infarcts showed a degree of functional recovery after AMI. Volume and intensity of hyperenhancement on T2w CMR may give insights into functional recovery post reperfused AMI.

090 PRE-CONTRAST T1 MAPPING ALLOWS ASSESSMENT OF SEVERITY OF ACUTE ISCHAEMIC MYOCARDIAL INJURY

doi:10.1136/heartjnl-2012-301877b.90

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Introduction Cardiovascular magnetic resonance (CMR) is the gold standard technique to assess myocardial viability (using late gadolinium enhancement (LGE)) and reversible injury (using T2-Weighted (T2W) for oedema imaging) in acute myocardial infarction (MI). However, both LGE and T2W are hampered by methodological issues such as threshold-based method for post-processing with scope for error and the need for MR contrast agent. The interpretation of CMR is also challenged by the dynamic changes occurring in the acutely ischaemic tissue as part of the healing process. Pre-contrast T1-mapping can overcome these limitations by providing voxel-based quantitative tissue characterisation. In acute MI patients, we sought to investigate whether pre-contrast T1-mapping11 (1) detects acute myocardial injury, (2) allows for quantification of the severity of damage when compared to standard techniques such as LGE and T2W, and (3) has the ability to predict long term functional recovery.

Methods 41 patients with acute MI (30% non-ST elevation MI (NSTEMI)) underwent 3T CMR including T2W, T1 mapping and LGE, 12–48 h after chest pain onset and at 6 months. Patients with ST elevation MI (STEMI) underwent primary PCI first. Acute mean segmental T1values, acute and chronic regional and global function and segmental damaged fraction by T2W and LGE were assessed.

Results The diagnostic performance of acute T1-mapping was at least as good as that of T2W CMR for detecting myocardial injury; however, in NSTEMI it was significantly higher than T2W oedema imaging. Also, T1 values could define the segmental damaged fraction, as assessed by either by LGE or T2W ($p<0.01$). Furthermore, the likelihood of improvement of segmental function at 6 months decreased progressively as acute T1 values increased ($p<0.0004$).

Conclusions In patients with acute MI, pre-contrast T1 mapping allows to delineate the extent of myocardial injury and to predict functional recovery at 6 months. Further investigations will be needed to determine whether T1 mapping can distinguish oedema from necrosis in acute MI.

091 T1-MAPPING HAS A HIGH DIAGNOSTIC PERFORMANCE IN PATIENTS PRESENTING WITH ACUTE MYOCARDITIS: A CARDIOVASCULAR MAGNETIC RESONANCE STUDY

doi:10.1136/heartjnl-2012-301877b.91

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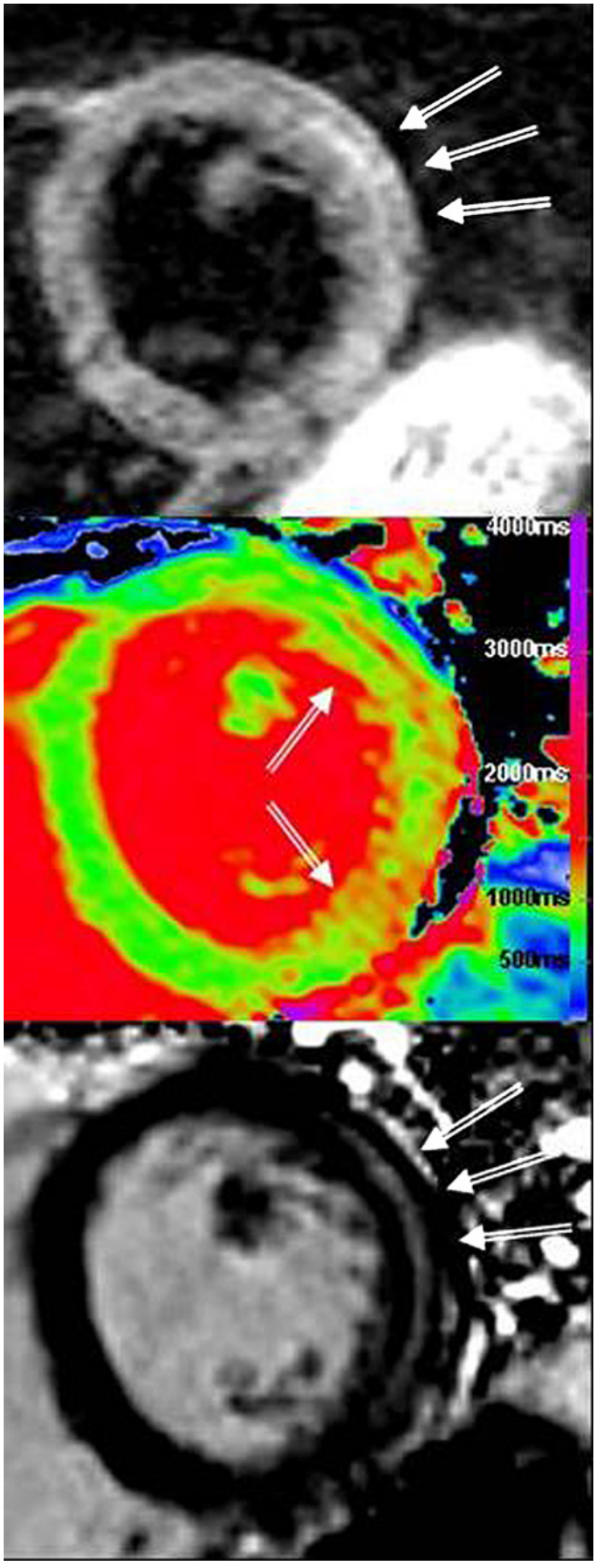
Background The diagnosis of acute myocarditis can be challenging. Cardiovascular magnetic resonance imaging (CMR) can be a useful tool in this setting but often requires multiple modalities for tissue characterisation, including T2-weighted (T2w), early and late gadolinium imaging. Cardiac T1-mapping is a novel technique that

