

TR=3.04/1.11 ms, 18 inversion times at consecutive R waves, 10⁰ excitation pulse, slice thickness=1.0 mm, FOV=25.6 × 25.6 mm, matrix size=128 × 128) as described.²

Results As soon as 1 hour after MI, R1 values increased in Viable-MI tissue compared with AAR-MI (p<0.0001) and naïve controls. R1 values continued to rise in the Viable-MI at 2 and 3 hours (p=0.02, p=0.01. Figure 1 and 2). When the same animals were imaged 2 days post-MI, R1 values were still significantly higher in the Viable-MI tissue compared with AAR-MI tissue (p=0.03). However, Viable-MI tissue had similar R1 to naïve hearts, while R1 in the infarcted AAR-MI was lower than of the naïve myocardium (p=0.03. Figure 1 and 2).

Discussion Acutely after ischemic injury a large increase in R1 (reflecting increased Mn²⁺ uptake) occurred in Viable-MI myocytes, likely due to elevated catecholamine levels acutely post-MI; increased cardiac work and thus increased Ca²⁺/Mn²⁺ uptake. By 2 days the catecholamine storm has passed and R1 levels in the surviving myocardium normalise, while Mn uptake in the dead infarct region was reduced due to lack of functional myocytes.

Conclusions T1-Mapping Manganese-enhanced-MRI offers a valuable *in vivo* tool for optimisation of the many emerging pharmacological and biological interventions which aim to modulate Ca²⁺ homeostasis acuity after MI.

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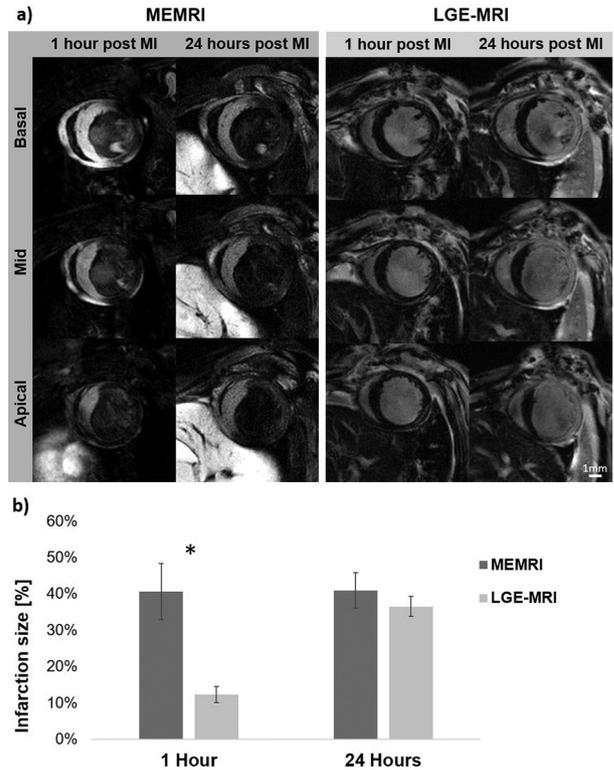
5 MANGANESE ENHANCED MRI CAN QUANTIFY MYOCARDIAL INFARCT SIZE EARLIER THAN GADOLINIUM ENHANCED MRI

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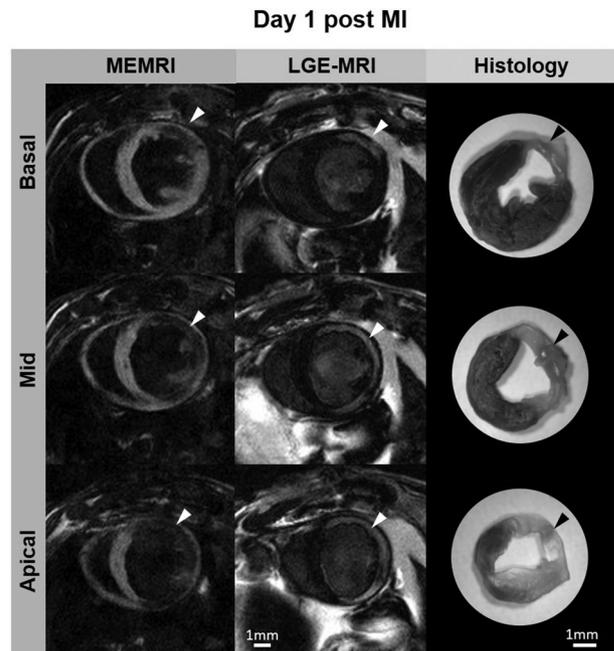
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Introduction Late gadolinium enhanced MRI (LGE-MRI) can quantify infarct size after myocardial infarction (MI) but is non-specific and reflects the increased membrane rupture and extracellular space that develop post MI.¹ Mn is a potent T1-contrast agent that enters myocytes through active calcium channels, thus reducing T1 in viable myocardium.² This active process rapidly ceases under ischemia. Hence, we hypothesised that Mn-enhanced MRI (MEMRI) could quantify final infarct size earlier than LGE-MRI.

