Comparing In vivo T2* and T2 Measurements in Tissues of Liver and Heart in thalassemia

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Introduction: Cardiac complications secondary to iron overload are the leading cause of death in thalassemia major (TM). Cardiovascular magnetic resonance (CMR) can provide a noninvasive means of measuring the amount of tissue iron. With CMR, tissue iron is detected indirectly by the effect on relaxation times of hydrogen nuclei in the presence of ferritin and hemosiderin iron. The iron results in shortening of proton relaxation times and both CMR T2* and T2 have been validated as non-invasive means for assessment of iron overload in tissues of liver and heart. (1,2,3,4,5,6). A recent study (6) has shown that both liver T2* and T2 can accurately measure hepatic iron overload in thalassemia. We have demonstrated the linear correlation between myocardial T2 and T2* in patients with iron overload (7) indicating that iron loading becomes the dominant effect in determining the CMR T2* and T2 in the heart. However, there is currently little data on comparing this relationship of T2*/T2 between tissues of liver and heart. Although the mechanisms of iron-enhanced relaxation may be similar in these two human organs, the exact manner can differ due to biological differences.

It is of clinical interest to compare the T2*/T2 relationship between tissues of liver and heart. In this study therefore, we are aiming at the direct comparison of myocardial T2*/T2 measurements in vivo in order to establish the relationship between them and compare this relationship in the tissues of liver and heart. We hypothesize that T2 will correlate with T2* linearly when iron becomes dominant in the liver similar to our previous findings in the heart (7); this relationship, however, can be different between different tissues of liver and heart.

Material and Methods: 50 TM patients (age 25±23 years old, 27 males) were studied on a 1.5T MRI scanner (Siemens Sonata) using a cardiac phased array coil and with ECG gating. All patients were scanned using the T2* and the T2 sequence subsequently, each within a breath-hold. For heart scan, a single mid-ventricular short axis slice was acquired with both T2* and T2 measured in the left ventricular septum (Figure. 1). For liver scan, a single trans-axial slice through the center of the liver was imaged without cardiac gating. Signal intensity analysis was performed in the periphery of the liver away from the large central vessels (Figure 2). The mono-exponential decay model and the nonlinear curve fitting algorithm were employed for the patient data analysis (7,8) (CMRtools, Cardiovascular Imaging Solutions, London).

Results: Figure 3 shows the relationship between myocardial T2 and T2* from all patients, while Figure 4 the relationship between hepatic T2 and T2*. The vertical broken lines represent the threshold T2* values (20ms for myocardium and 6.3ms for liver) to distinguish abnormal and normal patients (1). There was clearly a good linear correlation between T2* and T2 measurements in the heart (Fig. 3. trend line, $R^2 = 0.932$) and liver (Fig. 4. trend line, $R^2 = 0.698$) in iron overloaded patients. In the normal group (T2*>20ms for heart and T2*>6.3 for liver), however, no linear correlation between these two measurements was found. The results demonstrate linear T2/T2* correlations in both heart and liver with iron overload, this T2/T2* relationship in the liver differ from that of heart

Conclusions: CMR T2* is a useful tool to identify patients with cardiac siderosis both in the liver and heart. Myocardial T2 measurement correlated linearly with T2* measurements in TM patients with iron overload, suggesting that both T2* and T2 can be used for assessment of iron overload in the tissues of liver and heart. The T2/T2* correlation in the liver differ from that of heart likely caused by biological difference, suggesting the calibration results of T2*/T2 against biopsy in the liver may not be directly transferred to the heart.

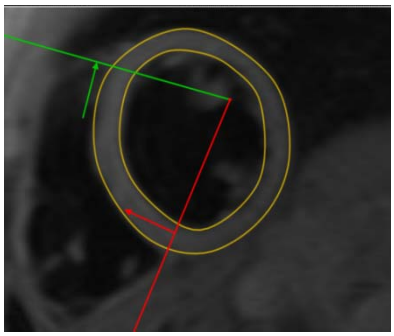

Fig 1. Myocardial T2/T2* measurements in the septum

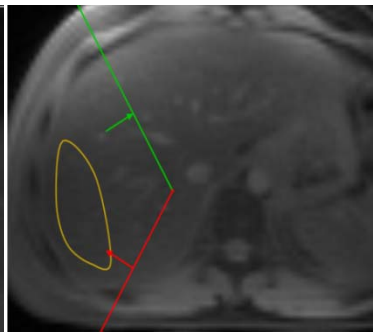

Fig 2. T2/T2* measurements in the liver

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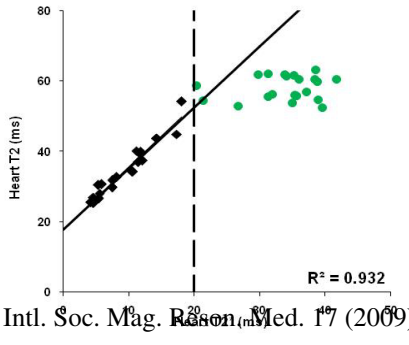

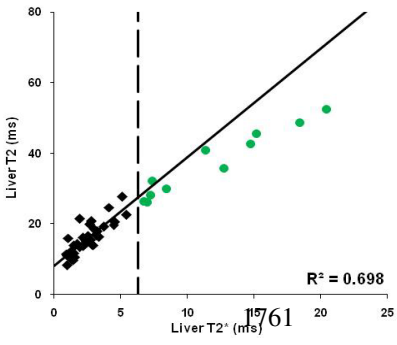

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