Myocardial T1 and T2 measurement in patients with cardiac amyloid and comparison with normal controls

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***Introduction*:** Cardiac involvement is frequent in systemic amyloidosis of immunoglobulin light chain (AL) and transthyretin (TTR) types and is a major determinant of treatment options and prognosis. Noninvasive diagnosis of cardiac amyloidosis is challenging. Cardiac MRI is often helpful, in particular visualization of diffuse myocardial delayed enhancement and abnormal myocardial nulling after gadolinium administration. Many patients with systemic amyloidosis, however, have renal involvement, reduced renal function, and are unable to receive gadolinium contrast agents. Recently it has been suggested that myocardial T1 is shorter in patients with cardiac amyloid. We investigated whether characterization of myocardial T1 and T2 could help distinguish patients with cardiac amyloid from a group of control patients.

Materials and Methods: All data were acquired using a GE 1.5T Signa Excite system (GE Healthcare, Waukesha, WI). Myocardial T1 measurements were performed using an ecg-gated fast gradient echo Look-Locker (Cine-IR) sequence with the following parameters: TR/TE 6.0/2.8ms, flip angle 12, bandwidth 32 kHz, matrix 128x128, 10mm slice thickness, NSA 1, 8 views per segment. T2 measurements were performed using an ecg-gated 4 echo fast spin echo sequence with TE's approximately 6, 42, 80, and 120 ms. Additional parameters included flip angle 90, matrix 224x128, slice thickness 8 mm, NSA 1. A single mid-ventricle short axis slice was prescribed from localizing 2-chamber and 4-chamber views. T1 and T2 measurements were performed using investigational software (Cinetool, GE Healthcare). A ROI was drawn in the ventricular septum and copied to the remaining phases or echoes, generating a plot of signal intensity versus TI for T1 measurements or SI vs TE for T2 measurements. Resulting plots were solved for T1 or T2 by fitting to single exponential decay functions. Measurements were performed in 14 patients with known systemic amyloidosis and both echocardiographic and contrast-enhanced MRI examinations positive for cardiac amyloid. The control group consisted of 14 patients (3 healthy volunteers, 4 patients with negative exams and relatives with suspected ARVC or HCM, 2 patients with vascular aneurysms and no cardiac symptoms, 2 patients with suspected coronary artery anomaly and negative exam, and 3 patients with atypical chest pain and negative exam).

***Results and Discussion*:** Average myocardial T2 was 57 $\pm$ 10 ms for amyloid patients and 49 $\pm$ 11ms for the control group (Fig. 1A). This difference was not statistically significant ($p = 0.09$). Average myocardial T1 was 455 + 107 for the amyloid group and 390 + 72 for the control group (Fig. 1B). This difference was also not statistically significant ($p = 0.18$).
There was a trend toward higher T1 and T2 measurements in the amyloid patients, but this did not reach statistical significance. While it is possible that a larger study would be able to separate small intrinsic differences in myocardial relaxation times, these results suggest that distinguishing patients with cardiac amyloid would be difficult based on myocardial relaxation measurements alone.

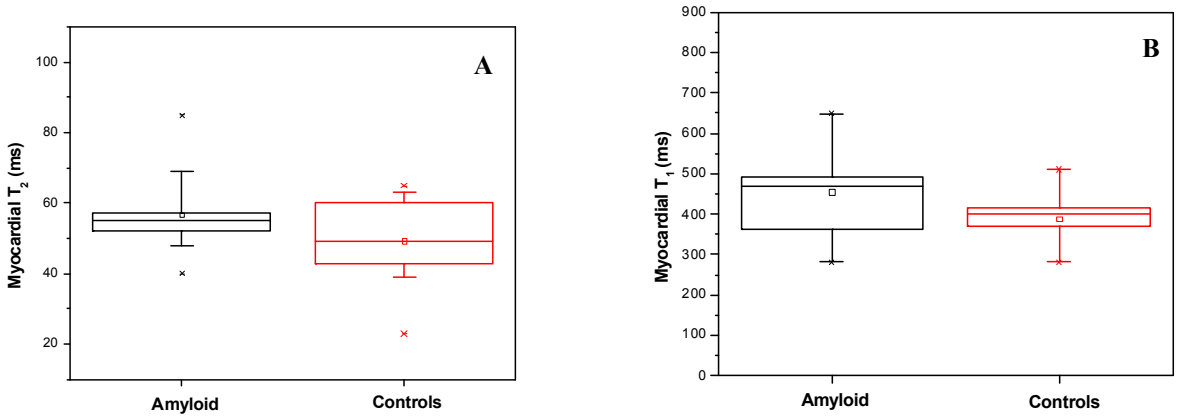

Fig. 1. Box and whiskers plot of septal myocardial T2 (A) and T1 (B) for amyloid and control patients.