On the Origin of Myocardial Edema Contrast in T2-STIR Images

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Introduction Acute myocardial infarcts (AMI) are typically discriminated with T2-weighted short TI inversion recovery (STIR) with turbo spin echo (TSE) readouts [1], albeit with limited specificity [2]. Guided by the association that T2-STIR images identify AMI territories on the basis of edema-related T2 changes, even some of the recently proposed improvements [2,3] have relied on preferential sensitization of magnetization to T2-weighting. However, whether T2-STIR imaging itself may also be sensitive to other sources of image contrast have not been fully investigated. We hypothesize that in addition to T2-weighting, edema detection with T2-STIR imaging has substantial weighting from proton density (PD) changes.

Methods Imaging Studies: Dogs (n=4) subjected to an ischemia-reperfusion injury (occlusion of the left anterior descending artery (LAD) for 3 hours followed by reperfusion) were studied 2-hours post reperfusion (day 0), and on days 2, 5, and 7. Multiple breath-held and ECG-triggered T2-STIR images, and T1- and T2 maps covering whole left ventricle with contiguous short axis slices and long axis views were acquired using an Espree system (1.5T, Siemens, Erlangen, Germany). T1 maps were acquired using MOLLI [4] (IR preparation with 11 TIs ranging from 80ms to 4000ms and with bSSFP readout); T2 maps were acquired using T2-prepared bSSFP method with different T2-prep times (0, 24, 55ms) [5]; STIR images were acquired at the minimum TE (7.1 ms) and at 64 ms with body coil to remove the possibility of surface coil bias. Other T2-STIR scan parameters were: TR=2 R-R interval; TI=170 ms; spatial-resolution=0.9x0.9x8.0mm^3. All studies were terminated with PSIR late-gadolinium-enhancement (LGE) acquisitions to confirm LAD infarction. Data Analysis: A semi-automated approach, employed in [1], was used to identify edematous territories; a similar approach was used to identify the affected areas from T1 and T2 maps. Mean signal intensities (SI) from STIR images, corresponding mean relaxation values, and residual longitudinal magnetization between the segmented TSE readouts were used to compute the relative changes in PD between affected and normal territories. In detail, the proton density (PD) was calculated from the equation PD = SI/([1-(Mz+1)exp(-TI/T1)][exp(-TE/T2)]), where SI is the measured signal intensity in edematous and remote (healthy) areas from T2-STIR images, and Mz=[1-exp(-TR/T1)]/[1+exp(-TR/T1)] is the steady-state longitudinal magnetization at the end of each TR. The independent contributions from the different sources (T1, T2, and PD) were computed as a difference between edematous and remote territories normalized by the value in the remote myocardium. For each source of contrast, the mean and standard deviation of the fractional contribution across all studies were computed. A one-tailed t-test was performed to test whether the mean fractional contributions from PD and T2 are greater than zero at both TEs (7.1 ms and 64 ms). A two-sample t-test was performed to determine if there was a significant difference in the contribution of PD and T2 to edema contrast in STIR images acquired at both TEs. Statistical significance was set at $p < 0.05$.

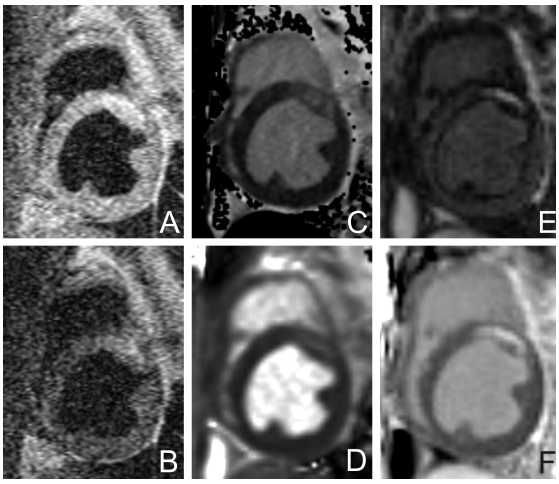

Figure 1. Representative short-axis images obtained from a dog subject to ischemia-reperfusion injury in the LAD territory on day 0 following reperfusion. **A**: STIR (TE=7.1ms); **B**: T2-STIR (TE=64ms); **C**: T1 map; **D**: T2 map; **E**: PSIR LGE magnitude and **F**: PSIR phase images. **A** and **B** were obtained with body coil, while **C**-**F** were obtained with surface coil. Note that the LAD territory is hyperintense in **A** and **B**, indicating that even in the presence of low T2-weighting (**A**), the affected region can be readily discriminated. Corresponding myocardial territories that appear hyperintense in **A** and **B**, also show elevations in T1 and T2 values (**C** and **D**, respectively). **E** and **F** confirm the presence of LAD infarct.

Results Affected territories were identifiable on STIR (at both TEs) and LGE images, and on T2- and T1-maps as hyperintense zones (Fig. 1). Table 1 lists the source and their corresponding fractional contribution towards edema contrast in STIR images obtained at the different TEs. One-tailed t-test confirmed that the mean T2 and PD based contrast at both TEs were greater than zero. STIR signal changes at TE=7.1 ms were dominated by PD than T2 effects ($p<0.05$). However, at TE=64ms, T2 contributions were greater than PD contrast ($p<0.05$), although there was significant contribution from PD. Mean signal intensity changes between affected and healthy territories from STIR images over all studies were: 33.4±7.1%(TE=7.1ms) and 65.7±12.9% (TE=64ms).

Source	STIR (TE=7.1ms)	STIR (TE=64ms)
T1 effects	-25.3.7±12.7%	-13.0±6.7%
T2 effects	15.1±3.7%	86.2±25.7%
PD effects	115.9±15.6%	25.5±17.3%

Table 1. Mean T1, T2, and PD contributions to image contrast between affected and healthy myocardial territories assessed during each study. At the low TE (7.1 ms), PD effect dominates, while at the higher TE, T2 contributions are dominant with a reduced, but a significant, contribution from proton density. Note that the T1-effects are always in the opposite direction to PD and T2 effects, as expected.

Conclusion In addition to the T2 changes, edematous territories identified on the basis of long TE STIR (T2-STIR) images, have substantial contribution from PD. Our findings suggest that methods that rely on the identification of edematous territories solely on the basis of T2 changes may compromise sensitivity in detecting myocardial edema. Approaches that combine PD and T2 contrast in a synergistic manner are expected to be the most sensitive for detecting myocardial edema.

References: [1] Abdel-Aty *et al*, Circulation 2004; [2] Kellman *et al*, MRM, 2007; [3] Altetras *et al*, MRM 2008; [4] Messroghli *et al*, JMRI 2007; [5] Giri *et al*, SCMR 2009