

Abstract 088 Figure 2 Detection rate of CMR and SPECT in patients with  $\geq$ 70% LMS stenosis.

of inducible perfusion defects in 18/22 (82%) of the LMS group; SPECT identified 13/22 (59%). For CMR and SPECT respectively, inducible perfusion defects were found in both LAD and LCx territories for 6/18 (33%) and 2/13 (15%). Only one patient had normal perfusion analyses (false negative) for both CMR and SPECT. Of 11 patients with  $\geq$ 70% LMS stenosis, 10 (91%) had inducible perfusion defects with CMR vs 5 (45%) with SPECT. Six (55%) vs 2 (18%) had a LAD and LCx disease pattern. Perfusion abnormalities were detected with similar frequency in  $\geq$ 50% and  $\geq$ 70% groups by both CMR (p=0.64) and SPECT (p=0.49). Abstract 088 figures 1 and 2 summarise the detection rate of CMR and SPECT in LMS disease in the  $\geq$ 50% and  $\geq$ 70% LMS groups respectively.

**Conclusions** CMR stress perfusion imaging identifies ischaemia in a higher proportion of patients with significant LMS disease than SPECT, and identifies a "classical" LMS pattern with higher frequency. Perfusion abnormalities are detected with similar frequency in patients with  $\geq$ 50% and  $\geq$ 70% LMS stenosis.

## **REFERENCE**

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REGRESSION OF MYOCARDIAL OEDEMA IS RELATED TO IMPROVEMENT IN MYOCARDIAL CONTRACTILITY FOLLOWING REPERFUSED ACUTE MYOCARDIAL INFARCTION

doi:10.1136/heartjnl-2012-301877b.89

A Kidambi,\* A N Mather, P Swoboda, M Motwani, J P Greenwood, S Plein. *Multi-disciplinary Cardiovascular Research Centre & Leeds Institute of Genetics, Health and Therapeutics, University of Leeds, Leeds, UK* 

**Introduction** Myocardial oedema is a feature of reperfused acute myocardial infarction (AMI), and contributes to stunning of perinfarct myocardium (the "area at risk"). Regression of oedema on T2 weighted (T2w) cardiovascular magnetic resonance (CMR) imaging is related to improved myocardial contractility post AMI in animal models, but has not been established in man. We hypothesised that resolution of tissue oedema correlates with recovery of regional contractile function.

**Methods** Patients after primary percutaneous coronary intervention for first ST-elevation AMI underwent CMR with T2w imaging,

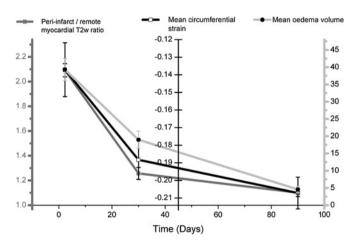
myocardial tagging and late gadolinium enhancement at 2, 30 and 90 days following reperfusion. Infarct size, regional circumferential strain, T2w signal intensity and volume of myocardial oedema were measured for infarct zone, peri-infarct zone and remote myocardium. Oedema and infarction were defined as zones with signal intensity 2 SDs above remote myocardium in T2w and LGE imaging respectively. T2w signal intensity was normalised to remote myocardium.

Results 30 patients had CMR imaging at all 3 time points with adequate image quality and sufficient peri-infarct oedema for quantitative analysis. Circumferential strain was significantly diminished in infarct and peri-infarct zones compared to remote myocardium (means -0.149 vs -0.184 vs -0.236, p<0.01 between groups and p<0.01 for trend). Remote myocardium showed no significant change in strain over time (F=1.44, p=0.24), while the peri-infarct zone (F=6.03, p=0.004) and infarct zone (F=20.34, p<0.001) showed a significant increase in magnitude (Abstract 089 table 1). This change closely mirrored resolution of both intensity and volume of T2w hyperenhancement (Abstract 089 figure 1). Decreased circumferential strain correlated significantly with T2w volume (r=0.30; p<0.01) and normalised T2w signal intensity (r=0.28; p<0.01). Due to the finding of recovery of function in the infarct zone, we analysed 8 patients with complete transmural infarction. The area of fully transmural infarction showed significant resolution of strain with time (means -0.103 (day 2), -0.148(day 30) and -0.194 (day 90); p=0.04 for trend).

