QUANTITATIVE T2 MAPPING VISUALIZES HEMORRHAGE AND EDEMA AFTER ACUTE MYOCARDIAL INFARCTION IN SWINE

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Introduction: T2 relaxation time correlates with pathologic changes within myocardial tissue. In the setting of myocardial infarction (MI), there is particular interest in detection of edema,[1] hemorrhage and microvascular obstruction (MVO).[2,3] Clinically accepted T2W edema imaging was recently challenged due to limited validation studies and inadequate data to support its use to delineate the area at risk in patients with ischemic myocardial injury.[4] In addition, edema detection through quantitative mapping has been shown to be more robust than qualitative clinical T2 W imaging.[5] We hypothesized that quantitative T2 measurement may be a reliable indicator for tissue characterization both for edema (T2 elevation) and hemorrhage (T2 reduction[6, 7]) post MI.

Methods: *Animal Model:* Under an IACUC-approved protocol, reperfused MI was induced in a swine model (*N = 8, 80-100 lbs*) by 120 min occlusion of the middle left anterior descending coronary artery. Seven animals developed intramyocardial hemorrhage. Imaging was performed 2-7 days post MI using an Achieva 3T TX system (Philips Healthcare, Best, Netherlands) and a 32-channel cardiac phased array (InVivo, Gainsville FL). Two additional normal swine were used for control.

MRI: Breath-hold T2W (Black-blood T2-STIR)[8] and 3D whole-heart free breathing T2-mapping[9] of the ventricles were carried out before the injection of contrast agent (0.2 mmol/kg, Magnevist). Three interleaved volumes were acquired with T2Prep TEs = 0, 25, 45 ms (3D T2-mapping imaging parameters: TR/TE 4/1.2 ms, flip angle 18°, voxel size 1.25×1.25×5.0 mm^3 interpolated to 0.98×0.98×2.5 mm^3). Post-contrast 3 min early (EGE, single slice) breath-hold phase sensitive inversion recovery (PSIR)[10] and 15-20 min late (LGE) independently navigated 3D PSIR images[11] were acquired post infusion (3D PSIR imaging parameters: TR/TE 5.6/2.7 ms, flip angle 18°, voxel size 0.99×1.27×3.0 mm^3 interpolated to 0.74×0.74×1.5 mm^3). Both 3D sequences were respiratory navigator gated.

Post processing: 3D T2 maps were calculated per voxel using linear regression with the three T2Prep volumes. 3D T2 maps from the manually segmented left ventricle (LV) are represented in 2D using bull's eye plots with 90 radial segments per slice and spanning whole LV. Corresponding whole-heart T2 histograms are also displayed. Hemorrhage was manually identified as area of low intensity/T2 surrounded by higher intensity/T2 regions of edema in both T2W images/T2 maps. MVO was defined as the area of low intensity surrounded by enhanced MI as defined by EGE. T2 values within hemorrhage were compared to those of healthy myocardium relative to controls and to healthy remote myocardium in the same animal. Areas identified as hemorrhage where correlated via regression to MVO derived from EGE.

Results: Representative slices from three swine as normal (first row), hemorrhage free MI (case #1, second row) and both edema and hemorrhage MI (case #2, bottom row) are shown in Fig 1. The areas of hemorrhage were detected in the high-resolution, free-breathing T2 maps, and correlated well with the areas of MVO observed as darker contrast defects in the EGE images. Bull's eye plots demonstrate 3D extent of edema/hemorrhage and the elevated/reduced T2 values can be seen in the histograms of infarcted animals. Quantitative comparison of left ventricular T2 values between normal and infarcted animals showed little differences (42.7±4.4 ms and 46.1±1.8 ms respectively, p=0.02). T2 in areas of suspected hemorrhage was significantly lower (35.7±4.9 ms) than both normal swine myocardium T2 (p =0.004) as well as remote myocardium within the same animal (p=0.00009). Good correlation was found between areas of MVO from EGE and areas of suspected hemorrhage from T2 Maps (R^2=0.899, p = 0.001), though the latter area was significantly smaller.

Conclusion: Our study suggests that the variations in myocardial tissue after MI and reperfusion are detectable with high resolution quantitative T2 mapping. Quantitative T2 imaging allows the detection and segmentation of hemorrhage without the temporal variability imposed by contrast-enhanced imaging. Future correlation with histology/pathology and the use of T2*-weighted imaging should confirm hypothesis of hemorrhage and allow us to investigate the source of of the different size of T2 depression and low EGE signal.