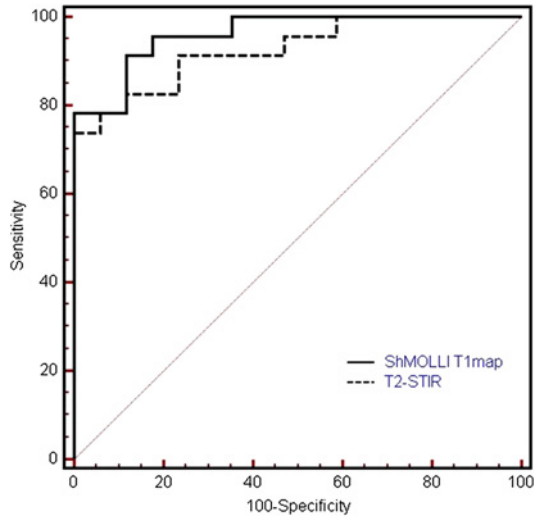


Abstract 091 Figure 1 Acute myocarditis. (Top) STIR demonstrating increased signal intensity in the mid lateral wall. (Middle) ShMOLLI T1-map demonstrating increased T1 values (1100–1200 ms) in the lateral wall. (Bottom) LGE imaging demonstrating mid-wall enhancement in the lateral wall.

is, quantitative and is also sensitive to acute changes in free water content without the need for exogenous contrast agents. We hypothesised that non-contrast T1-mapping can serve as a new diagnostic criterion for acute myocarditis.

Methods We studied 23 patients with suspected acute myocarditis and 17 healthy controls. All patients presented with chest pain and troponin I >0.04 ug/l and non-obstructive coronary arteries (either on coronary angiogram or ruled out by clinical criteria such as young age or no cardiac risk factors). CMR (at 1.5 T) within 10 days included (1) T2-weighted imaging (using the STIR sequence) for oedema; (2) T1-mapping (using the ShMOLLI sequence); and (3) late gadolinium enhancement (LGE) imaging for patterns of cell necrosis (Abstract 091 figure 1). Myocardial T2 signal intensity (SI) relative to skeletal muscle (T2 SI ratio) for detection of oedema and absolute T1 values per-subject were analysed.

Results All patients had a CMR diagnosis of acute myocarditis based on both positive T2-weighted imaging and typical non-ischaemic type LGE pattern. Compared to controls, both mean myocardial T1 and T2 SI ratio in patients were significantly higher (T1=1036±71 ms vs T1=938±19; T2 SI ratio=1.77±0.24 vs 1.52±0.10, p<0.0002 for both). Receiver operator characteristics analysis showed excellent diagnostic performance for both methods: the area-under-the-curve for T1-mapping=0.96 and T2-weighted imaging=0.93 (p=0.3, Abstract 091 figure 2). At a T1 value of 958 ms, the sensitivity and specificity were 87%.



Abstract 091 Figure 2 ROC curves for ShMOLLI T1-mapping and T2-STIR in acute myocarditis.