Abstract 089 Table 1 Mean circumferential strain by myocardial location and time

|                            | Infarct zone | Peri-infarct zone | Remote zone |
|----------------------------|--------------|-------------------|-------------|
| 2 days                     | -0.102       | -0.137            | -0.226      |
| 30 days                    | -0.160       | -0.188            | -0.240      |
| 90 days                    | -0.186       | -0.207            | -0.241      |
| 2 days—30 days comparison  | (<0.001)     | (0.01)            | (0.10)      |
| 2 days-90 days comparison  | (<0.001)     | (0.002)           | (0.17)      |
| 30 days-90 days comparison | (0.04)       | (0.14)            | (1.00)      |
|                            |              |                   |             |

p Values in parentheses.



Abstract 089 Figure 1 Change in strain, T2w signal intensity, and oedema volume in the peri-infarct zone.

**Conclusions** Early after AMI, oedematous myocardium in the perinfarct zone demonstrated significantly reduced strain as compared to remote myocardium. Improvement of strain in stunned myocardium closely followed the regression of myocardial oedema. Patients with larger oedema volumes and higher signal intensity on T2w imaging demonstrated greater improvement of strain within the area at risk. In addition, both transmural and subendocardial

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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.

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

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

<sup>1</sup>E Dall'Armellina,\* <sup>1</sup>S Piechnik, <sup>1</sup>V M Ferreira, <sup>1</sup>M D Robson, <sup>1</sup>J M Francis, <sup>2</sup>F Cuculi, <sup>2</sup>R K Kharbanda, <sup>2</sup>A P Banning, <sup>3</sup>R P Choudhury, <sup>1</sup>T D Karamitsos, <sup>1</sup>S Neubauer. <sup>1</sup>OCMR Department of Cardiovascular Medicine, John Radcliffe Hospital, Oxford, UK; <sup>2</sup>Department of Cardiology, John Radcliffe Hospital, Oxford, UK; <sup>3</sup>Department of Cardiovascular Medicine, Oxford University, Oxford, UK

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 T1mapping11 (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.

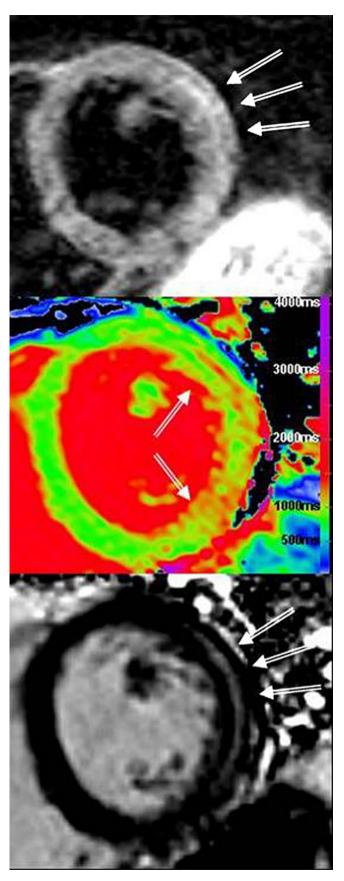
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## T1-MAPPING HAS A HIGH DIAGNOSTIC PERFORMANCE IN PATIENTS PRESENTING WITH ACUTE MYOCARDITIS: A CARDIOVASCULAR MAGNETIC RESONANCE STUDY

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

<sup>1</sup>V M Ferreira,\* <sup>1</sup>S K Piechnik, <sup>1</sup>E Dall'Armellina, <sup>1</sup>T D Karamitsos, <sup>1</sup>J M Francis, <sup>1</sup>R P Choudhury, <sup>2</sup>A Kardos, <sup>3</sup>M G Friedrich, <sup>1</sup>M D Robson, <sup>1</sup>S Neubauer. <sup>1</sup>University of Oxford, Oxford, UK; <sup>2</sup>Milton Keynes NHS Hospital Foundation trust, Milton Keynes, UK; <sup>3</sup>Université de Montréal, Montréal, Quebec, Canada

**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.

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