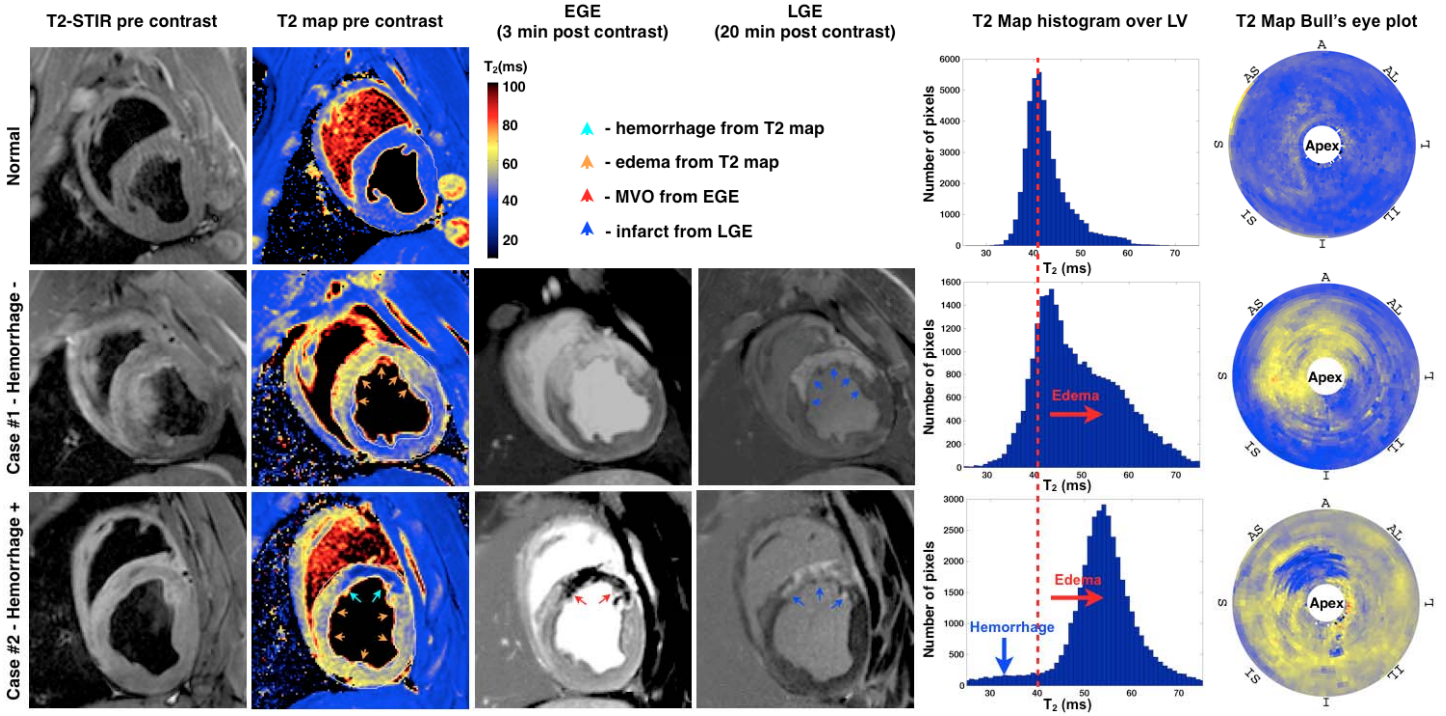

Figure 1: Representative short axis slices (SAX) from a normal (top row) and 2 different swine (without hemorrhage case #1 and with hemorrhage case #2) 3 days after MI. T2-weighted imaging (first column), T2 maps (second column), early and late gadolinium-enhanced imaging (third and fourth columns) are shown. T2 histograms and the associated Bull's eye plot reflecting T2 values through the whole LV (fifth and sixth columns, respectively) are also shown for comparison.

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References: [1] Aletras et al. Circ 2006 113:1865; [2] Judd et al. Circ 1995 92:1902; [3] Wu et al. Circ 1998 97:765; [4] Croisille et al. Radiol 2012 265:12; [5] Verhaert et al. JACC Card Img 2011 4:269; [6] Bradley Radiol, 1993 189:15; [7] Ghugre et al. MRM, 2011 66:1129; [8] Simonetti et al. Card Radiol 1996199:49; [9] Ding et al. ISMRM 2011; [10] Kellman et al. MRM 2002 47:372; [11] Lee et al. ISMRM 2011.