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[2601] CARDIAC IRON DEPOSITION IS NOT PREDICTED BY CONVENTIONAL MARKERS OF IRON OVERLOAD IN HOMOZYGOUS β-THALASSAEMIA.

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The management of iron overload requires the identification of patients at highest risk of heart failure, the commonest cause of death in thalassaemia major. Because it has not previously been possible to estimate cardiac iron deposition non-invasively and reproducibly, management has relied upon assessment of other variables. Thus while whole liver iron levels >15mg/g dry weight or serum ferritin values consistently >2500ng/l have been shown to predict patients at highest risk of cardiac death, it is not known how these variables relate to cardiac iron deposition. We report the relationship between myocardial and liver iron, measured by magnetic resonance, in 151 regularly transfused patients with homozygous β-thalassemia. We measured T2*, an inherent value of any tissue which is inversely related to iron concentration. This measurement gives a high degree of reproducibility in both the liver (coefficient of variation=3.3%) and the heart (coefficient of variation=5.0%). Liver T2* correlated with liver iron measured at biopsy ($r=0.93$, $p<0.0001$, $n=13$ for non-fibrotic samples and $r=0.81$, $p<0.0001$ and $n=30$ for all samples), but there was a striking discordance between myocardial iron deposition and liver iron or serum ferritin in many patients. Although the correlations between liver T2* and heart T2* ($r=0.19$, $p=0.02$) and serum ferritin and heart T2* ($r=0.14$, $p=0.01$) achieved statistical significance in this large cohort, the scatter was great. No significant relationship could be demonstrated between liver iron and left ventricular ejection fraction ($r=0.11$, $p=0.19$) or serum ferritin and left ventricular ejection fraction ($r=0.032$, $p=0.70$). There was also no significant relationship between mean serum ferritin for one year prior to the scan and myocardial T2* ($r=0.13$, $p=0.11$) or ejection fraction ($r=0.001$, $p=0.98$). The highly variable relationship between liver and heart iron is likely to reflect differences in chelation history between patients as well as differences in the rates at which iron is taken up and removed from hepatocytes and myocytes. We conclude that neither serum ferritin or liver iron measurements usefully predict those patients with increased myocardial iron deposition.

Keywords: β-Thalassemia; Iron overload

Poster Session: Thalassemia & Globin Gene Regulation: Switching/Clinical Studies (10:00 AM-6:30 PM)

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