

Multislice Multiecho T2* MRI assessment of regional pancreatic iron overload and correlation with cardiac biventricular function and myocardial iron overload in thalassemia major patients.

G. Restaino¹, A. Meloni², A. Pepe², V. Positano², M. Missere¹, P. Pepe², D. De Marchi², G. Secchi³, A. Luciani⁴, G. Sallustio¹, and M. Lombardi²
¹Catholic University, Campobasso, Italy, ²MRI Lab, "G. Monasterio Foundation" and Institute of Clinical Physiology, CNR, Pisa, Italy, ³Azienda USL n° 1, Sassari, Italy, ⁴Istituto di Radiologia Az. Osp. "Garibaldi", Catania, Italy

Introduction: Thalassemia major (TM) patients require lifelong iron load monitoring to assess the effectiveness of chelation therapies. Multiecho T2* MRI is a well established technique for heart and liver iron overload assessment [1]. There are few reports on the use of MRI to study iron deposits in the pancreas. The aims of this study were to describe the T2* values of the pancreas in patients with TM, to investigate the correlation between pancreatic and myocardial siderosis and to investigate the correlation between pancreatic iron overload and biventricular cardiac function.

Materials and methods: 147 TM patients enrolled in the MIOT (Myocardial Iron Overload in Thalassemia) network [2] underwent MRI. For the measurement of myocardial iron overload, a fast-gradient-echo multislice multiecho T2* technique was used [3]. The left ventricle was segmented into a 16-segments standardized model [4] and the T2* value on each segment was calculated as well as the global value. Steady-state free precession cines were obtained to quantify biventricular morphological and functional parameters in a standard way using the MASS[®] software. For the measurement of pancreatic iron overload, a gradient-echo multislice multiecho T2* sequence was used. T2* measurements were performed in pancreatic head, body and tail. The global value was calculated as the mean.

Results: The ANOVA test showed regional differences in the regional pancreatic T2* values ($P < 0.005$). Specifically, the mean T2* over the pancreatic head was significantly higher than the mean T2* value over the pancreatic body ($P = 0.002$) and also over the pancreatic tail ($P = 0.036$). The global pancreatic T2* value did not show a significant difference amongst men and women and increased weakly with age in a significant manner. There was a significant negative correlation between serum ferritin levels and pancreatic iron overload ($r = -0.474$, $P < 0.0001$). Significant positive correlations of the global pancreas T2* value were demonstrated for global heart T2* value ($r = 0.355$, $P < 0.0001$) and number of heart segments with normal T2* ($r = 0.389$, $P < 0.0001$). Pancreatic T2* value was positively related with left and right ejection fractions ($r = 0.211$, $P = 0.028$ and $r = 0.33$, $P = 0.015$, respectively).

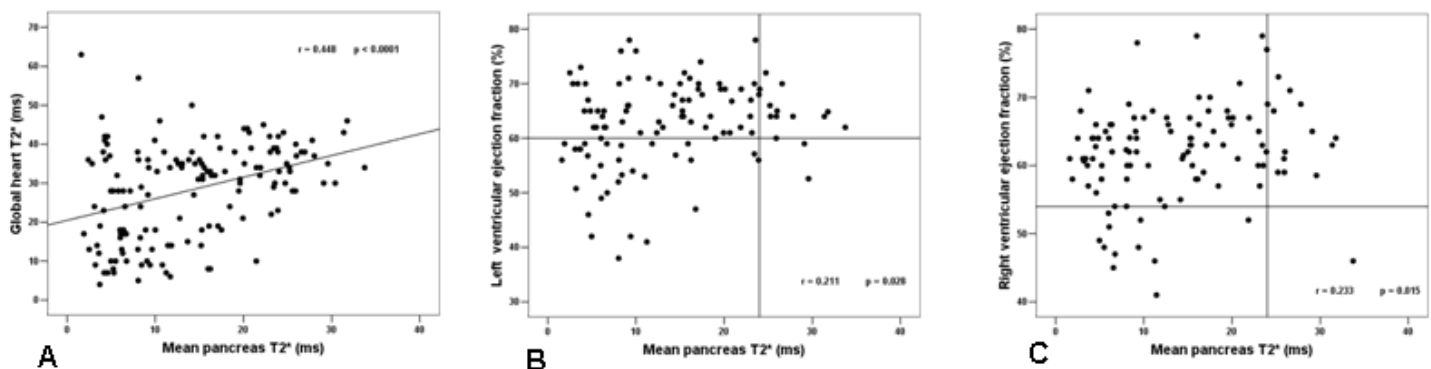


Figure 1: Scatter diagram of pancreatic iron overload compared to myocardial iron overload (A), left ventricular ejection fraction (B) and right ventricular ejection fraction (C).

Discussion and Conclusions: The pancreatic head derives from the ventral pancreatic anlage, while the other pancreatic regions originate from the dorsal anlage. These two parenchymal portions differ from each other in many histologic and functional aspects. So, we can hypothesize that the two parenchymal portions may load iron to a slight different extent (lesser in the head) and thus account for the slightly higher T2* in this region. The strong correlation between hemosiderosis of the pancreas and heart found in this study is consistent with previous reports considering the mid-septum T2* value [5]. Furthermore, for the first time we showed that this correlation is true also considering a myocardial segmental analysis. Moreover, pancreatic iron overload is negatively correlated to biventricular systolic function.

References: [1] Cogliandro T et al. J Cardiovasc Med 2008;9(5):515–525. [2] Meloni A et al. Int J Med Inform 2009;78(8):503-512. [3] Pepe A et al. JMRI 2006;23(5):662-668 [4] Cerqueira MD et al. Circulation 2002 ;105:539-542. [5] Au WY et al. Haematologica 2008;93(1):116-119.