

Poster presentation

Iron overload measurements by CMR in patients with suspected hemochromatosis - comparison of methods for T2* calculation in myocardium and liverCarlos E Rochitte*¹, Leonardo Sara¹, Afonso A Shiozaki¹, Gilberto Szarf², Roberto Blasbalg² and Dany Jasinowodolinski²Address: ¹Fleury Medicina e Saúde and Heart Institute-InCor-University of São Paulo Medical School, São Paulo, Brazil and ²Fleury Medicina e Saúde, São Paulo, Brazil

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from 13th Annual SCMR Scientific Sessions
Phoenix, AZ, USA. 21-24 January 2010

Published: 21 January 2010

Journal of Cardiovascular Magnetic Resonance 2010, **12**(Suppl 1):P285 doi:10.1186/1532-429X-12-S1-P285This abstract is available from: <http://jcmr-online.com/content/12/S1/P285>

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Introduction

Iron tissue overload can be detected non-invasively by CMR using T2* measurements techniques. Hemochromatosis is a disorder of iron metabolism that results in excess iron accumulation in tissues and organs. If left undiagnosed and untreated, iron overload can cause serious, sometimes fatal health problems. Early detection of iron overload and hemochromatosis treatment can prevent complications and prolong life.

Purpose

Our objective was to investigate T2* measurements in patients with suspected hemochromatosis and to compare different approaches on T2* calculation for the myocardium and liver.

Methods

We evaluated 42 consecutive patients with suspected hemochromatosis using CMR in a 1.5 T GE HDx scanner. We performed breath-hold fast GRE sequence on a short-axis view (including liver tissue in the same image) with several in-phase TEs (from 27.6 to 4.2 ms) and fixed TR of 31 ms to calculate T2* for the liver and myocardium using both, the entire signal intensity (SI) curve ($T2^* = 1/exp$) and only 2 points (9.6 and 4.2 ms) with the following equation: $-((9.6-4.2)/LN(SI @9.6/SI@4.2))$. We will call them curve and formula T2* calculations, respectively. For the liver iron overload we also used the technique

described by Rennes University, described in detail elsewhere <http://www.radio.univ-rennes1.fr/Sources/EN/HemoTech.html>.

Results

From the 42 patients with suspected hemochromatosis, 14 had a mutation of HFE gene detected (more commonly heterozygosis of C282Y or H63D). Mean serum ferritin level was $745.9 \pm 364.8 \mu\text{g/L}$ (NI < 300 for men and < 200 for women). Mean myocardial T2* was 32.1 ± 10.0 and 29.5 ± 11.3 ms for curve and formula calculations, respectively ($p = 0.04$). Mean liver T2* was 15.5 ± 6.8 and 15.2 ± 8.1 for curve and formula calculations, respectively ($p = 0.41$). Both methods correlated well for myocardium ($r = 0.74$, $p < 0.001$) and for liver ($r = 0.94$, $p < 0.001$). For myocardium a better correlation was seen for the T2* < 20 ms ($r = 0.87$, $p < 0.001$).

Bland-Altman analysis showed T2* mean difference of 2.6 (CI 0.1-5.0) and 0.4 ms (CI -0.6-1.3) for myocardium and liver comparisons, respectively, between curve and formula calculations. Most of cases had mild liver iron overload by Rennes method, with mean T2* of 15.4 ± 5.7 and maximal T2* of 31.5 ms (below the normal value of 35 ms), indicating good agreement.

Conclusion

Measurements of T2* in patients with suspected hemo-chromatosis could detect all ranges of myocardial and liver iron overload, and thus contributing to clinical management. The different approaches of myocardial and liver T2* calculations led to similar results, indicating that they could be used interchangeably. The 2 point formula approach could be a faster option for T2* image acquisition and calculation.

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