Quantitative Detection of Myocardial Edema using a Breath-hold T2 Mapping Pulse Sequence

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Introduction: An inflammatory response to various diseases, including acute myocardial ischemia, cardiac transplantation rejection and acute myocarditis, results in water accumulation in the myocardium. Excess water accumulation results in myocardial edema, which can lead to various conditions including myocardial stiffness, diastolic dysfunction, and tissue swelling [1]. Current methods for diagnosis of myocardial edema pose several challenges. Echocardiography is often used initially but findings are frequently nonspecific and not apparent until significant irreversible damage has occurred. Pathologic diagnosis is the gold standard but carries risks secondary to invasiveness of endomyocardial biopsy. Conventional cardiac T_2-weighted (T_2w) MRI has emerged as the diagnostic test of choice and can be used to qualitatively detect myocardial edema. While this method enables detection of myocardial edema, surface coil effects often yield non-uniform signals which may hinder interpretation [2]. We propose to quantitatively detect myocardial edema using a breath-hold T_2 mapping pulse sequence based on multi-echo, spin-echo (ME-SE) imaging [3]. The purposes of this study were to validate the quantitative T_2 mapping pulse sequence against the qualitative T_2w pulse sequence in patients with clinical evidence of cardiac disease and to correlate T_2 mapping with delayed contrast-enhancement (DCE) imaging in a patient with acute myocardial infarction.

Methods: For validation, we imaged 7 female patients with various types of heart disease (see Table 1 for clinical history) on a 1.5T whole-body MR scanner (Siemens; Avanto) equipped with a standard coil array, using both the conventional T_2w and ME-SE T_2 mapping pulse sequences in 3 short-axis planes of the heart. The relevant imaging parameters for the T_2 mapping sequence are: spatial resolution=2 mm x 2 mm x 8 mm, echo spacing = 4.5 ms, turbo factor=4, number of images=8, fat saturation, double-inversion black-blood preparation pulse, and breath-hold duration=13 s. Conventional T_2w images were qualitatively evaluated by a cardiologist for presence of myocardial edema. For the ME-SE data, myocardial contours were segmented manually using short-axis planes, and the corresponding pixel-by-pixel T_2 maps were calculated by non-linear least square fitting for three parameters of the mono-exponential relaxation equation. The three unknown parameters were: initial signal amplitude, T_2, and background noise. For each short-axis plane, T_2 values were averaged over the entire myocardium. The reported T_2 represents mean T_2 averaged over the three short-axis planes. An upper limit cutoff T_2 value of 62.9ms (5 standard deviations [SD] above the mean) was chosen based on prior ME-SE data obtained from a control group [3]. For each patient, mean T_2 > 62.9 ms was considered as having myocardial edema. The accuracy of quantitative detection of myocardial edema was correlated with qualitative evaluation. The clinical reading and T_2 data analysis were performed independently in a blinded fashion.

For initial clinical application, using a 3T whole-body MR scanner (Tim Trio, Siemens) equipped with a standard coil array, we imaged one 38-year-old man who presented 5 days after onset of chest pain using the ME-SE T_2 mapping pulse sequence. Diagnostic x-ray coronary angiography performed 5 days before the MRI demonstrated an occluded distal posterior descending artery not suitable for percutaneous revascularization. Conventional cardiac MRI protocol was performed including DCE MRI with phase-sensitive inversion recovery reconstruction [4]. Specifically, DCE MRI was performed 10 min after administration of 0.2 mmol/kg of gadolinium-DTPA. We also imaged 5 normal control subjects (mean age = 28.4 ± 2.7 years), in order to determine an upper limit cutoff T_2 value at 3T for detection of edematous tissue in acute MI. The edematous area per short-axis plane was calculated as the number of pixels with T_2 > 5 SDs above the mean T_2 of control on the ME-SE data. For the DCE data, the infarct area per short-axis plane was calculated as the number of pixels with signal intensity > 5 SDs above the mean intensity of normal myocardium on the DCE data. Both edematous and infarct areas were normalized by their corresponding total number of pixels in the myocardium.

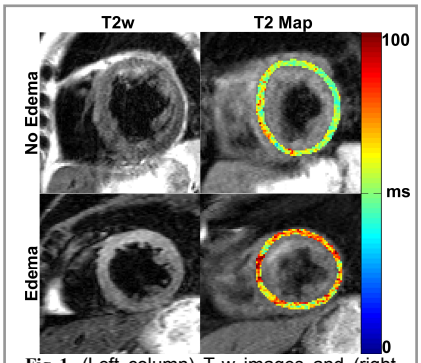

Fig 1. (Left column) T_2w images and (right column) T_2 maps acquired at 1.5T: (top row) patient without edema; (bottom row) patient with edema.

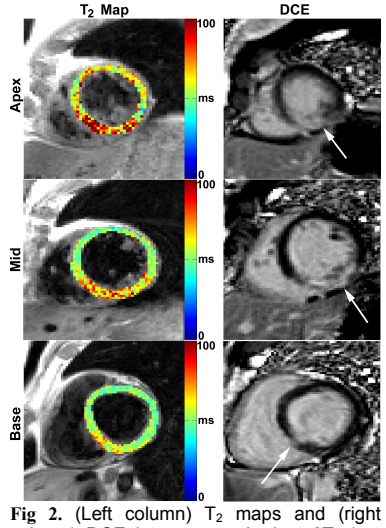

Fig 2. (Left column) T_2 maps and (right column) DCE images acquired at 3T: (top row) apex, (middle row) mid-ventricular, and (bottom row) base.

Results: Validation: Figure 1 shows T_2w images and corresponding T_2 maps acquired at 1.5T from a patient diagnosed with myocardial edema compared to those from a patient without edema. Three out of seven patients were diagnosed with myocardial edema based on increased signal intensity on conventional T_2w images and based on increased T_2 (> 62.9 ms) on ME-SE data (Table 1).

Application: The mean T_2 of the control group at 3T was 48.5 ± 3.7 ms, and consequently an upper limit cutoff T_2 value of 62.9 ms was used to detect edematous tissue. The percent edematous areas were 19, 30, and 10% for the apical, mid-ventricular, and basal short-axis planes, and the corresponding infarct areas were 17, 18, and 3%, respectively. Consistent with findings by Abdel-Aty et al [5], the increased T_2 regions coincided with, but were larger than hyper-intense "infarct" regions on DCE images (Figure 2).

Table 1: Clinical history, T_2 measurement, and diagnosis of myocardial edema for patients at 1.5T.

Patient Age (years)	Disease	T_2 (ms)	Diagnosis of Edema (T_2w/T_2)
23	sarcoidosis	60.9	No/No
65	acute coronary syndrome	67.1	Yes/Yes
78	chronic coronary artery disease	56.0	No/No
62	sarcoidosis	88.1	Yes/Yes
58	sarcoidosis	57.9	No/No
69	myocarditis	71.1	Yes/Yes
21	mildly elevated troponin with normal coronary	57.4	No/No

Discussion: This study demonstrates the feasibility of quantitatively detecting myocardial edema using a breath-hold ME-SE T_2 mapping pulse sequence. Clinical evaluation for myocardial edema is challenging with conventional T_2w imaging due to non-uniform signal intensities and lack of a normal myocardium reference signal. The use of T_2 mapping can help alleviate these difficulties. Future directions for this research include comprehensive evaluation of T_2 values with specific cardiac conditions and the clinical utility of T_2 mapping for assessment of myocardial edema.

References:

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