Myocardial Contrast Concentration Estimated in First-Pass MR Perfusion Imaging Does Not Increase Proportionately with the Dose of Gadolinium-DTPA

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Introduction

Quantification of first-pass contrast-enhanced perfusion MR imaging depends on measuring signal intensity in the left-ventricle (LV) and myocardium as a function of time. This T1-related signal intensity is affected by several factors including gadolinium concentration, T2*, pulse sequence, sequence parameters, contrast dose, perfusion, water exchange rates, and interstitial loading with gadolinium. A conversion of signal intensity to gadolinium contrast concentration was validated for the LV and myocardial signal intensity during dynamic contrast passage [1]. The primary purposes of this study were to determine: 1) if myocardial contrast concentrations during first-pass perfusion imaging are proportional to dose, and 2) how myocardial gadolinium concentrations vary between rest and dipyridamole stress perfusion.

Materials and Methods

Ten normal volunteers underwent dual-bolus MR perfusion imaging using 0.005/0.05 mmol/kg and 0.005/0.1 mmol/kg Gd-DTPA contrast doses on separate days. For each contrast dose, dipyridamole (0.56 mg/kg) stress perfusion was acquired >4 hours before the rest study. Imaging was performed on a Siemens 1.5T scanner using a segmented gradient recalled echo – echo planar imaging (GRE–EPI) sequence. A look-up-table (LUT) for signal intensity versus T1 magnetization was simulated [2] using the following imaging parameters (90° composite prep, 25° readout, TR 7.5ms, TE 1.48ms, 8 mm slice, acquisition matrix 128x80, FOV 360x270). The T1 value was then converted to contrast concentration [Gd] using the equation $1/T1 = 1/T1_{init} + \gamma \bullet [Gd]$ ($T1_{init}$: 850ms, γ: 4.5 L/mmol). Proton density weighted reference images were also acquired at the beginning of each study using a 5° readout and without saturation preparation. The time-intensity curves were generated based on 6 circumferential sectors of a mid ventricular slice. Myocardial contrast concentrations at peak (during the first-pass) and on late perfusion images (last 8 of 60 images acquired) were measured. Dose-related ratios of myocardial and LV contrast concentration were also calculated to study whether gadolinium concentration increased proportionately with dose.

Results

Figure-1 shows an example of myocardial time-concentration curves using the LUT conversion. During the late perfusion images where the LV gadolinium concentration can be estimated by the LUT conversion, gadolinium concentrations in the blood doubled as expected from the 0.05 mmol/kg to the 0.1 mmol/kg dose for both rest and stress perfusion (dose-related ratios were 2.10±0.42 for rest and 2.06±0.42 for stress, figure-2). However, myocardial gadolinium concentrations at peak enhancement did not increase in a dose predicted manner. The dose-related ratio of peak myocardial gadolinium concentration from a 0.1 mmol/kg dose to a 0.05 mmol/kg dose was only 1.64±0.22 at rest and even lower at stress (1.46±0.26) – a large fraction shorter than the expected doubling for the doses used. Both of these ratios were significantly lower than in the LV ($p<0.01$ for both rest and stress). The dose-related increase in myocardial gadolinium concentration on late perfusion images was closer to the expected ratio for the doubling of contrast dose used (1.78±0.29 for rest and 1.85±0.46 for stress) but both were still significantly lower than the concentration ratios as seen in the LV ($p=0.01$ for rest and $p<0.1$ for stress).

Discussion

Myocardial contrast concentration can be estimated using previously validated LUT signal intensity modeling to better understand the effect of contrast dose on signal intensity dynamics during first-pass perfusion imaging. Peak myocardial contrast concentration did not double from the 0.05 mmol/kg dose to the 0.1 mmol/kg dose despite the fact that blood gadolinium concentration doubled as expected. The relationship between signal intensity and gadolinium concentration can be more complicated than T1 and T2 relaxations considered in our simulation and the current experiments may have some limitations. Although the gadolinium concentration estimates after the first-pass of contrast are partially contaminated by second-pass kinetics, the LV gadolinium concentrations were within 10% of the expected dose dependent doubling. The degree to which myocardial gadolinium concentrations increased indicates there are factors modulating myocardial signal intensity that are more severe than this 10% difference. We speculate that interstitial loading or T2* effects within intramyocardial blood vessels are important factors affecting myocardial signal intensity. More complete kinetic analysis will be necessary to understand the reasons why peak myocardial gadolinium concentrations did not double by the amount expected for a 2-fold increase of contrast dose. In the meantime, these data raise concerns about optimal dose of gadolinium for first-pass perfusion quantification.

References

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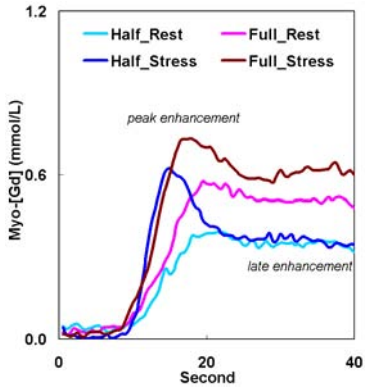

Figure-1

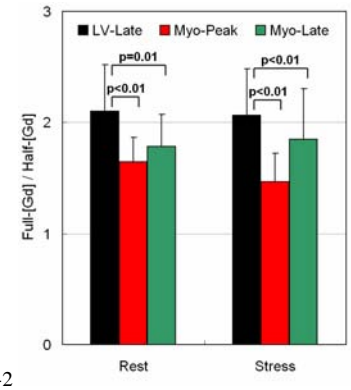

Figure-2