Measurement of Extracellular Volume Fraction by Cardiac Magnetic Resonance Imaging Detects Diffuse Myocardial Fibrosis in Hypertrophic cardiomyopathy

Aya Kino, Darshit Thakrar, Rahul Rustogi, Brandon Benefield, Jeremy D Collins, Daniel Lee, Lubna Choudhury, and James C Carr

1Radiology, Northwestern University, Chicago, IL, United States, 2Radiology, Northwestern University, Medicine Cardiology Division, Northwestern, 4Medicine Cardiology Division, Northwestern University

Introduction

Late gadolinium enhancement cardiac magnetic resonance (LGE CMR) imaging has been used to detect myocardial hypertrophy and scar/fibrosis in Hypertrophic cardiomyopathy (HCM) patients. The presence of hyperenhancement has been associated with progressive ventricular dilation, ventricular arrhythmias and clinical risk factors for sudden cardiac death. Recently studies have shown that myocardial extravascular extracellular volume fraction (Ve) measures quantify diffuse fibrosis not readily detectable by conventional late gadolinium (Gd) enhancement (LGE) in ischemic cardiomyopathy patients.

The purpose of the study is to determine if patients with HCM demonstrate higher myocardial extravascular extracellular volume fractions in regions of scar/fibrosis obtained by using a single-shot modified Look Locker inversion recovery sequence (MOLLI) with balanced SSFP MR Sequence compared to published Ve values from normal myocardium.

Material and Methods

Ten patients with known history of HCM underwent LGE CMR images using a 2D PSIR TurboFLASH protocol after administration of 0.2 mmol of gadopentetate dimeglumine per kilogram of body weight. A series of T1 weighted images were acquired on a 1.5 T Siemens scanner (MAGNETOM Avanto, Siemens Healthcare) using a single-shot modified Look Locker inversion recovery sequence (MOLLI) with balanced SSFP before and 10 minutes after the contrast injection. Ve measures were calculated as described by Jerosch-Herold et al.[1], in areas with focal LGE, diffuse/patchy LGE, areas without LGE but increased myocardial wall thickness (wt) (<25mm) and areas without LGE and normal myocardial wall thickness (Fig1). The student’s t test was used to compare Ve measurements.

Results

All patients were successfully scanned. LGE focal scar was found in 3 patients, diffuse scar was detected in all patients and 1 patient demonstrated a region with myocardial hypertrophy without detectable scar. Mean Ve values for these different myocardial regions are shown on Table 1. Areas with focal and diffuse scarring on LGE presented significant higher Ve values (p = 0.0015 and p = 0.0415 respectively). Areas presenting myocardial thickening (<30mm) but no visible LGE presented a higher Ve as well compared to the areas with normal WT and no LGE (p=0.0004).

Conclusion

This study demonstrates the utility of CMR T1 mapping for in vivo identification of diffuse interstitial myocardial fibrosis in HCM patients which is represented by myocardial extravascular extracellular volume fraction (Ve) in addition to the visible areas of fibrosis by LGE and even when no fibrosis is apparent by LGE.

References:


Table 1

<table>
<thead>
<tr>
<th>No LGE and no WT</th>
<th>No LGE and WT +</th>
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<tbody>
<tr>
<td>23.988 (±3.6)</td>
<td>38.68</td>
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<tr>
<td>Focal scar on LGE</td>
<td>Diffuse scar on LGE</td>
</tr>
<tr>
<td>48.845 (±14.7)</td>
<td>28.643 (±6.1)</td>
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Figure 1: CMR T1 MOLLI mapping images from a 47 years old HCM presenting a septal hypertrophy (maximal WT=33 mm) and focal and diffuse scar in the LGE MR images.