Myocardial Velocity and T2 Mapping Reveals Changes in LV Structure and Function after Heart Transplantation

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Introduction: Monitoring asymptomatic post cardiac transplant rejection remains problematic and relies on cardiac catheterization with myocardial as biopsy. Assessment of important functional cardiac parameter such as diastolic (dys)function is poorly reproducible by echocardiography. We propose spatially co-registered T2 mapping and myocardial velocity mapping (MVM) as valid reproducible and non-invasive methods which provide information about underlying structural and functional changes in post–transplant myocardium. T2 mapping provides a quantitative measure of myocardial edema, while MVM involves tridirectional velocity measurement assessing diastolic (dys)function covering entire left ventricle

Methods: Study cohort: Seven patients post cardiac transplantation (TX, age=52 ± 11years, 2 female) were examined with both myocardial velocity mapping and T2-mapping. The time between transplantation and MRI was variable (mean=29 months, min=3 months, max=51 months). At the time of the MRI exam n=6 patients showed no clinical symptoms and no history of biopsy proven cellular rejection. One patient had acute cellular rejection in the past (8 months prior; biopsy grade 2R). Patient data were compared to previously acquired control cohorts for T2-mapping (14 normal volunteers [3] and MVM (20 age matched controls, age = 52±4) [4].
All measurements were performed on 1.5T MR system (Espree, Erlangen, Germany). **T2-mapping:** T2 relaxation measurements were obtained from the left ventricle during diastole. Three T2-prepared SSFP images with varying T2-prep times (0, 24 and 55 ms) were acquired in a breath-hold fashion in 3 short-axis orientations (basal, mid, apical). Integrated image registration was utilized to eliminate subtle respiratory and cardiac motion. Automated pixel-wise fit was carried out to generate a T2 color map. Mean regional T2 values were calculated by manually drawing ventricular borders in 16 short axis segments. **Myocardial velocity mapping:** For the same 3 short-axis slices, MVM was performed based on an ECG gated black-blood prepared phase-contrast sequence with 3-directional velocity encoding (venc=25cm/s, temporal resolution 25ms; spatial resolution =2.9x2.4mm, slice thickness = 8mm, flip angle = 15^o). Spatio-temporal imaging acceleration (k-t parallel imaging PEAK GRAPPA [2]) with a net acceleration factor of R_{net}=3.6 allowing data acquisition during breath holding. Data analysis (Matlab, The Mathworks, USA) included manual segmentation of the LV contours, correction for background phase offsets, and transformation of the measured three-directional velocities (v_x,v_y,v_z) into radial(v_r), rotational (v_{phi}) and long-axis (v_z) velocities. For all velocity components, systolic and diastolic peak radial velocities were derived. The American Heart Association 16 segment model was used forl analysis for both MVM and T2 mapping allowing direct comparison. MVM data analysis was performed by 2 independent readers to evaluate observer variability. Agreement between readers was assess by Bland-Altman analysis. Variation between observers was calculated as (range of limits of agreement, 2 SD) / range of peak velocities x 100.

Results: In TX patients, the range of peak systolic and diastolic myocardial velocities was v_r = [-8.4m/s, 5.7m/s] for radial velocities and v_z = [-9.4m/s, 10.0m/s.] for long-axis motion. As shown in fig.1, Bland Altman analysis shows very good inter-observer agreement with low inter-observer variation of 6.7% for peak radial velocities and of 4.8% peak long-axis velocities. For both T2-mapping and MVM, substantial regional differences between controls and transplant patients relating to left ventricular structure and diastolic function were seen. Increased and more heterogeneous segmental T2 distribution (Fig 2) (indicating structural changes such as edema) and reduced diastolic peak radial and long axis velocities (Fig 3) (representing functional myocardial changes) were seen in transplant patients.

Discussion: T2 mapping and myocardial velocity mapping are proposed as reproducible and non-invasive methods of detecting underlying disordered structure and function respectively. One patient with remote rejection with biopsy grade 0R (by the time of MRI exam) showed reduced and more heterogeneous myocardial T2 and velocity values suggestive of permanent structural changes in myocardium even after acute changes of rejection were resolved. Limitations include small cohort, no subject with acute rejection, and non- age/gender matched normal cohort for T2 mapping..

