

POSTER PRESENTATION

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Diffuse myocardial fibrosis in early forms of dilated cardiomyopathy: insights from T1 mapping cardiovascular magnetic resonance

Fabian aus dem Siepen^{1*}, Sebastian A Seitz¹, Mohamed A Abdelrazek², Evangelos Giannitsis¹, Hugo A Katus¹, Henning Steen¹, Hassan Abdel-Aty¹

From 16th Annual SCMR Scientific Sessions San Francisco, CA, USA. 31 January - 3 February 2013

Background

The underlying myocardial injury in early forms of dilated cardiomyopathy (DCM) is unclear. Cardiovascular magnetic resonance (CMR) myocardial T1-mapping monitors the enlargement of the cardiac extra-cellular space and thus offers a non-invasive tool of quantitative measurement of myocardial fibrosis. We used T1-mapping techniques to explore the hypothesis that diffuse myocardial fibrosis is related to DCM.

Methods

We investigated 77 DCM patients (55 \pm years, 51 males) and 46 healthy controls (52 ± years, 23 males). Significant coronary artery disease was excluded invasively. All CMR examinations were performed using 1.5 T CMR scanner (Philips Achieva). Short axis slices covering the LV were acquired using SSFP sequence to measure volumes and ejection fraction. T1-relaxation times were measured before and 15 minutes after Gd-DTPA (Magnograf) injection (0.2 mmol/kg). T1-maps were created out of 11 midventricular short axis views with increasing inversion times (TI;100 - 4400 msec.) using a single breath-hold modified Look Locker inversion recovery sequence (MOLLI, TR/TE=3.5/1.8 msec, flip angle = 35°) in late diastole. Early DCM with mild reduction of LVEF was defined as EF between 45-55% while severely depressed LVEF was defined as EF<30%. Delta-T1 was calculated as non-contrast myocardial T1-post-contrast myocardial T1.

Results

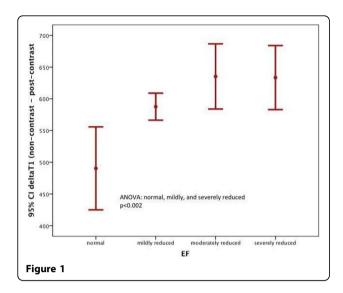
Mean LVEF was $43 \pm 12\%$ in DCM and 62 ± 4 in controls. Compared to controls, deltaT1 was significantly higher in 42 early DCM patients (588 \pm 60 ms; vs. 490 \pm 220ms; p=0.006). Patients with advanced DCM (n=10) had the highest delta-T1-values (634 ± 71 ms). Non-contrast-T1 $(371 \pm 164 \text{ ms vs. } 399 \pm 41 \text{ ms; p =ns})$ and post-contrast-T1 (1018 \pm 40 ms vs. 1037 \pm 45 msp=ns) were not significantly different between controls and patients with advanced DCM. ANOVA analysis showed significant differences between the groups (p<0.002, Figure 1). There was no significant association between the presence of late gadolinium enhancement (LGE) and the severity of LV dysfunction (31% of the patients in early DCM and 45% in severely depressed EF group; p=0.5). No significant differences in T1-values (non-contrast-, post-contrast- or delta-T1) between moderately and severely depressed EF were found (p=0.9).

Conclusions

Diffuse myocardial fibrosis commences already at an early disease stage of DCM when LVEF is only mildly reduced. In contrast to focal fibrosis seen in LGE, diffuse fibrosis is more linked to the severity of LV dysfunction. Importantly, the differences between T1-values before and after contrast administration (delta-T1) appear to be a more reliable parameter in the assessment of myocardial fibrosis than a single time-point measurement.

¹Universitatsklinikum Heidelberg, Heidelberg, Germany Full list of author information is available at the end of the article





Funding

none.

Author details

¹Universitatsklinikum Heidelberg, Heidelberg, Germany. ²Radiology, Cairo university, Faculty of Medicine, Cairo, Egypt.

Published: 30 January 2013

doi:10.1186/1532-429X-15-S1-P147

Cite this article as: aus dem Siepen *et al.*: Diffuse myocardial fibrosis in early forms of dilated cardiomyopathy: insights from T1 mapping cardiovascular magnetic resonance. *Journal of Cardiovascular Magnetic Resonance* 2013 **15**(Suppl 1):P147.

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