Signal Intensity in Myocardial Scars Detected by Delayed-Enhancement MRI Differs in Subjects With and Without Previously Known Myocardial Infarction

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Purpose: To investigate differences in tissue characteristics between Unrecognized Myocardial Infarction (UMI) and Recognized Myocardial Infarction (RMI) scars, by assessing the signal intensity (SI) detected by delayed-enhancement Magnetic Resonance Imaging (DE-MRI).

Introduction: DE-MRI is a valuable and accurate diagnostic tool to detect myocardial infarction¹. Although the relationship of DE-MRI to the underlying pathophysiology is not completely elucidated, it is thought that delayed-enhancement of myocardial infarction scars is due to an increased extracellular space, allowing a larger distribution volume for the extracellular contrast agent².

Materials and Methods: A randomized group of 259 community-living 70-year-old subjects underwent cardiac MRI. DE-MRI detected myocardial scars were divided in 2 groups, UMI and RMI, according to the hospital medical records. Myocardial infarction scars, defined as delayed-enhancement involving the subendocardial layer and identified in short-, as well as in at least one long-axis view, were found in 60 subjects (24.2%), in whom 49 were UMIs (19.8%)³. Nulled myocardium by the inversion pulse was considered viable and assumed to represent normal myocardium tissue. For each individual, the short-axis slice with the biggest brightest area of delayed-enhancement was chosen as the most representative of the infarct. A region of interest (ROI) was drawn to delineate the scar area in the chosen short-axis slice. An additional ROI was drawn to delineate the normal myocardium (Figure 1 and 2). For each ROI, the mean SI was calculated with computer-assisted software. The SI ratio was calculated as the ratio between the mean SI in normal myocardium.

Results: In both groups, the mean SI of the scars was different from the mean SI in the normal myocardium (UMI group, *p*-value<0.0001; RMI group, *p*-value=0.0003). The inversion time chosen to null viable myocardium did not differ between the UMI, RMI and normal subjects groups (*p*-value=0.38). The mean SI ratio in the UMI group (4.5 ± 3.0 , mean \pm SD) was lower than in the RMI group 8.9 ± 5.1 (*p*value=0.0004) (Figure 3). In a multiple regression model with the SI ratio as the dependent variable, this difference was still significant (*p*<0.0001) after adjustment for gender, BMI, time of image acquisition after gadolinium injection, scar transmurality or total myocardial infarction scar mass.

Discussion: The present study revealed that UMIs have a different behavior from RMIs in DE-MRI, as expressed by the significant difference in SI ratio between the two groups. This difference in SI ratio between the groups most likely reflects different contrast distribution volumes of the tissues, and may indicate a different pathophysiologic process. Epidemiological studies state that subjects with UMI have a similar mortality rate as the ones with RMI⁴. In these studies, UMI diagnosis was based on ECG-criteria. High resolution DE-MRI provides a more sensitive method to detect myocardial infarctions, some of which are very small, and do not have the sufficient extent of necrosis to produce a significant Q wave on ECG ⁵. In patients with clinical suspicion of coronary artery disease, positive DE-MRI carried an increased risk of future cardiac events⁶ that, to our knowledge, has not been presented in a general population study.

References:

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Figure 1. Example of an UMI scar in the inferoseptal wall of the left ventricle. Signal intensity ratio 5.9. (1) Myocardial scar area; (2) Normal myocardium area.



Figure 2. Example of a RMI scar in the anterior wall of the left ventricle. Signal intensity ratio 7.6. (1) Myocardial scar area; (2) Normal myocardium area.



Figure 3. Box-plot graph of the Signal Intensity (SI) ratio. The mean SI ratio in the UMI group $(4.5\pm3.0, \text{mean}\pm\text{SD})$ was lower than in the RMI group (8.9 ± 5.1) (*p*-value=0.0004).