Methods Myocardial infarction was induced in 7 mice which then underwent MEMRI (n=4, 0.1 mmol/kg MnCl₂) or LGE-MRI (n=3, 0.5 mmol/kg Gd-DTPA) at 1 hour post MI. All animals then underwent both MEMRI and LGEMRI at ~24 hours post MI with a contrast washout period of at least 5 hours between scans. Imaging was performed using a 9.4T Agilent MRI system and a multi-slice inversion recovery sequence in the short-axis orientation covering the whole left ventricle TE/TR=3.04/1.11 ms, TI~600 ms for MEMRI



Abstract 5 Figure 1 MEMRI and LGE-MRI acquired at 1 and 24 hours post MI
At 1 h post MI, Mn uptake in viable myocardium allowed the estimated final infarct area to be distinguished, whilst only subtle enhancement was seen in LGE-MRI resulting in a smaller measure of occlusion zone. At 24 h post MI the infarct shows hypoenhancement in MEMRI and hyperenhancement in LGE of similar sizes



Abstract 5 Figure 2 Direct comparison of MEMRI and LGE-MRI
Direct comparison of MEMRI and LGE-MRI acquired in the same animal at 22 and 27 hours after MI, respectively. Arrowhead shows hypoenhancement of the infarct in the MEMRI image and hyperenhancement of the infarct in LGE. *In vivo* data corresponded with TTC histological staining for infarct

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

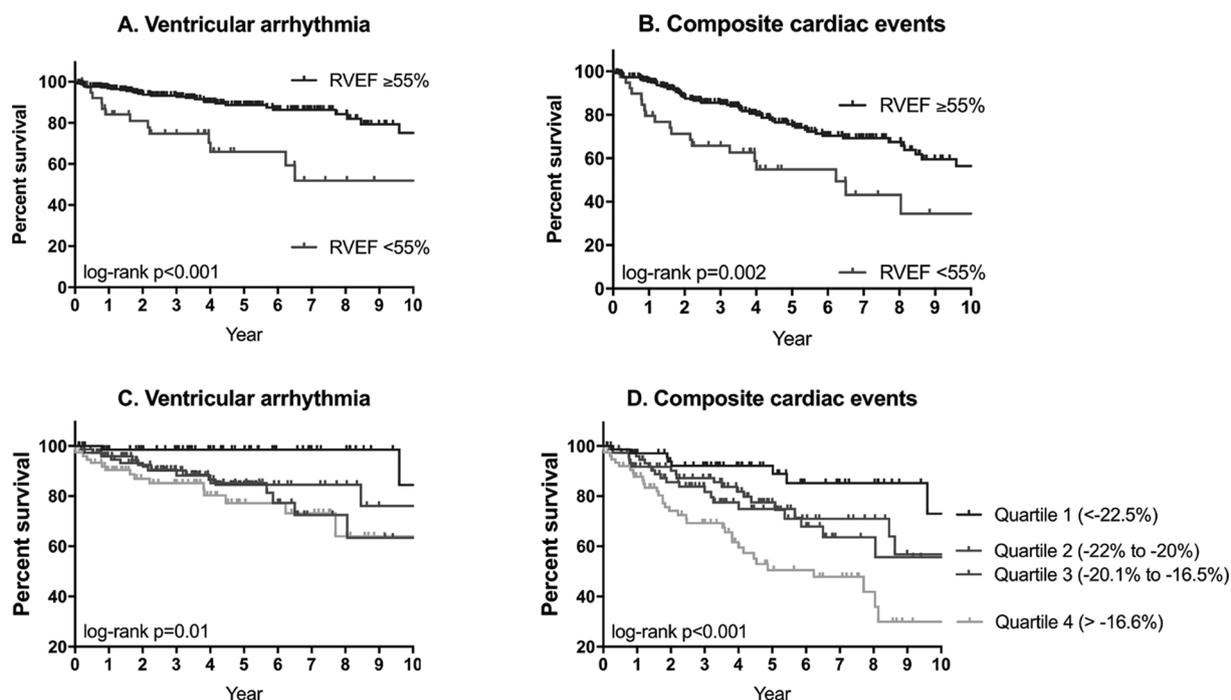
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Background The current assessment of hypertrophic cardiomyopathy (HCM) places emphasis on left ventricular (LV) function which is usually preserved in early disease. There are limited data on right ventricular (RV) assessment in HCM, progressive functional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