

Quantitative T2* MRI for Kidneys Iron Overload Assessment in a large cohort of thalassemia major patients.

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Introduction. Renal dysfunction has been reported in adult subjects with thalassemia since 1975 [1]. One of the main cause is the iron overload consequent to regular transfusions. Multiecho T2* MRI is a well-established technique for cardiac and hepatic iron overload assessment [2], but there very few report concerning the kidneys [3]. The aims of this study were to describe the T2* values of the kidneys in patients with thalassemia major (TM), to investigate the correlation between renal and myocardial or hepatic siderosis and to investigate the correlation between pancreatic iron overload and biventricular cardiac function.

Materials and Methods. 119 TM patients (58 men and 61 women, 12-51 years old, mean age 30.7 ± 8.2 years) enrolled in the Myocardial Iron Overload (MIOT) networks [4] underwent MRI (1.5T GE Signa/Excite HD, Milwaukee, WI, USA). For the measurement of iron overload, fast-gradient-echo multiecho T2* sequences were used [5]. The left ventricle was segmented into a 16-segments standardized model [6] and the T2* value on each segment was calculated as well as the global value. In the liver, the T2* value was assessed in a single region of interest (ROI) defined in a homogeneous area of the parenchyma. For each kidney, T2* values were calculated in three different ROIs and were averaged to obtain a representative value for the kidney. The mean T2* value over the kidneys was also calculated. Steady-state free procession cines were obtained to quantify biventricular morphological and functional parameters in a standard way using the MASS® software.

Results. The ANOVA test showed that the T2* values in the right kidney were significant lower than the T2* values in the left kidney (40.3 ± 11.9 vs 44.1 ± 12.7 , $P < 0.0001$).

The mean T2* value over the kidneys was 42.2 ± 11.9 ms and 40 patients (33.6%) had a pathological value ($T2^* < 36$ ms, lower limit of normal evaluated on 20 healthy subjects). The mean T2* value did not show a significant difference amongst men and women (men 43.2 ± 11.7 versus women 41.3 ± 12.1 , $P = 0.378$). The mean T2* values increased with age in a significant manner ($r = 0.321$, $P < 0.0001$).

There was a significant negative correlation between serum ferritin levels and mean renal T2* values ($r = -0.446$, $P < 0.0001$; figure 1 left). Significant positive correlations of the mean T2* values were demonstrated for liver ($r = 0.511$, $P < 0.0001$; figure 1 center) and global heart ($r = 0.262$, $P = 0.004$; figure 1 right) T2* values.

The Kidney T2* values were significantly correlated with the right atrial area ($r = 0.305$, $P = 0.003$). No correlation was found between renal iron overload and bi-ventricular function parameters.

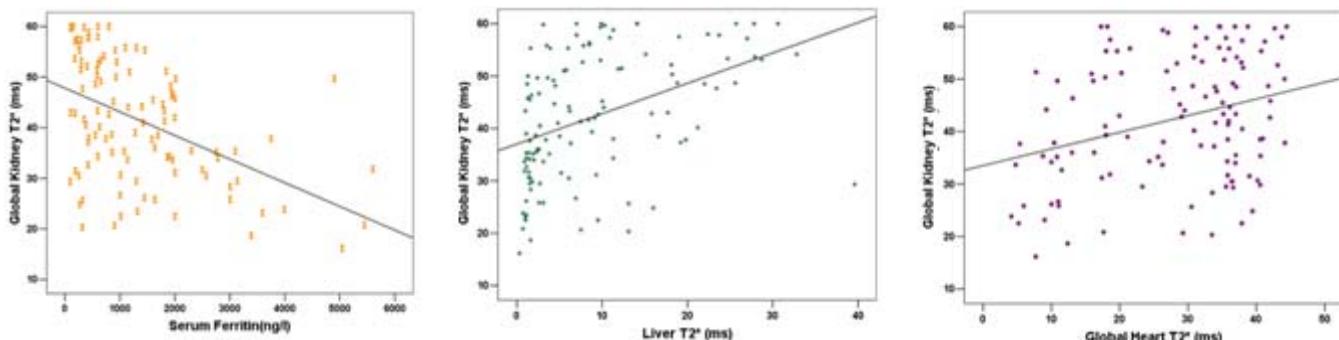


Figure 1: Scatter diagram of renal iron overload compared to serum ferritin (left), hepatic iron overload (center) and myocardial iron overload (right).

Conclusions. Systemic T2* differences between left and right kidneys were found, with significant lower values in the right one. Mean T2* value increased with age. We confirmed that kidney iron deposition was not very common in TM, but it was correlated with iron deposition in other organs (liver and heart).

References. [1] Mastrangelo F et al. Nephron 1975;143:229-236. [2] Cogliandro T et al. J Cardiovasc Med 2008;9(5):515-525. [3] Schein A et al. JMRI. 2008;28(3): 698-704. [4] Ramazzotti A et al. JMRI 2009;30(1):62-68. [5] Pepe A et al. JMRI 2006;23(5):662-668. [6] Cerqueira MD et al. Circulation 2002;105:539-542.