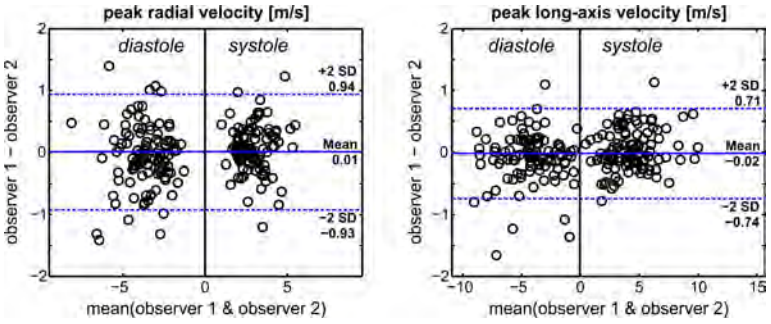

Fig. 1: Bland-Altman analysis of inter-observer variability for peak myocardial velocities in the 16-segment model in n=7 patients after heart transplantation. Both graphs show very good agreement as reflected by only very minor mean differences (0.01 and -0.0s m/s) and low observer differences (limits of agreement, ± 2SD) relative to the range of peak velocities.

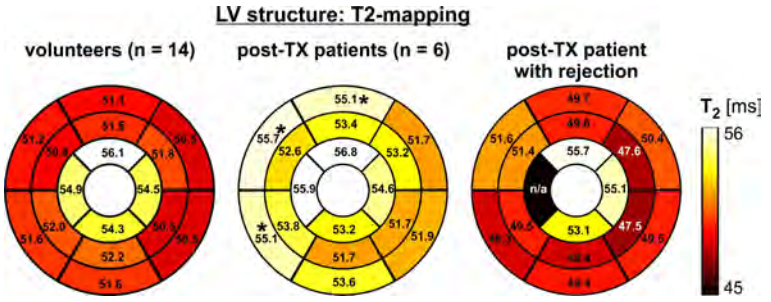

Fig. 2: LV mapping of T2-relaxation times(AHA 16-segment model) in age matched normal control (left), a group of 6 patients after heart transplantation with no clinical symptoms and one patient who presented with a previous acute cellular rejection (right). Transplant patients showed generally increased T2 in most LV segments. * significant differences (2-sided t-test, p<0.05).

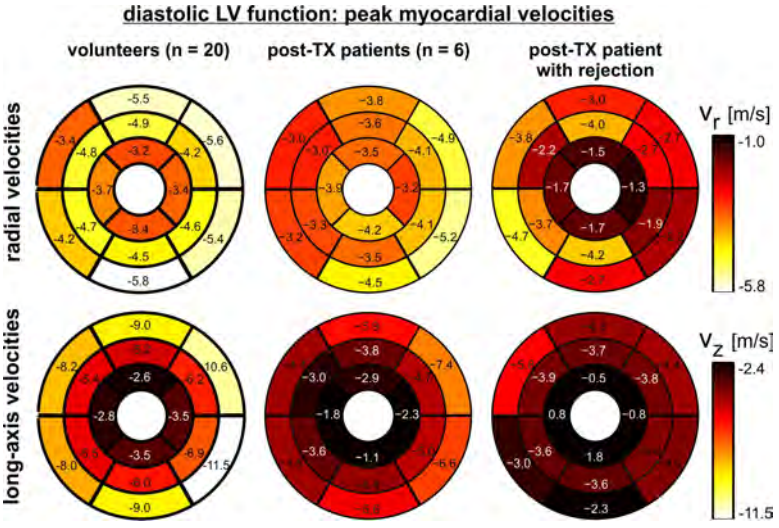

Fig. 3: Diastolic left ventricular function in age matched normal control (left), a group of 6 patients after heart transplantation with no clinical symptoms and one patient who presented with a previous acute cellular rejection (right). The individual plots show the distribution of peak radial (v_r) and long-axis (v_z) myocardial velocities in the AHA 16-segement model.

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