Cardiac diffusion-weighted MR imaging in acute myocarditis: initial experience

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Purpose

The purpose of this preliminary study was to assess the patterns and diagnostic accuracy of diffusion-weighted cardiac imaging with a particular concern to its sensitivity in acute myocarditis, by comparison with classic comprehensive MR acquisitions including black-blood fatsat T2 acquisitions for detection of edema, cineMR sequences for cardiac function, and first-pass perfusion and delayed-enhancement studies for disease characterization.
Methods and materials

Patients

Thirty-eight consecutive patients with strong suspicion of acute myocarditis were prospectively investigated with MR imaging.

The final diagnosis was made on the basis of a normal coronary angiogram with normal, a transient rise in biochemical markers of myocardial injury, followed by a spontaneous resolution of clinical symptoms and of segmental or global wall motion abnormalities, and optionally, a history of a recent infectious episode.

MR patterns were suggestive of myocarditis in 32 patients whereas 6 patients with a fully normal cardiac MR examination were retained to serve as control for assessment of specificity.

MR Imaging

MR examinations were performed using a GE Twinspeed Excite 1.5 Tesla system (amplitude, 40 mT/m, slew rate, 150 mT/m/msec) with a dedicated eight-channel cardiac phased array coil. All the sequences were ECG-gated. Diffusion-weighted imaging (DWI) was added to the comprehensive cardiac MR examination currently used in these patients.

After scout localization, breath-hold ECG-gated DWI was performed at end-diastole in short axis and four chamber views. Twelve DW (variable b) and 12 T2w (b=0) slices were acquired per breath hold. Each breath hold lasted 12 to 18 seconds, according to heart rate. Typical DWI parameters were single-shot spin-echo EPI, TR/TEeff 3000-4000/60-80 msec (with a TR of 3 to 5 R-R intervals depending on the heart rate), b factor, 300sec/mm2, field-of-view 44x44 cm, section thickness 8 mm with a gap of 1 mm as for other sequences, bandwidth 167 kHz,. Matrix size was 256 x 128. The diffusion gradient was applied in 3 orthogonal directions (x, y, and z), and an average of these measurements was calculated to give the trace of the diffusion tensor.

Reading criteria

a) Visual inspection

On DWI images, the hyperintense regions were considered as diseased, when compared to normal myocardium which displayed no signal. On post-contrast delayed-enhanced sequences, the hyperenhanced regions were defined as distinct myocardial high signal (Fig. 1).
Analysis was done segment by segment. The LV myocardium was divided into 17 segments (18) encompassing the entire LV like in echocardiographic segmentation, and a location within the myocardium (subepicardial, centromyocardial or transmural) was described for each diseased segment. Signal intensities of normal and abnormal areas of enhancement were measured at different b-values in order to determine the best contrast-to-noise ratio.

b) ADC measurements

ADC maps were calculated from regions of interest (ROI) placed on the DWI hypersignal area on images with b factor at 300sec/mm², and on an adjacent segment close to the DWI hypersignal and remote normal myocardium on images with b factor at 0sec/mm². In other words, ADC was measured after having determined ROIs: a) for the area of abnormal hyperintensity on the initial DW image coregistered with the delayed hypersignal on DE image; b) for an area of neighbouring myocardium judged as normal close to abnormal DE; and c) for a remote normal myocardial area located in the septal wall. Absolute and relative pixel values and mean values were generated for all ROIs on each section of the initial DW image and ADC map.

Statistical Analysis

Statistical analysis was performed by using commercially available software (SPSS-PC, version 10.0; SPSS, Chicago, IL). For the statistical analysis of ROI-based data, two-way analysis of variance (ANOVA) was used to compare the mean values of each ROI. Comparisons between the mean values of the ROIs were performed by using a paired t test. The number of segments involved with DWI and DE was analyzed by the chi-square test for the location of abnormalities; and the Mann-Whitney U test for number of segments involved in each group. Additionally, Pearson’s correlation coefficient was used to compare the location of hypersignal areas on DWI and DE acquisitions. Quantitative variables are presented as mean ± standard error of the mean (SEM). A p value <0.05 was considered significant.
**Fig. 1:** Acute viral myocarditis involving the inferolateral wall. Short-axis DWI images shows high signal of the lateral wall (short arrows)

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Fig. 2: Same patient. ADC map shows decreased ADC (arrows)

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**Fig. 3:** Same patient. Short axis delayed enhancement image shows hypersignal in the same area (arrows)

