Case Report: Clinical Usability of MyoMaps in Myocardial Infarction

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Introduction

Cardiac magnetic resonance (CMR) imaging offers a lot of information about myocardial tissue characterization with high spatial resolution, high-quality imaging and contrast. In the past, the key sequences for tissue characterization have been T1-weighted imaging for scar (late gadolinium enhancement: LGE) and T2-weighted imaging for edema. However, because with these two methods clinical cases are evaluated by visual interpretation of relative signal intensities in relation to normal myocardium (no quantification), there has been no agreement on a specific definition of ‘abnormal signal’. Recent advances in CMR allow measurement and quantification of myocardial T1, T2, or T2* values (in ms) using short breathhold mapping sequences [1, 2]. The results are displayed in color-coded pixel maps where each pixel represents a physical estimate for T1, T2, or T2* in milliseconds. If the clinical context is known, T1, T2 and T2* mapping can provide useful information. Pre-contrast T1 (native T1) generally increases in conditions that increase total myocardial water, such as acute myocardial infarction (AMI), myocarditis, or stress cardiomyopathy. In addition, the myocardial extracellular volume (ECV) can be calculated from pre- and post-contrast T1 values of myocardial tissue and the blood pool together with the patients’ hematocrit [1, 3]. It can quantify the myocardial extracellular space essentially without dependence on magnetic field intensity, amount of contrast medium and imaging parameters. T2 mapping depicts myocardial edema of acute myocardial infarction or myocarditis, while T2* mapping allows early detection of iron overload as in thalassemia.

MyoMaps is a new approach for quantification of myocardial tissue characteristics by Siemens Healthcare. Based on HeartFreeze Inline Motion Correction, MyoMaps provides pixel-based myocardial quantification for T1, T2 and T2*, on the fly [4, 5]. Pixel-based myocardial quantification and color mapping techniques enable a more accurate diagnostic characterization of cardiac territories.

CMR sequences

CMR imaging was performed at 3T (MAGNETOM Skyra, Siemens Healthcare, Erlangen, Germany) with an 18-element surface coil. After the acquisition of scout images, Dark Blood T2w short axis images were obtained of the left ventricle using a Turbo SE sequence. Imaging parameters were as follows: TR 800 ms, TE 50 ms, flip angle 180°, slice thickness (SL) 6 mm, field-of-view

Case 1

Patient history

A 58-year-old male patient presented to our emergency department with chest pain and clammy sweat. ST-elevation of II, III, aVF was seen. Emergency invasive coronary angiography was performed, 90% stenosis was detected in right coronary artery (RCA) segment #2 (Fig. 1A). Percutaneous coronary intervention was performed in segment #2.

Imaging findings

CMR was performed for comprehensive myocardial assessment 6 days following onset. Short axis Dark Blood T2w (Fig. 1B) and LGE images (Fig. 1C) clearly demonstrate the presence of localized high intensity in inferior segment. With MyoMaps (Figs. 1D-F), the affected regions clearly demonstrate increased native T1 (1550 ms) and T2 (54 ms) values. Additionally, ECV was calculated for the same area as 67.6% compared to 25.2% for normal myocardium (T1 1210 ms, T2 36 ms).

Discussion

Imaging findings (Dark Blood T2w, LGE images) well agree with clinical course for inferior AMI (day 6 from onset, culprit vessel: RCA #2). Color images of T1 and T2 map clearly demonstrate the lesion. This case is a relatively localized lesion, where the native T1 maps demonstrate equivalent visual diagnostic capability to delayed-enhancement images. In addition, quantitative assessment (T1, T2 map and ECV) suggests edematous change and myocardial fibrosis in the area of the lesion. Thus, for this relatively localized lesion, the quantitative assessment allowed an accurate evaluation of myocardial tissue characteristics to support the diagnosis.
370 mm, matrix size 256 x 75%, voxel size 1.4 x 1.4 x 6 mm³, parallel imaging acceleration factor 2, ECG trigger pulse 2; turbo factor 13, fat sat, SPAIR. A gadolinium-based contrast agent (gadopentetate dimeglumine, Magnevist; Schering, Berlin, Germany) was administered intravenously at 0.1 mmol/kg body weight. LGE images were obtained using a TrueFISP IR single-shot and phase sensitive inversion recovery (PSIR) sequence about 10 min after the administration of contrast. The imaging parameters were as follows: TR 852 ms, TE 1.26 ms, echo spacing 3 ms; inversion time, measured by TI scout, flip angle 55°, SL 6 mm, FOV 350 mm, matrix size 224 x 65%, voxel size 1.6 x 1.6 mm, iPAT 2, ECG trigger pulse 2.

