

Title: Analyzing myocardial R2* mapping post myocardial infarction using stabilized probability plots

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Structured Abstract:

Introduction: Hemorrhage within myocardium often occurs following myocardial infarction (MI). Red blood cell degradation leads to iron deposition that can be detected by T2*-weighted magnetic resonance imaging (MRI). To improve detection of myocardial hemorrhage, we analyzed R2* relaxation rate ($1/T2^*$) maps. Currently, manual delineation of the remote myocardial region is used as a reference for differentiating hemorrhagic from non-hemorrhagic regions. Automated segmentation would be valuable for analyzing the temporal evolution of myocardial hemorrhage post-MI. We proposed an automated segmentation method based on the stabilized probability plot, a graphical technique for assessing whether or not a data set is normally distributed, in order to examine R2* distribution in cardiac tissue and distinguish hemorrhage-associated changes in R2*.

Hypothesis: The distribution of R2* values in post-MI images shows greater deviation from normality than baseline images.

Methods: Canine MR images ($n=4$) were acquired at baseline (day 0) and several time points (3 to 41 days) following experimentally-induced MI. Images consisted of 10 to 13 short-axis slices (slice thickness=6mm) covering the entire left ventricle (LV) obtained with a multi-gradient echo sequence (8 echo times (TE) covering 3-23 ms). Images were processed to generate R2* maps by applying exponential curve fitting to signal intensity on a pixel-by-pixel basis. Endocardial and epicardial borders were manually drawn on images with the shortest TE, and then applied to the R2* maps. The stabilized probability plot method was used to threshold R2*, below which the myocardial voxel-by-voxel values followed normal distribution. Pixels with R2* values above this value were considered abnormal tissue. The volume of tissue was determined, relative to LV volume, with R2* values above the threshold. Mean values of R2* were also determined for these abnormal and normal R2* regions.

Results: For baseline images, R2* values followed an approximately normal distribution over the full range of values. For post-MI analysis, R2* values showed significant deviation from normality. The mean R2* values of normal regions in the post-MI images remained approximately constant as a function of time post-MI and were close to the mean baseline R2* values. The mean R2* values of abnormal regions appeared to peak at approximately day 3 post-MI. Temporal evolution of the extent of injury was also greatest at day 3 post-MI.

Discussion: For all time points post-MI, our finding that mean R2* values of normal tissue regions remained close to the mean R2* of baseline tissue, is consistent with what one might expect of healthy tissue, remote from the infarct. One limitation of this method is that deviations from normality could also be created by macroscopic magnetic field variations. Techniques for reducing these variations, in addition to thinner slice acquisitions, may be combined with our approach and improve results.