The value of severe liver iron overload to predict cardiac iron level

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Introduction: With the development of magnetic resonance imaging (MRI) techniques, it is feasible to assess iron overload in tissues especially the heart and liver in transfusion-dependent patients with thalassemia major(TM). Cardiac failure is one of the main causes of death in patients with thalassemia major[1,2]. The diagnosis of TM is often hysteretic in clinical practice, which renders unpredictability of cardiac iron deposition. Thus, it is vital to monitor the cardiac iron level even in asymptomatic patients. Hepatic iron concentration has been widely used as the indicator of this disease. However, it lacks extensive investigation about the relationship between hepatic iron overload and myocardium iron overload, especially when the liver has severe deposition.

Purpose: This study sought to investigate the relationship between the liver and heart iron overload in transfusion-dependent thalassemia patients. Whether hepatic iron can be served as an index to predict heart iron deposition has clinical value.

Methods: ***Subjects:*** 102 patients with TM (62 male, mean age 11.6±4.1 years) from July 2012 to September 2013 were included in this study. Consents were obtained for these MRI examinations from the participants. The myocardium T2* and hepatic T2* were measured for each patient.. ***MR imaging:*** All the participants underwent MR imaging on a 1.5T whole-body scanner (Intera, Philips Medical System, Best, The Netherlands) with 16-channel RF coil. A black blood 8-echo gradient-echo sequence, was used for the measurement of myocardial T2* within a single breath-hold. A single short-axis mid-ventricular slice with slice thickness of 10 mm, positioned halfway between the base and the apex of the left ventricle (LV) was scanned. Other imaging parameters were, TE/delta-TE=1.7/2.7ms, flip angle=20°, TFE factor=6, matrix=248×90, filed of view=400×262 mm². A second multi-slice multi-echo gradient echo sequence was applied for acquisition of liver T2* within a single breathhold. The following parameters were used: TE/deltaTE=0.85/0.9ms, flip angle=20°, matrix=160×65, filed of view=400×200 mm², slice thickness=10mm. ***Data analysis:*** A region of interest (ROI) encompassing a large homogeneous liver region excluding large blood vessels was drawn in liver parenchyma (Fig. 1). In the heart, a homogeneous ROI was chosen in the ventricular septum (Fig. 1).Then T2* values were calculated by measuring the mean signal within the ROIs. T2* values of the liver and heart were computed using CMR Tools software(Cardiovascular Imaging Solutions, London, UKEngland). Spearman's correlation was used to assess the correlation between the liver and heart R2* for the entire group data. To demonstrate these correlations we used scatter plots with a regression line. Further, we divided the data into two groups. Group A consisted of the patients with normal cardiac R2* with normal cardiac iron ($R2^* \leqslant 40$ sec^{-1} i.e. $T2^* \geq 25$ms)[3]. Group B consisted of these with cardiac iron overload ($R2^* \geq 40$ sec^{-1} i.e. $T2^* \leqslant 25$ ms). Student T-test was used to compare the hepatic R2* between group A and group B. And regression analysis was used to calculate the odds ratio (OR) and 95% confidence interval (CI) of the liver R2* with increment of 10 in discriminating presence of cardiac iron overload. Pearson correlation analysis was utilized to determine the relationship between the liver R2* and cardiac R2* in patients with cardiac iron overload.

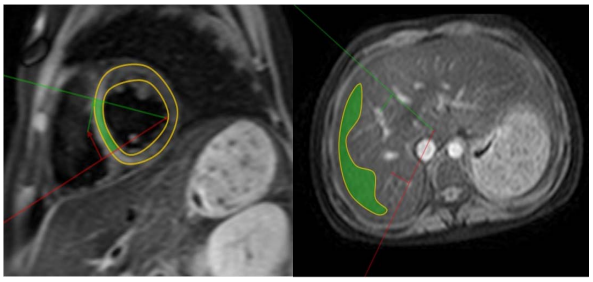

Fig. 1 the ROI of liver and cardiac images

Results:

Of 102 subjects, the mean value of liver T2* is 1.40ms, ranging from 0.61ms to 3.14 ms. 28 of them had the T2* less than 0.96ms while 74 less than 25ms. The T-test results are shown in Fig. 2. It is clear that the mean value of Group A (mean value of 696.5) is obviously lower than that of Group B (mean value 971.8). Based on these results, we conduct the logistic regression with the results of OR=1.032, (95% CI, from 1.016 to 1.049)(P<0.01). It suggests every liver R2* grows one step (the absolute step of R2* is 10), the probability of cardiac iron overload will become higher with the increment of 3.2%. Fig. 3 shows the correlation between the liver R2* and cardiac R2* (r=0.242, p=0.197>0.01).We can observe that there are no significant correlation between the liver R2* and cardiac R2*.

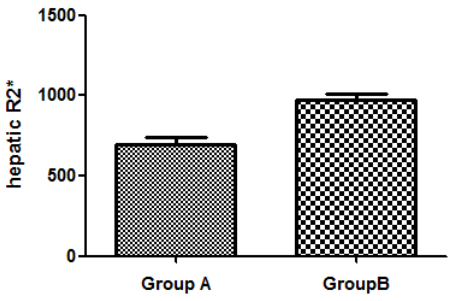

Fig. 2 T-tests of Group A and B

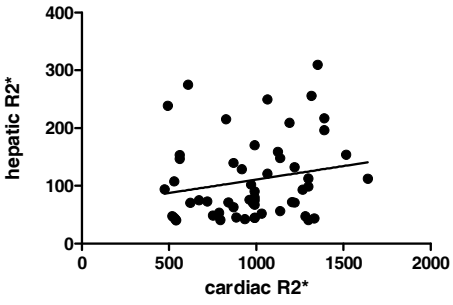

Fig. 3 The scatter diagram and regression line of liver R2* compared to the cardiac

Discussion and Conclusions:

This study investigated the correlation between hepatic and heart R2*. From the T-test results of group A and group B, it is obvious that the mean values of hepatic R2* of the two groups are significantly different, which consistent with the results of logistic regression of the two groups. In other words, with the increment of hepatic R2*, the probability of the occurrence of cardiac iron overload will become higher, which had not been mentioned in the previous papers[4,5]. It might be that the majority of the patients had severe liver iron overload in our study. Whereas, these findings suggest that the value of hepatic R2* has no significant linear correlation with that of heart. In conclusion, this study investigated the correlation between hepatic R2*and cardiac R2* in severe iron overload TM patients. And the hepatic R2*might be an effective indicator for occurrence of myocardial iron overload. Further accurate method of measuring T2* such as Ultra short TE sequence is needed to improve the results in severe liver iron overload patients.

References:

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