Detecting Myocardial Hemorrhage in the Setting of Ischemia-Reperfusion Injury: T2 vs T2*

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Introduction: Reperfusion after prolonged myocardial ischemia can lead to severe microvascular injury and myocardial hemorrhage (MH). Studies based on T2-weighted imaging have shown that MH is a significant predictor of poor LV remodeling1. Although both T2-weighted and T2*-weighted imaging are commonly used to assess MH1-3, a direct comparison of the two techniques is not available. This work aims to evaluate the T2 and T2* changes associated with MH and determine the effectiveness of the two techniques for reliably discriminating MH in ischemia-reperfusion (I/R) injury.

Methods: Canines (n=9) were subjected to I/R injury by fully occluding left-anterior descending (LAD) artery for 3 hours followed by reperfusion. Multiple breath-held ECG-triggered images of contiguous short-axis slices covering the entire LV were acquired at mid-diastole post-reperfusion on days 2, 5 and 7 on a 1.5T Espree System (Siemens Healthcare, Germany). The following imaging sequences were used: T2*-weighted images - mGRE acquisition with TR=17ms, 6 TEs=3.43ms, 6.42ms, 9.41ms, 12.40ms, 15.39ms, 18.38ms, bandwidth=566Hz/pixel, flip angle=12°, voxel size=1.3x1.3x8.0mm3; T2-weighted images - T2-prepared bSSFP with 3 different preparation times (0, 24 and 55ms), TR/TE=2.2/1.1ms, bandwidth=1002Hz/pixel, flip angle=70°, voxel size=1.3x1.3x8.0mm3 and Late Enhancement (LE) PSIR images - non-selective IR prepared bSSFP with TR/TE=1.75/3.5ms, bandwidth=789Hz/pixel, flip angle=40°, voxel size=1.3x1.3x8.0mm3. T2* and T2 maps were computed by pixel-wise fitting of multi-echo data to 2-parameter model of monoexponential decay. Data Analysis: Hemorrhagic infarctions (Hem+) were identified on the basis of hypointensities on T2* maps confined within the infarcted regions seen on LE images. For the Hem+ group, ROIs were manually drawn around the hemorrhagic territories on T2* maps and copied onto the T2 maps. For non-hemorrhagic infarctions (Hem-), ROIs were drawn around the infarcted territories on LE images and copied onto both T2* and T2 maps. Remote territories were identified on the basis of regions showing absence of hyperintensity on LE images. T2* and T2 changes were computed from hemorrhagic and non-hemorrhagic territories with respect to remote territories and compared. Statistical significance was set at p<0.05.

Results: A representative set of T2* map, T2 map and LE image from a dog with MH is shown in Figure 1. Table 1 shows the mean T2 and T2* values from hemorrhagic, non-hemorrhagic and remote territories averaged across all the animals and days. A bar plot of this data is shown in Figure 2. In the presence of hemorrhage (Hem+), mean T2* of hemorrhagic territories decreased by 43% with respect to remote territories. Mean T2, within the same territories, was elevated by 12% likely due to its high sensitivity to edema. In the absence of hemorrhage (Hem-), mean T2* of infarcted territories increased by only 5% with respect to remote territories, while mean T2 showed a 37% increase. All T2 and T2* changes were statistically significant (p<0.05).

Conclusion: T2* changes are highly sensitive to the presence of hemorrhage, and relatively insensitive to edema. Although T2 decreases significantly in the presence of hemorrhage when compared to non-hemorrhagic infarcts, it is still significantly higher than that of remote territories making hemorrhage less conspicuous. This is possibly due to the refocusing effects of 180° pulses in T2-weighted imaging and its high intrinsic sensitivity to edema. We conclude that T2* maps are more effective at discriminating MH and T2 maps are best suited for detecting myocardial edema associated with I/R injury.