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Results

Clinical description and outcome

MR studies were performed 4.7 ± 1.1 days (range, 1 to 21 days) after the acute event. No patient had history of prior myocardial infarction. Twenty-three patients had a clinically obvious infectious episode 9.9 ± 2.1 days prior to admission. The primary symptom was acute chest pain in all patients. No patient had dyspnea or sign of cardiac failure. The mean serum concentration of troponin I was 8.1 ± 1.7 ng/ml. Electrocardiographic changes in the ST segment were observed in 17 patients (ST elevation in 11, ST depression in 6 patients) and T waves inversion in 11 patients associated with acute type II atrial-ventricular block in one patient. Coronary angiography revealed no significant coronary stenosis in each of the patients included. All the patients were discharged in the absence of any alternative diagnosis and without any complication; all of them were treated with beta-blockers and ACE inhibitors. At follow-up (6 ± 2 weeks), no patient had any complication (heart failure, arrhythmias or sudden death) or any recurrent chest pain episode.

MRI study

Nineteen patients had focal edema on T2-weighted images. First-pass perfusion studies were normal in all patients examined. The abnormal enhancement patterns on DE images included hyperenhancing nodules in 10 patients, transmural involvement in a focal form in 5 patients, and centromyocardial or subepicardial thick bands in 17 patients. There was no correlation between troponin level and the number of segments affected (r = 0.1557, p = 0.46).

DWI displayed a focal hypersignal in 29/32 patients, resulting in a per-patient sensitivity of 91%. The abnormal DWI patterns included hyperintensity nodules in 10 patients, transmural involvement in a focal form in 4 patients, and centromyocardial or subepicardial thick bands in 15 patients (Figs. 1-4). The ADC maps of diseased areas vs normal remote myocardium showed a relative decrease (Figs. 3,5) of the apparent diffusion coefficient in 28 patients (67.9% ± 4%) and an increase in 4 patients (117.2% ± 8.8 %), resulting in a mean relative ADC of 74.1 % ± 5.6 %. The increased relative ADC observed in the 4 patients corresponded to an area of diffuse edema. Absolute ADC was 0.00751±0.00042 mm²/s (confidence interval 0.00697-0.00805 mm²/s) in abnormal DE area, 0.00824±0.0004 mm²/s (confidence interval 0.00771-0.00877 mm²/s) in neighbouring normal myocardium (NNM) close to abnormal DE area, and 0.00928±0.00049 mm²/s in remote normal myocardium (RNM) (confidence interval 0.00864-0.00992), P=0.01 or p = 0.02 respectively between abnormal DE area or normal myocardium close to abnormal DE area and remote myocardium (Fig. 6). On the opposite, absolute ADCs were not significantly different between abnormal DE area and normal myocardium close to abnormal DE area (P=0.08).
Both relative and absolute ADCs were normal in the 6 controls.

**Comparison of DWI and DE patterns**

A total of 90 involved segments was detected with DWI, and of 108 involved segments with DE. DWI and DE studies gave similar results for the extent of involvement (2.81 ± 0.62 and 3.37 ± 0.65 segments respectively, p>0.05, range, 1 to 17 segments for both). There was also a good correlation between the two methods in the number of segments involved (Spearman, \( y = (0.94) x + 0.72, r = 0.96, p < 0.0001 \)). Segment-by-segment comparison showed a high level of correlation (\( y = (0.91)x + 0.47, r = 0.9049, \) confidence interval 0.8219 to 0.9763, p < 0.0001).

Among the 31 segments missed at DWI, 17 were located in the inferior wall (segments 4,10 or 15), 8 in the anterior wall (segments 1, 7 or 13), 4 in the lateral wall (segments 5 or 6) and 2 in the septum (segment 14).

Per-patient and-per-segment in controls, there was no false positive DWI study, resulting in a specificity of 100%.
Fig. 4: Four-chamber DWI image of the heart shows a subepicardial high intensity in the basal lateral wall (arrow)

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**Fig. 5:** Same patient. The lateral hypersignal spot in the basal lateral wall seen in Fig. 4 displays a decreased apparent diffusion coefficient, approximating one half that of the normal interventricular septum.

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Fig. 6: Box plot of absolute ADCs in ADE, NNM and RNM.

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Conclusion

MRI has recently become a reference examination test in suspected acute myocarditis, except in the fulminant forms. DWI imaging, despite yet very low resolution, is feasible within one minute examination, without use of contrast medium, and should provide additional rapid information in this disease, in particular when subepicardial enhancement is subtle on delayed-enhancement images. ADC maps as well as calculation of absolute ADC should help better understand inflammatory phenomena such as an underestimated extent of lesions by DE sequences. Hence, this sequence should help better appreciate the real extent of myocardial infection.

It is too early, however, to know if DWI should be integrated or replace as a single fast acquisition the comprehensive and more cumbersome sequences used in this disease.
