Quantitative Assessment for Delayed Gadolinium Enhancement of Myocardium: Detection of Abnormal Enhancement in Apparently Non-Enhanced Myocardium of Cardiac Amyloidosis

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BACKGROUND AND PURPOSE:

Delayed gadolinium enhancement (DE) displays irreversible myocardial damage in myocardial infarction. However, current assessment of DE in myocardium has limitation as based on relative signal intensity to the neighboring myocardium that is assumed to be normal. Infiltrative cardiomyopathy such as cardiac amyloidosis may have diffuse involvement of myocardium, and apparently non-enhanced myocardium may not be normal. To solve this problem, we tried to quantify myocardial signals as relative value to nearby lumen signals in the left ventricle. Major limitations for quantification of myocardial signals include sensitivity inhomogeneity of surface coil and variability of heart rate among patients. The purpose of this study was to evaluate variability of the ratio depending upon heart rate and time after contrast injection in normal myocardium, and to display abnormality in apparently non-enhanced myocardium in cardiac amyloidosis as compared with normal myocardium.

METHODS:

We have performed cardiac MRI by 1.5T system (Magnetom Sonata) using a standardized protocol. DE was evaluated with segmented IR-true-FISP (ECG triggered, TI=300msec, data acquisition at late diastolic phase) at 2, 5, 10, 20 minutes after administration of 0.15mmol/kg of Gadolinium-DTPA. Seven short axial and 3 long axial sections were obtained in a single breath-hold at each time point.

We reviewed DE images of normal myocardium from 9 patients (6 with single vessel myocardial ischemia, 3 with conduction abnormality). All the 9 patients had regular rhythms with mean heart rate 59 BPM (range 44-75BPM). We set region of interest (ROI) on the myocardium in segments #1, 2, 4, 5 of the American Heart Association, and on blood pool in the left ventricular cavity close to myocardium of each segment, and then calculated myocardium-to-lumen ratio (M/L). In patients with myocardial ischemia, ROI in the remote region was selected for analysis. ROI was set by tow observers independently, and interobserver variability was calculated.

In addition, we reviewed DE images of 3 patients with histologically proven cardiac amyloidosis (CA) and 4 with hypertrophic cardiomyopathy (HCM). We set ROI on the ventricular wall localized within "apparently non-enhanced area" and on blood pool in the left ventricular cavity near to the wall's ROI (Figure 1), measured the SI of each ROI, and then calculated M/L.

RESULTS:

A total of 284 M/L data from 71 regions of normal myocardium in 9 patients were obtained, from which 41 data with low SNR (<3.0) lumen was excluded from analysis. Mean and SD of M/L at each time-point from observer 1 is displayed in Figure 2. SD of M/L was sufficiently small especially at 2 and 5 minutes, but a little bit large at 20 minutes. Larger SD at 20 minutes is probably ascribed to longer T1 and lower SNR. Mean difference of M/L between the two observers was 0.005, and 1SD was 0.022, that seems to be successfully small. Heart rate dependence of M/L was small: mean (SD) of M/L was 0.41 (0.04) and 0.44 (0.41) for HR≥60 (n=4) and HR<60 (n=5) respectively.

M/L of CA in "apparently non-enhanced area" was significantly higher than normal myocardium (Figure 2). On the contrary, M/L of HCM was higher than normal myocardium, but not significant.

CONCLUSION:

Normalization of myocardial signals by blood pool signals in DE (M/L) is useful to cancel out sensitivity inhomogeneity of surface coil and heart rate dependence of signal intensity at least partially. M/L can detect abnormality in apparently non-enhanced area of myocardium with CA.

Figure 1

DE of CA at 20minutes

Circle: ROI on blood pool

Polygon: ROI on apparently non-enhanced myocardium with cardiac amyloidosis

DE is shown in the subendocardium and papillary muscle

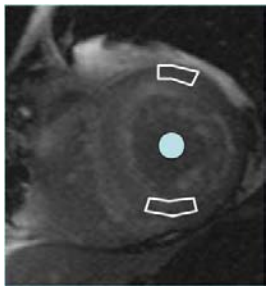

Figure 2