Conclusions Non-contrast T1-mapping has a high diagnostic performance for acute myocarditis and may be used as a novel additional CMR diagnostic criterion.

092 INTERSTITIAL EXPANSION IN HEALTH AND DISEASE—AN EQUILIBRIUM CONTRAST CMR STUDY

doi:10.1136/heartjnl-2012-301877b.92

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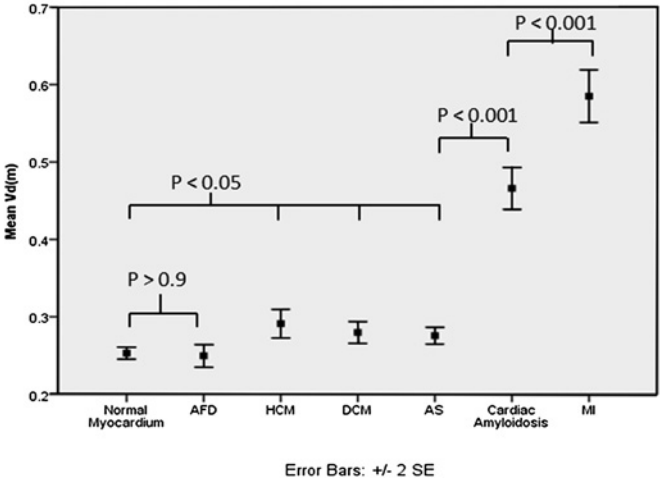
Introduction Interstitial myocardial volume expansion is an important factor in cardiac disease but until recently could only be accurately assessed with myocardial biopsy. It is usually a result of diffuse fibrosis, but can also be caused by infiltration (such as by amyloid deposition) or oedema. We have used a new method, Equilibrium Contrast Cardiovascular Magnetic Resonance (EQ-CMR) to accurately quantify the interstitial space in normal subjects and across a broad spectrum of cardiac diseases.

Methods The three steps in EQ-CMR are: (1) a primed gadolinium infusion to achieve contrast equilibrium, (2) Signal (T1) measurement pre and post equilibrium, (3) measurement of blood contrast volume (1−haematocrit). This allows calculation of the contrast volume of distribution, Vd(m), by:

$$\mathrm{Vd(m)} = (1 - \mathrm{haematocrit}) \times \Delta(1/\mathrm{T1})\mathrm{myo} \div \Delta(1/\mathrm{T1})\mathrm{blood}.$$

Vd(m) was measured in 278 subjects: 86 normal subjects (median age 43, range 24−81, 51% male) and 192 patients with Anderson-Fabry disease (AFD, n=17), dilated cardiomyopathy (DCM, n=31), hypertrophic cardiomyopathy (HCM, n=31), severe aortic stenosis (AS, n=66), cardiac AL amyloidosis (n=27) or myocardial infarction (MI, n=20).

Results In normal subjects, mean Vd(m) was higher in females (0.274) than males (0.237, p<0.001). In all diseases, Vd(m) was higher than normal subjects (p<0.001) except the intracellular storage disease AFD (0.250, p=0.9). Vd(m) was the same in DCM (0.280), HCM (0.291) and AS (0.276), but higher in the exemplar of infiltrative disease, cardiac AL amyloidosis (0.466) and higher again in MI (0.585, each p<0.001), Abstract 092 figure 1. These trends were also present when disease data were compared to gender matched normal subjects. Where Vd(m) was elevated, correlations existed with important clinical CMR parameters including ejection fraction, indexed left ventricular mass, end systolic volume and left atrial area, in apparent disease specific patterns, Abstract 092 table 1.



Abstract 092 Figure 1

Abstract 092 Table 1 Correlations assessed using Pearson coefficient

Disease	Ejection fraction	Indexed end systolic volume	Indexed left ventricular mass	Indexed left atrial area
Normal subjects	No correlation	No correlation	R=−0.36**	No correlation
AFD	No correlation	No correlation	No correlation	No correlation
DCM	R=−0.35*	No correlation	R=−0.36**	R=0.65***
AS	No correlation	R=0.51*	No correlation	No correlation
HCM	No correlation	No correlation	No correlation	No correlation
Cardiac AL amyloidosis	R=−0.57**	R=0.63***	R=0.44*	No correlation

*p<0.05, ** p<0.01, ***p<0.001.

Conclusions This study shows the ability of EQ-CMR to non-invasively quantify interstitial expansion using this novel technique in both the healthy population and a spectrum of differing cardiac