Late Enhancement of Infarct May Overestimate Myocardial Ischemia in Stress/Rest MR Perfusion Imaging

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Introduction: Delayed contrast-enhanced cardiac MR has been shown to provide key clinical information regarding infarct size and location [1]. However this same phenomenon of delayed enhancement of infarcted myocardium has the potential to decrease the specificity for ischemia detection on stress/rest MR myocardial perfusion imaging. Currently it is unknown whether the late enhancement of infarcted myocardium present after intravenous MR contrast administered during dynamic pharmacologic stress first-pass perfusion imaging causes an overestimation of perfusion within an infarct zone that can be detected on subsequent rest MR perfusion imaging. Our goal for this study was to determine whether the contrast-to-noise (CNR) ratio of infarcted myocardial tissue was decreased on rest perfusion imaging performed after pharmacologic stress MR perfusion imaging as opposed to rest MR perfusion imaging performed alone.

Methods:

Subjects and Recruitment: Eight patients (4M:4F, Age 50-76 yrs, mean 61 yrs) with a history of myocardial infarction and with evidence of myocardial infarction on rest/adenosine dual-isotope SPECT imaging were recruited to undergo MR examination.

Imaging Protocol: Studies were performed on a Siemens 1.5T MR system with the Sonata gradient coil (40 mT/meter; 200 μsec rise time) using a phased-array coil. The study was EKG gated. MR examinations were performed on two days. On Day 1, the MR myocardial perfusion examination was performed during the intravenous administration of adenosine infused at a rate of 0.140mg/kg/min. Adenosine was infused for 3 min after which 0.05 mmol/Kg of Omniscan was injected through a separate antecubital IV and images acquired. The adenosine infusion was continued until the trueFISP image acquisition was complete (50-60 secs). After 15 minutes, rest images were obtained during the readministration of 0.05mmol/Kg of Omniscan using the same trueFISP sequence. After completion of the rest study, late-enhanced images were obtained. On Day 2, an MR myocardial perfusion examination was performed at rest only.

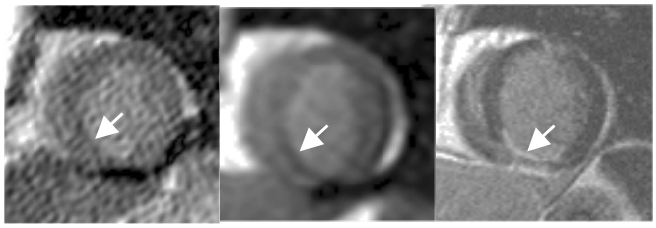
Perfusion: Three myocardial perfusion images of the heart were obtained in the short axis using a trueFISP sequence (TR 2.2 msec/TE 1.1 msec, matrix 80*256, temporal resolution 200 msecs, including time for the saturation recovery pulse preparation, 60 measurements). Slice thickness was 6 mm, with in-plane resolution approximately 1.4 x 3.2 mm, but larger or smaller depending on patient size and the required FOV. Images were obtained during power injection of 0.05 mmol/Kg of Omniscan at a rate of 5 mls/sec followed by a 15 ml normal saline flush into an antecubital vein.

Viability: To identify late enhancement of myocardial tissue (an indicator of nonviable myocardium), two-dimensional T1-weighted inversion recovery segmented fast gradient recalled echo images (TR 8 ms, TE 4 ms, flip angle 30º, segments 13, FOV 300-380 mm, matrix 256 x 172, triggered every 2 heartbeats) were performed in the same planes as the perfusion images. The inversion recovery time was set to null the myocardium.

Image Analysis: Images were assessed at a satellite Siemens MR console. Late-enhanced images were inspected for the region of infarct and perfusion images from Day 1 and Day 2 were inspected for the most pronounced corresponding region of signal deficit. For each perfusion examination, CNR with normal adjacent myocardium was calculated for this region on this image and the immediate two images before and after to ensure that the CNR represented a peak difference. Intra and interpatient coil profile differences were corrected using base-line images.

Results: Although, in general, a perfusion deficit in the infarcted territory was visible on Day 1, the average CNR of infarcted territory with normal adjacent myocardium was significantly less on Day 1 than on Day 2 (Figure). CNR within the infarct zone was lower on Day 1 (rest performed after stress) 0.066 (± 0.28), compared with CNR in the infarct zone on Day 2 (rest alone) 0.37 (± 0.080, p=.012).

Figure



Rest MR perfusion performed after stress perfusion imaging (Day 1)	Rest MR perfusion alone (Day 2)	Delayed contrast-enhanced MR imaging

The arrow points to a subendocardial inferoseptal myocardial infarction.

Discussion: The impact of late-enhancement on defect reversibility has not yet been explored. This study demonstrates that after contrast administration for stress MR perfusion imaging, the CNR of infarcted myocardial territory to normal myocardium is significantly less than when rest MR perfusion imaging is performed alone (without any prior contrast administration). Given that delayed-enhanced myocardial imaging accurately shows infarcted myocardial tissue [2], rest perfusion MR imaging performed after adenosine stress perfusion MR imaging in a subjective clinical setting may be redundant, if not misleading. This study also suggests that for myocardial perfusion reserve calculation , stress and rest MR perfusion imaging in patients with myocardial infarct or infarct/ischemic admixture be performed on subsequent days in order to permit adequate wash-out of contrast material from infarcted tissue.

Conclusion: CNR of infarcted myocardial territory to normal myocardium is significantly less when rest MR perfusion imaging is performed after contrast administration for stress MR perfusion imaging than when performed alone. This phenomenon may reduce the accuracy of ischemia detection if both stress and rest components of an MR perfusion examination are performed in the same setting.

References:

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