Pre-contrast T1 maps were obtained from three short-axis images (basal, mid, and apical) of the left ventricle using single shot TrueFISP based on modified look-locker inversion-recovery (MOLLI) sequence. The imaging parameters were as follows: TR 280.56 ms, TE 1.12 ms, echo spacing 2.7 ms, flip angle 35°, SL 8 mm, FOV 360 mm, matrix size 256 x 66%, voxel size 1.4 x 1.4 x 8 mm³, iPAT 2, MOLLI type 5(3)3 for long T1. Post-contrast T1 maps were obtained in the same locations as the pre-contrast T1 maps. The imaging parameters were as follows: TR 360.56 ms, TE 1.12 ms, echo spacing 2.7 ms, flip angle 35°, SL 8 mm, FOV 360 mm, matrix size 192 x 75%; voxel size 1.9 x 1.9 x 8 mm³, iPAT2, T2 prep., duration 0, 30, 55 ms.

Conclusion

T1 and T2 mapping not only enables visual diagnosis but also offers pixel-based physical quantification of myocardial tissue characteristics in myocardial infarction. Thus, in our institutions, the number of MyoMaps examinations has increased over the recent months. Some of the reasons for this are:

1. Few burdens on patients (a single, short breathhold)
2. Non-contrast imaging for renal failure patients (Native T1 or T2 map)
3. Diagnosis of non-ischemic cardiomyopathy (assessment of minute lesions based on pixel-based color mapping)
4. Diagnosis of diffuse myocardial impairment (quantification)
5. Supporting MR image interpretation (objectivity vs quantification)

Current advances in T1- and T2-mapping and ECV quantification might have the potential to improve the diagnosis of cardiovascular disease, refine myocardial risk stratification and guide patient personalized therapeutic strategies.

References


Continued on page 30.
Case 2

Patient history
A 66-year-old male patient presented to our emergency department with chest pain. High serum levels of CK (303 IU/L) and CK-MB (19.0) were seen. Acute myocardial infarction was suspected and emergency invasive coronary angiography was performed. 99% stenosis was detected in left coronary artery (LAD) segment #6 (Fig. 2A). Percutaneous coronary intervention was performed in segment #6.

Imaging findings
CMR was performed for myocardial viability assessment after 10 days of onset. Dark Blood T2-weighted imaging (Fig. 2B) shows slow moving blood signal (slow flow artifacts) in anterior-septal segments. LGE image (Fig. 2C) demonstrates transmural enhancement in the same segments, and subendocardial low intensity in the septal region. In MyoMaps (Figs. 2D-F), slow flow artifacts are not found in T2 map, and mild myocardial T2 prolongation is shown in anterior-septal segments. The enhanced regions demonstrate increased native T1 (1633 ms) and ECV (55.1%) compared to normal myocardium (native T1 1180 ms, ECV 25.7%). On the other hand, native T1 of subendocardial low intensity area (1200 ms) is approximately equivalent to normal myocardium.

Discussion
The findings on the Dark Blood T2w images are consistent with anterior and septal subacute myocardial infarction (culprit vessel: LAD #6). However, we may overestimate an edematous region due to slow flow artifacts by the cardiac hypofunction in Dark Blood T2w images. T2 map images do not show this artifact and are thus valuable for the correct diagnosis of the edematous region. In quantitative assessment, increased native T1 and ECV suggest myocardial fibrosis and edema. A subendocardial linear area of low signal intensity is shown within the delayed enhanced lesion, suggesting microvascular obstruction (MO). T1 is generally lower in hemorrhage or iron accumulation, but in this case, native T1 value of MO is approximately equivalent to normal myocardium (1200 ms and 1180 ms). It seems that simultaneous occurrence of both fibrosis and iron deposition offset each T1 prolongation effect and T1 shortening effect. In this patient emergency stent insertion was performed. Nonetheless, findings of not only transmural infarction but also MO in this case suggest myocardial viability of infarcted segment to be poor.

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A 66-year-old man, septal subacute myocardial infarction. (2A) Left coronary artery (LAD) segment #6. (2B) DB T2w with fat sat. TSE_ELT 13, SL 6 mm, FOV 312 x 370 mm, matrix 162 x 256, TR 800 ms, TE 50 ms. (2C) LGE image. TFI_segment 67, SL 6 mm, FOV 319 x 350 mm, matrix 133 x 224, TI 400 ms, TR 852 ms, TE 1.26 ms, TR/TE 2RR/1.3 ms. (2D) Native T1 map. IR Tfi with MOCO, SL 6 mm, FOV 307 x 360 mm, matrix 144 x 256. (2E) Native T2 map. T2prep Tfi with MOCO. SL 6 mm, FOV 289 x 360 mm, matrix 116 x 192. (2F) Post-contrast T1 map. IR Tfi with MOCO. SL 6 mm, FOV 307 x 360 mm, matrix 144 x 256.