Journal of Cardiovascular Magnetic Resonance

Oral presentation

Open Access

T2-mapping of ischaemia/reperfusion-injury in the in vivo mouse heart

Steffen Bohl*[1], Craig A Lygate[1], Jeanette Schulz-Menger[2], Stefan Neubauer[1] and Jurgen E Schneider[1]

Address: [1]University of Oxford, British Heart Foundation Experimental MR Unit, Oxford, UK and [2]Franz Volhard Klinik, Charite University Medicine Berlin, HELIOS Clinics, Berlin, Germany

* Corresponding author

from 13th Annual SCMR Scientific Sessions
Phoenix, AZ, USA. 21-24 January 2010

Published: 21 January 2010

Journal of Cardiovascular Magnetic Resonance 2010, **12**(Suppl 1):O61 doi:10.1186/1532-429X-12-S1-O61

This abstract is available from: http://jcmr-online.com/content/12/S1/O61

Introduction

Oedema is a key feature of acute ischaemia/reperfusion (IR) injury. As such, it is a diagnostic - and potentially therapeutic - target, assessable using MRI. To date, its application in the mouse heart is limited due to the challenges associated with low SNR inherent in T2-weighting, miniscule anatomy, and rapid motion. Absolute quantification of transverse relaxation time (T2-mapping) circumvents SNR constraints and may be an alternative to T2-weighted imaging. We have therefore measured myocardial T2 in IR-mice and related T2-maps to the histological area-at-risk (AAR)

Methods

The left coronary artery (LCA) was occluded for 45 minutes followed by 24 hours of reperfusion. For histology, hearts were excised, cannulated, dye-perfused after LCA-reocclusion, and sliced. AAR was determined planimetrically (ratio of unstained to stained myocardium, %LV). Myocardial T2 was measured in healthy and IR mice (n = 5/9) on a 9.4 Tesla MR system using a double-gated spin-echo pulse-sequence (matrix 128 × 128; field-of - view 25.6 × 25.6 mm; 6-8 contiguous slices (1 mm); 8 echo-times (TE, 7-34 ms); repetition time = 1 respiratory cycle. Regions of interest (40-80 voxels) were placed in healthy (septal) and IR (anterior) myocardium. High-T2 myocardium was quantified using a semi-automated threshold tool (cut-off $T2_{NORMAL}$ + 1, 2 and 3 standard-deviations, SD), expressed as fraction of left ventricular volume (%LV) and the spatial extent compared with histology (n = 4). In order to improve congruence with histology, the 1SD datasets were manually corrected (1SD-c) by excluding high-T2 pixels located remotely to the LCA territory. Correlation (r^2) between methods was determined.

Results

Myocardial T2 in healthy mice was 21.3 +/- 1 ms. Septal T2 in IR mice was normal (21.2 +/- 2 ms; p = 0.8) while anterior T2 was elevated (27.9 +/- 2 ms; P < 0.01, Figure 1). Histologically, the AAR was 53 +/- 7%LV. T2-AAR was 58.3 +/- 4 (1SD), 37.8 +/- 5 (2SD) and 23.3 +/- 11%LV (3SD). AAR-size in the 1SD-c datasets was 48.7 +/- 6%LV. The correlation between methods was best when remote high-T2 pixels were excluded prior to volume measurements: r^2 = 0.99, 0.713, 0.06 and 0.21 for 1SD-c and 1×, 2× and 3× SD, respectively.

Conclusion

The area-at-risk exhibits prolonged T2, likely reflecting myocardial oedema. T2-mapping can be used to identify and quantify the AAR non-invasively and without the SNR constraints impeding T2-weighted imaging. Semi-automated analysis requires a low T2-cut-off. However, accuracy can be improved by manual exclusion of remote pixels, which would otherwise be erroneously included in the threshold-measurements. This problem is most prominent in basal sections where bright flow and motion artifacts exist.

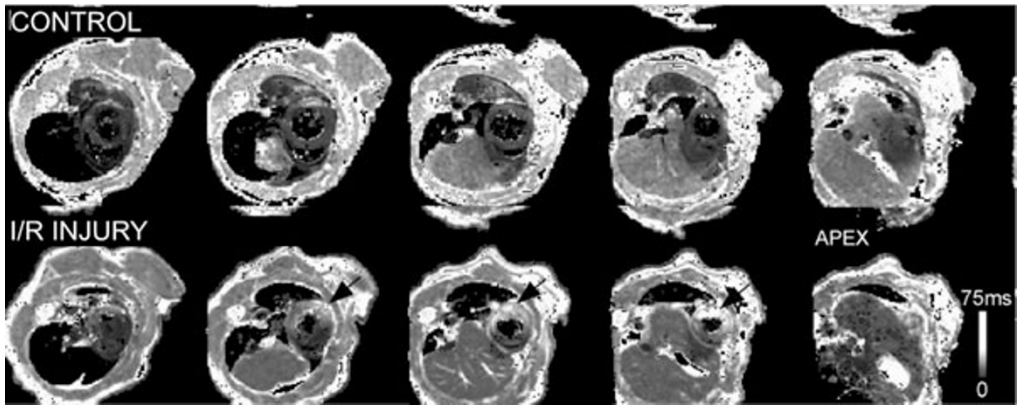

Figure 1