

5 CARDIOVASCULAR MAGNETIC RESONANCE (CMR) IN TRANSTHYRETIN AMYLOID CARDIOMYOPATHY – A STUDY OF NATURAL HISTORY AND TREATMENT RESPONSE

¹Rishi K Patel*, ²Ana Martinez-Naharro, ²Yousuf Razvi, ²Adam Ioannou, ^{1,2}Aldostefano Porcari, ²Liza Chacko, ²James Brown, ²Daniel Knight, ²Tushar Kotecha, ⁴Charlotte Manisty, ⁴James C Moon, ²Helen J Lachmann, ²Ashutosh Wechalekar, ²Carol Whelan, ²Lucia Venneri, ⁵Peter Kellman, ²Philip N Hawkins, ²Julian D Gillmore, ²Marianna Fontana. ¹Royal Free London NHS Trust, London; ²National Amyloidosis Centre, Division of Medicine, University College London, Royal Free Campus, Rowland Hill Street, London NW3 2PF; ³Center for Diagnosis and Treatment of Cardiomyopathies, Cardiovascular Department, Azienda Sanitaria Universitaria Giuliano-Isontina (ASUGI), University of Trieste, Italy; ⁴Barts Heart Centre, The Cardiovascular Magnetic Resonance Imaging Unit, and the Inherited Cardiovascular Diseases Unit, St Bartholomew's Hospital, West Smithfield, London EC1A 7BE; ⁵National Heart, Lung and Blood Institute, National Institutes of Health, Bethesda, MD, USA

10.1136/heartjnl-2024-BSCMR.3

Introduction Cardiovascular magnetic resonance (CMR) and extracellular volume (ECV) mapping can quantify amyloid burden in patients with cardiac amyloidosis. At present, there is a paucity of data on tracking changes in amyloid deposition in both untreated and treated cohorts with transthyretin (ATTR) disease. Our aim was to use CMR and ECV mapping to characterise the natural history of untreated ATTR amyloidosis, measure the response to treatment with patisiran, and correlate ECV with changes in structural and functional cardiac parameters.

Materials and Methods In total, 119 untreated patients and 70 patients treated with patisiran were assessed with serial biomarkers, echocardiography and CMR with ECV mapping at baseline, 1 year, and 2 years where possible. CMR response was categorized by changes in ECV as either: disease progression ($\geq 3\%$ increase), stable ($< 3\%$ change) or regression ($\geq 3\%$ decrease).

Results In untreated patients, 57% had disease progression at 1 year which significantly increased to 70% at 2 years ($p < 0.05$). Mean increase in ECV of 4.0% after 1 year and 6.8% after 2 years was observed, and associated with significant worsening in biomarkers, ventricular wall thickness and global longitudinal strain at each timepoint. Left ventricular (LV) ejection fraction, indexed stroke volume and LV mass significantly worsened after 2 years. Following treatment with patisiran, no significant difference in mean ECV was observed at both timepoints. Disease stability was observed in 66% and 64% of patients and ECV regression observed in 17% and 21% of patients after 1 and 2 years of treatment with patisiran respectively. In all patients followed up at 1 year ($n=160$), reduction in ECV was not associated with a change in biomarkers or imaging parameters. ECV stability or progression was associated with significant worsening in wall thickness, LV volumes and biventricular longitudinal function.

Conclusion CMR with ECV mapping demonstrates that cardiac amyloid deposition increases over time in ATTR amyloidosis and is associated with worsening cardiac structure and function. Although treatment with patisiran is associated with disease stability in the majority, stabilisation of cardiac structural and functional parameters is only observed in those with ECV regression, highlighting the importance of achieving a significant treatment response.

Acknowledgements I would like to thank Dr Ana Martinez-Naharro for her contributions to this project, the supervision of both Professor Julian Gillmore and Professor Marianna Fontana, the staff at the CMR unit at the NAC and finally

the patients who underwent CMR throughout the study period.

6 COMPARISON BETWEEN CMR AND A FULLY AUTOMATED CT TECHNIQUE IN THE ASSESSMENT OF EPICARDIAL ADIPOSE TISSUE IN TYPE 2 DIABETES

¹Anamika Banerjee*, ¹Abhishek Dattani, ¹Jian L Yeo, ¹Alice Cowley, ¹Sarah L Ayton, ¹Emer M Brady, ²Aparna Deshpande, ³Damini Dey, ¹Gaurav S Gulsin, ¹Gerry P McCann. ¹Department of Cardiovascular Sciences, University of Leicester and the National Institute for Health Research Leicester Biomedical Research Centre, Glenfield Hospital, Leicester, UK; ²Department of Imaging Services, Glenfield Hospital, University Hospitals of Leicester, Leicester UK; ³Biomedical Imaging Research Institute, Cedars-Sinai Medical Center, Los Angeles, CA USA

10.1136/heartjnl-2024-BSCMR.4

Introduction Dysregulated epicardial adipose tissue (EAT) may play an important role in the development of heart failure in Type 2 Diabetes (T2D). EAT can be measured using computed tomography (CT) but there are limited data directly comparing this with CMR-measured EAT, which offers unique advantages. This study aimed to assess CT- and CMR-measured EAT in people with T2D.

Materials and Methods In this single-centre, prospective study of people with and without T2D, participants underwent phenotyping including an ECG-gated non-contrast CT and a multiparametric stress CMR at 3T. CT total EAT volume was determined using a fully automated deep learning algorithm incorporating the whole heart. CMR EAT area was quantified using a 4-chamber long axis cine image at end-systole obtained using a steady-state free precession technique. Contouring was performed by a single observer using two methods to measure EAT area: 1) Contour around the right ventricle starting from the atrioventricular groove up to the left ventricular apex (RV-EAT), and 2) contour around both ventricles (BV-EAT). Values were indexed to height. Twenty scans underwent repeat assessment by a second observer for inter-observer variability.

Results 93 people with T2D (age 64.0 ± 6.5 , males 60.2%, HbA1c 7.0 [6.5–7.9]%) and 32 controls (age 59.0 ± 7.5 , males 65.6%, HbA1c 5.5 [5.3–5.7]%) were included in this analysis. The T2D group had significantly higher body mass index compared to controls and greater proportion of people with hypertension and hypercholesterolaemia. T2D participants had greater indexed EAT compared to controls as measured by CT (81.3 ± 32.7 vs. 48.4 ± 27.4 mL/m, $P < 0.001$) and CMR (RV-EAT: 3.28 ± 1.52 vs. 2.65 ± 1.44 cm²/m, $P = 0.042$; BV-EAT: 4.97 ± 1.99 vs. 4.07 ± 1.92 cm²/m, $P = 0.027$). CT-EAT positively correlated with CMR measured RV-EAT (Whole cohort: $r = 0.599$, $P < 0.001$; T2D: $r = 0.552$, $P < 0.001$; Controls: $r = 0.712$, $P < 0.001$) and BV-EAT (Whole cohort: $r = 0.688$, $P < 0.001$; T2D: $r = 0.649$, $P < 0.001$; Controls $r = 0.769$, $P < 0.001$). Inter-observer variability analysis demonstrated moderate reliability for RV-EAT (ICC: 0.636, Bias: 1.153, LLOA: -4.197, ULOA: 6.503) and BV-EAT (ICC: 0.567, Bias: 1.522, LLOA: -4.292, ULOA: 7.336).

Discussion

Conclusion CMR-measured RV-EAT and BV-EAT have moderate correlation with CT-measured EAT and have moderate inter-observer reliability. Further work needs to assess association between CMR-measured EAT with cardiac structure, function and outcomes.

Acknowledgements