed, <sup>2</sup>Alexander Fletcher, <sup>2</sup>Samuel Debono, <sup>1</sup>Rachael Forsythe, <sup>2</sup>Michelle Williams, <sup>3</sup>Marc Dweck, <sup>1</sup>Adriana Tavares, <sup>1</sup>Mark Macaskill, <sup>1</sup>Anoop Shah, <sup>1</sup>Martin Denvir, <sup>4</sup>Kelvin Lim, <sup>4</sup>William Wallace, <sup>1</sup>Jakub Kaczynski, <sup>1</sup>Tim Clark, <sup>5</sup>Stephanie Sellers, <sup>4</sup>Neil Masson, <sup>4</sup>Orwa Falah, <sup>4</sup>Roderick Chalmers, <sup>4</sup>Andrew Tambyraja, <sup>2</sup>Edwin van Beek, <sup>2</sup>David Newby. <sup>1</sup>BHF Department for Cardiovascular Sciences, University of Edinburgh, Edinburgh, UK; <sup>2</sup>Centre for Cardiovascular Sciences, University of Edinburgh; <sup>3</sup>University of Edinburgh; <sup>4</sup>NHS Lothian; <sup>5</sup>Centre for Heart Lung Innovation, University of British Columbia

10.1136/heartjnl-2021-BCS.158

**Background** Acute aortic syndrome is a catastrophic condition characterised by medial degeneration and cellular destruction within the aortic wall. 18F-Sodium fluoride (18F-NaF) positron emission tomography (PET) detects microscopic calcification as a marker of disease activity. This proof-of-concept study aims characterise 18F-NaF PET in patients with acute aortic syndrome.

**Methods** Aortic tissue obtained from patients with acute aortic syndrome was stained using von Kossa's stain for calcium-phosphate complexes and then exposed to 18F-sodium fluoride to confirm radiotracer binding to microcalcification. Next, patients with aortic dissection or intramural haematomas and healthy controls underwent 18F-NaF PET/CT and CT angiography of the aorta. A threshold of 12 weeks since diagnosis was used to classify patients to ‘recent’ or ‘prior’ acute aortic syndrome groups. Peak aortic 18F-NaF uptake was corrected for background blood pool activity to obtain a most-diseased segment tissue-to-background ratio (MDS TBRmax). Radiotracer binding was compared with aortic size in a linear regression model and major adverse aortic events (aortic rupture, aorta-related death or aortic repair) in a proportional hazards Cox survival analysis.

**Results** Aortic 18F-NaF uptake co-localized with histologically defined regions of microcalcification ( $n=15$ ). Patients with acute aortic syndrome had increased 18F-NaF binding compared to healthy controls (TBRmax  $2.02 \pm 0.42$  ( $n=47$ ) vs  $1.36 \pm 0.39$  ( $n=20$ ) respectively,  $p<0.001$ ). Peak radiotracer uptake occurred at the site of intimal disruption ( $+27.5\%$  compared to the proximal aorta,  $p<0.001$ ). 18F-NaF binding to the false lumen was associated with aortic growth ( $+7.1$  mm/yr,  $p=0.011$ ) and uptake in the outer aortic wall was associated with major adverse aortic events (hazard ratio 8.6 [95% CI, 1.1-68.1],  $p=0.041$ ) in patients with recent acute aortic syndrome.

**Conclusion** 18F-NaF PET/CT uptake was increased in patients with acute aortic syndrome at sites of disease activity. Radiotracer binding was associated with aortic growth and clinical

events.  $^{18}\text{F}$ -NaF PET-CT holds promise as a non-invasive marker of disease severity and future risk in patients with acute aortic syndrome.

**Conflict of Interest** None

## 162 MULTI-MODALITY IMAGING IN SURVIVORS OF COVID-19

<sup>1</sup>Trisha Singh, <sup>1</sup>Shruti Joshi, <sup>1</sup>Nick Spath, <sup>1</sup>Lucy Kershaw, <sup>1</sup>Andy Baker, <sup>1</sup>Helen Jordan, <sup>2</sup>Gaurav Gulsin, <sup>3</sup>Michelle Williams, <sup>3</sup>Edwin van Beek, <sup>2</sup>Jayanth Arnold, <sup>1</sup>Semple Scott, <sup>3</sup>David Newby, <sup>1</sup>Marc Dweck, <sup>2</sup>Gerry McCann. <sup>1</sup>University of Edinburgh, Edinburgh, UK; <sup>2</sup>University of Leicester; <sup>3</sup>Centre for Cardiovascular Sciences, University of Edinburgh

10.1136/heartjnl-2021-BCS.159

**Background** Widespread abnormalities of the myocardium have been reported in patients with COVID-19. However, these patients often have substantial co-morbidities and it is essential to understand whether cardiac abnormalities represent pre-existing disease or are the consequence of COVID-19.

**Objective** To determine the contribution and cardiac impact of co-morbidities in patients who have recovered from COVID-19.

**Methods** In a prospective observational study, adult patients hospitalized with confirmed COVID-19 were recruited from the Edinburgh Heart Centre between May and November 2020 and compared to healthy and co-morbidity-matched volunteers. Patients underwent gadolinium and manganese-enhanced magnetic resonance imaging and coronary computed tomography angiography.

