

PANCREATIC IRON OVERLOAD IN THALASSEMIA PATIENTS: T₂* MRI EVALUATION – INITIAL EXPERIENCE

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Introduction: Thalassemia patients require multiple transfusions in order to avoid chronic anemia sequelae. This regimen entails intrinsic deleterious effects, the majority of which are related to iron deposition in the reticuloendothelial system. Thus, iron is deposited in hepatic, myocardial and endocrine gland tissues. Pancreatic siderosis can affect both endocrine and exocrine pancreatic functions. In addition, diabetes has been postulated to result from excess iron deposition in the pancreatic acinar beta cells besides increased peripheral insulin resistance.

T₂* MRI sequences have been previously addressed as a reliable tool for noninvasive evaluation of iron load in the liver and the heart. Reports in the literature discussing pancreatic evaluation by MRI, in Thalassemia patients, utilized T₂* weighted sequences, as an indication of iron deposition (1, 2).

Purpose: The purpose of this work is to assess the degree of iron overload in the pancreas of Thalassemia patients compared with normal subjects. T₂* MRI mapping is used to define a range of normal VS pathologic pancreatic T₂* values, in an attempt to reach a better understanding of the full spectrum of iron pathophysiology in multi transfused patients.

Methods: MRI measurements were performed using 1.5T, GE MRI system (Signa HDx Ver 14) using a dedicated 8 channel cardiac phased-array coil. Axial sections were acquired through mid abdomen to include the pancreas. Breath-hold multi echo gradient echo T₂* sequence with: TR 28.5 - 35 ms, FA 30°, 128X128 matrix, FOV 36 cm, SW 8-10 mm, 16 echos starting with TE 1.2 -1.6 ms, echo interval 1.6 ms. T₂* evaluation is based on Anderson et. al (3). T₂* values were sampled across regions of interest (ROI), located at areas of pancreatic tissue that was best visualized and as previously described for the liver and heart (3).

Results: Table 1 summarizes the MRI measurements. As has been previously described liver siderosis precedes cardiac iron overload.

Table 1: Pancreatic T₂* values of Thalassemic patients (Pt) and controls (Ctr).

Pt/Ctr	Pt 1	Pt 2	Pt 3	Pt 4	Pt 5	Pt 6	Ctr 1	Ctr 2
Gender/Age (y)	M/14	M/13	M/27	M/30	M/16	M/24	M/56	M/54
Pancreas T ₂ * (ms)	13±2	8.1±1.5	6±2	8.1±1.4	22±1	6±2	40±5	35±3
Liver T ₂ * (ms)	3.7±0.4	3.4±0.3	5.8±0.2	2.6±0.5	2.6±0.3	1.4±0.4	33±7 ⁽³⁾	
Heart T ₂ * (ms)	40±5	39±3	37±2	40±2	42±3	13±1	52±16 ⁽³⁾	

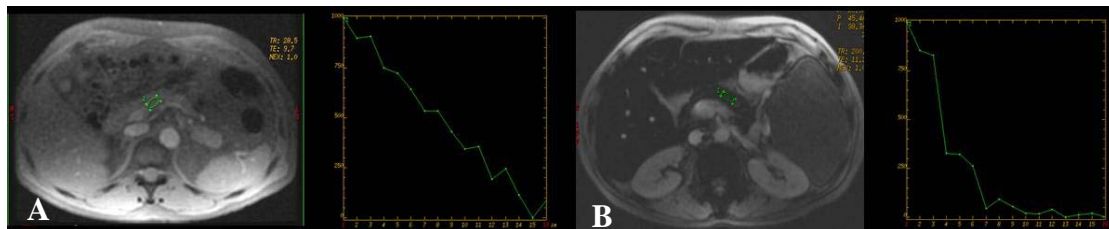


Fig 1: Anatomic images: ROI in the body of the pancreas, and the corresponding T₂* decay curves (A-Ctr 2, B-Pt 4). Note the markedly hypointense signal of the pancreas and liver in the Thalassemic pt.

Conclusions:

1. To our knowledge this is the first documentation of T₂* values for human pancreatic tissue.
2. In this small cohort, pancreatic T₂* values are significantly lower in all Thalassemic patients compared to normal subjects, suggesting pancreatic iron deposition.
3. No obvious correlation is found between pancreatic, liver and cardiac siderosis (2).
4. It should be noted that pancreatic fatty replacement occurring at the end and severe stage of pancreatic involvement, might affect the measured T₂* values.
5. Further studies in larger cohorts correlating pancreatic T₂* evaluation, with clinical course and response to chelation therapy, are planned.

References:

1. Midiri M et al, AJR 173:187,1999.
2. Papakonstantinou O et al, Eur. Radiol. 17:1535,2007.
3. Anderson et al, Eur Heart J 22:2171,2001.