and ~350 ms for LGE-MRI, 90° excitation pulse, slice thickness=1.0 mm, FOV=25.6 × 25.6 mm, matrix size=128 × 128) as described.³

Results At 1 hour post MI, viable myocardium was enhanced in MEMRI, allowing early delineation of the occlusion zone as 41%±8% of the myocardium, whereas only subtle enhancement was seen on LGE-MRI, yielding a significantly lower value of 12%±2% (p=0.03, Figure 1). At ~24 hours post MI, the MEMRI measure of infarct size remained constant (41%±5%) whilst LGE-MRI significantly increased to a level comparable with MEMRI (37%±3%). Figure 2 shows a direct comparison of MEMRI and LGE-MRI acquired in the same animal at 22 and 27 hours after MI, respectively, with matching histological TTC staining for infarct size.

Discussion Effected myocytes rapidly stop internalising Mn under ischemic conditions, allowing early delineation of the occlusion zone. The membrane rupture that underlies LGE-MRI occurs later, meaning LGE-MRI underestimates the occlusion zone during the first hours post MI.

Conclusions The present study shows MEMRI can quantify final infarct size earlier than LGE-MRI. This provides a sensitive approach which could be used as an early measure of cell death and myocardial viability when assessing the efficacy of new drugs which target acute MI.

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6

RV FUNCTION DETERIORATES EARLIER THAN LV FUNCTION AND PREDICTS ADVERSE CARDIOVASCULAR OUTCOMES

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Background The current assessment of hypertrophic cardiomyopathy (HCM) places emphasis on left ventricular (LV) function which is usually preserved in early disease. There are limited data on right ventricular (RV) assessment in HCM, progressive functional changes, and impact of RV dysfunction on clinical outcomes.

Objectives To examine the natural history of RV functional changes (ejection fraction and strain) and investigate its prognostic role in HCM.

Methods 311 patients (mean age 52±15 years) with HCM and preserved LV function (ejection fraction; EF ≥50%) underwent cardiac magnetic resonance (CMR) imaging and were compared to age- and sex- matched healthy controls (n=30). In 71 patients, follow-up CMR imaging was further undertaken at a median interval of 5.3 years. All patients were followed up for a composite endpoint of adverse

Abstract 6 Figure 1 Kaplan Meier curves shows that in HCM patients with impaired RVEF (<55%), there is a reduced freedom from ventricular arrhythmia (A) and composite cardiac outcomes (B). In patients with the lowest quartile of RV longitudinal strain (>-16.6%), there is a reduced freedom from ventricular arrhythmia* (C) and composite cardiac outcomes* (C) compared to other patients (* comparison between first two quartiles and lowest quartile were significant, # comparison between top three and lowest quartile were significant)

cardiovascular outcomes, including new-onset atrial fibrillation, ventricular arrhythmia, hospitalisation due to heart failure or embolic events, or cardiovascular death.

Results HCM patients exhibited lower RV ejection fraction (RVEF) and global peak systolic strains (radial/circumferential/longitudinal) on feature-tracking analysis of cine images than healthy controls. On follow up CMR imaging, RVEF, peak RV circumferential and longitudinal strains decreased over time while LVEF remained preserved. All patients were followed up clinically for a median duration of 4.4 years. Both reduced RVEF and RV longitudinal strain were independent predictors of ventricular arrhythmias and composite cardiovascular endpoint (arrhythmia, cardiac hospitalisation and cardiovascular death), after adjusting for baseline NYHA class, medication use, maximal LV wall thickness, and late gadolinium enhancement. Patients with RVEF <55% had a 3-fold increase in ventricular arrhythmias (HR 3.03, 95% CI 1.63 to 5.66, **figure 1A**) and a 2-fold increase in composite cardiac events (HR 2.05, 95% CI 1.23 to 3.41, **figure 1B**). Similarly patients in the lowest quartile of RV longitudinal strain (>-16.3%) demonstrated a higher risk of ventricular arrhythmias (HR 2.20, 95% CI 1.20 to 4.01, **figure 1C**) and composite cardiac events (HR 2.49, 95% CI 1.61 to 3.84, **figure 1D**) compared to others.

Conclusions Despite preserved LVEF, RVEF and strain decline over time in HCM. RVEF and strain predict the occurrence of adverse cardiovascular outcomes and may have a role in prognostic risk stratification in HCM.

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DIFFUSION TENSOR CARDIOVASCULAR MAGNETIC RESONANCE IN CARDIAC AMYLOIDOSIS

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Introduction Cardiac amyloidosis is a disease of infiltration, typically by light chains (AL) or transthyretin (ATTR). We studied cardiac amyloidosis using diffusion tensor cardiovascular magnetic resonance (DT-CMR), which is the only non-invasive tool able to assess cardiac microstructure *in-vivo*.

Methods and results Biphasic STEAM DT-CMR was successfully performed in 20 cardiac amyloidosis patients (10 AL, 10 ATTR) and 10 age and sex matched controls. Compared with controls, amyloid patients had higher mean diffusivity (MD); median [IQR] $1.46 [1.36-1.54] \times 10^{-3} \text{ mm}^2/\text{s}$ vs $1.15 [1.03-1.20] \times 10^{-3} \text{ mm}^2/\text{s}$ ($p < 0.001$), shown in figure 1A. MD correlated with T1 ($R^2 = 0.76$, $p < 0.001$) and extracellular volume (ECV) ($R^2 = 0.47$, $p = 0.004$).

Abstract 7 Figure 1 Diffusivity biomarkers differ significantly in cardiac amyloid

Mean diffusivity is significantly higher in cardiac amyloid ($p < 0.001$) at both cardiac phases, reflecting increased freedom of diffusion in the expanded myocardium (panel A). In panel B, FA is significantly reduced ($p < 0.001$) at both cardiac phases, reflecting the greater disorganisation of the infiltrated myocardium

Amyloid patients had lower fractional anisotropy (FA) compared to controls; $0.43 [0.39-0.45]$ vs $(0.55 [0.52-0.60])$, $p < 0.001$, as shown in figure 1B. FA inversely correlated with T1 ($R^2 = 0.59$, $p < 0.001$). Amyloid patients had elevated diastolic E2A and reduced E2A mobility (both < 0.001).

Segmental analysis for co-location of FA and MD with ECV amyloid infiltration showed good correlation (both $p < 0.001$). Example maps are shown in figure 2. There was no significant difference in MD, FA or E2A mobility between amyloid subtypes.

Conclusion In cardiac amyloidosis, DT-CMR characterises the microstructural effects of infiltration, and increases in MD show good correlation with increases in ECV. The novel insights from DT-CMR offer a deeper understanding of pathophysiological mechanisms in cardiac amyloidosis. With further development, DT-CMR might offer a gadolinium free assessment of amyloid burden, which is of particular value in renal failure.