**Results** Twenty-three patients ( $54 \pm 11$  years, 20 male) who recovered from COVID-19 were recruited. Half ( $n=11$ , 48%) required admission to the intensive care unit and a third ( $n=7$ , 31%) received non-invasive or invasive ventilation. Patients had a high prevalence of known cardiovascular disease

( $n=18$ , 78%), associated risk factors ( $n=11$ , 45%) and coronary artery disease ( $n=8$ , 35%). Compared with younger healthy volunteers ( $n=10$ ), myocardial native T1 values ( $1202 \pm 25$  versus  $1162 \pm 27$  ms,  $P=0.008$ , **figure 1**) and extracellular volume fraction ( $31.9 \pm 1.7$  versus  $29.8 \pm 0.5$  %,  $P=0.001$ , **figure 1**) were higher with no differences in manganese uptake. Compared to co-morbidity-matched volunteers ( $n=20$ ), there were no differences in native T1 values ( $1202 \pm 25$  versus  $1196 \pm 39$  ms,  $P=0.61$ , **figure 1**), extracellular volume fraction ( $31.9 \pm 1.7$  versus  $31.0 \pm 0.5$  %,  $P=0.11$ ), presence of late gadolinium enhancement or manganese uptake. These findings remained irrespective of COVID-19 disease severity, presence of concomitant myocardial injury or coronary artery disease.

**Conclusions** Patients who have recovered following hospitalization with COVID-19 have no evidence of a major excess in myocardial injury or dysfunction compared to co-morbidity-matched volunteers. The presence of co-morbidities likely explains many of the previously reported myocardial abnormalities.

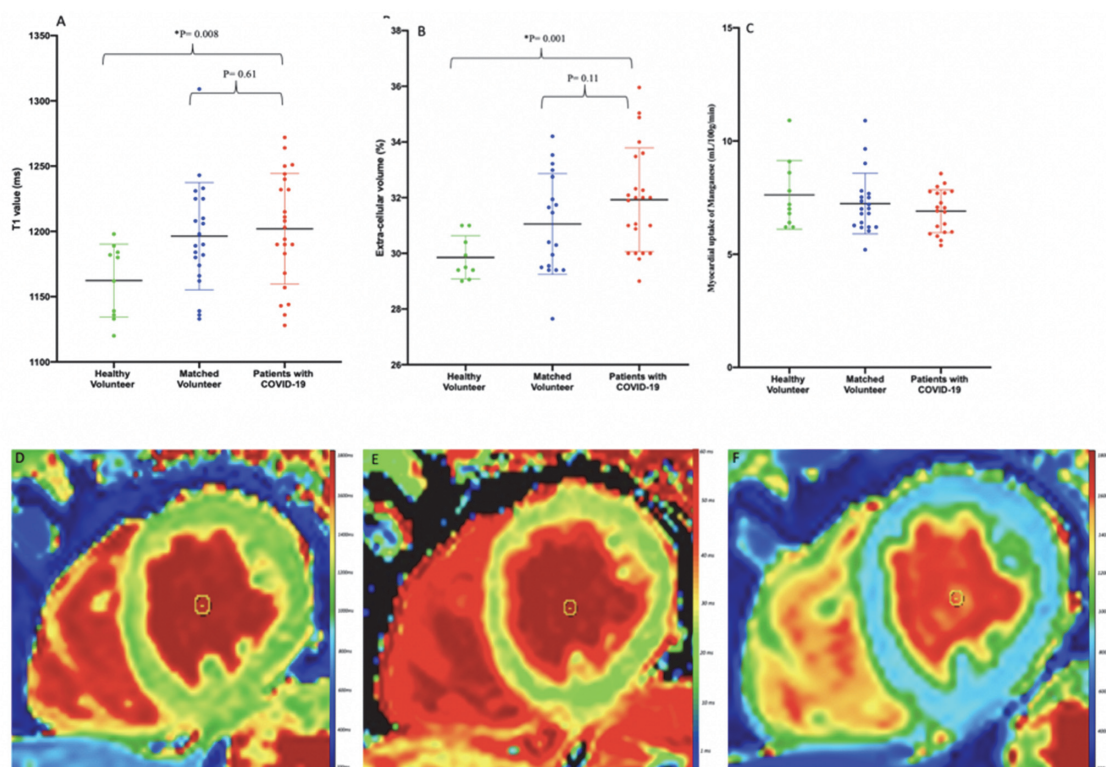
**Conflict of Interest** None

## 163 FIRST IN VIVO PRETARGETED PET IMAGING OF ATHEROSCLEROSIS WITH ANTIBODIES AGAINST FORMS OF MODIFIED LIPOPROTEINS

<sup>1</sup>Cinzia Marceddu, <sup>1</sup>Adam Hartley, <sup>1</sup>Mikhail Caga-Anan, <sup>1</sup>Samata Pandey, <sup>1</sup>Yasmin Morris, <sup>1</sup>Dorian Haskard, <sup>2</sup>Jan Passchier, <sup>1,2</sup>Ramzi Khamis. <sup>1</sup>Imperial College London, London, UK; <sup>2</sup>Invivo London

10.1136/heartjnl-2021-BCS.160

Atherosclerosis is a cardiovascular disease initiated by the deposition of Low Density Proteins (LDL) within the intima and



Abstract 162 Figure 1