Long-term Monitoring of Patients after Liver Transplantation: An Relaxometry Study

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Introduction

Liver cirrhosis is accompanied not only by serious metabolic problems, but also by effects on the mental abilities of the patients. Chronic encephalopathy as a complication of cirrhosis manifests itself on MRI by hyperintensities in the brain in T1 weighted images and hypointensities in T2 weighted images. The observed changes are caused by alterations in T1 and T2 relaxation times in the basal ganglia [1]. An increased manganese (Mn) concentration in the affected brain structures is probably responsible for the shortening of relaxation times. The concentration of Mn is known to be higher in both the liver and blood [2]. A previous study [3] has shown that chronic encephalopathy as a complication of cirrhosis can be stopped and even reversed by liver transplantation. Relaxation times in the basal ganglia return to normal values within 12 – 24 months after transplantation. The aim of this study was to check whether the improvement is permanent and whether MR - T2 relaxometry, reflecting the concentration of paramagnetic ions in the brain, correlates with other clinical data and the overall health of patients after liver transplantation.

Methods

A group of 42 patients who underwent liver transplantation and who were scanned before or during 3 years after transplantation was monitored long-term. Four patients died within 8 years after liver transplantation, one due to liver graft failure after the recurrence of HCV; three died due to other reasons with a functioning graft. Eighteen patients were scanned 5 – 8 years after transplantation. One patient was excluded from the study – in this case cirrhosis occurred due to resuming alcohol consumption. A control group of 20 healthy volunteers (divided to two subgroups according to their age to match them to the patients at the beginning and at the end of the study) was scanned using the same protocol.

We used a Siemens Magnetom Vision imager 1.5 T with a standard head coil. T2 relaxation time was obtained using a CPMG sequence with 16 echoes, echo-spacing 22.5 ms, recovery time TR = 2000 ms. A single axial slice through the anterior and posterior commissures was used. This slice included the head of the caudate nucleus (CN), globus pallidus (GP) and anterior putamen (Put). T2 values were evaluated in the whole slice, i.e., a T2 map was calculated on a pixel-by-pixel basis by 3-parameter fitting. Values of T2 were obtained from the basal ganglia, thalamus, and frontal white matter.

Results

T2 relaxation time was significantly shortened due to hepatic cirrhosis. Following liver transplantation the relaxation time recovered gradually, with near normal values reached after approx. 2 years (see Figure). This behavior could be observed in the GP, Put, CN, and Th. No recovery was observed in the frontal white matter. A moderate decrease in T2 in the basal ganglia was observed 5-8 years after transplantation. Similar decrease was found also in controls in the time span of the study.

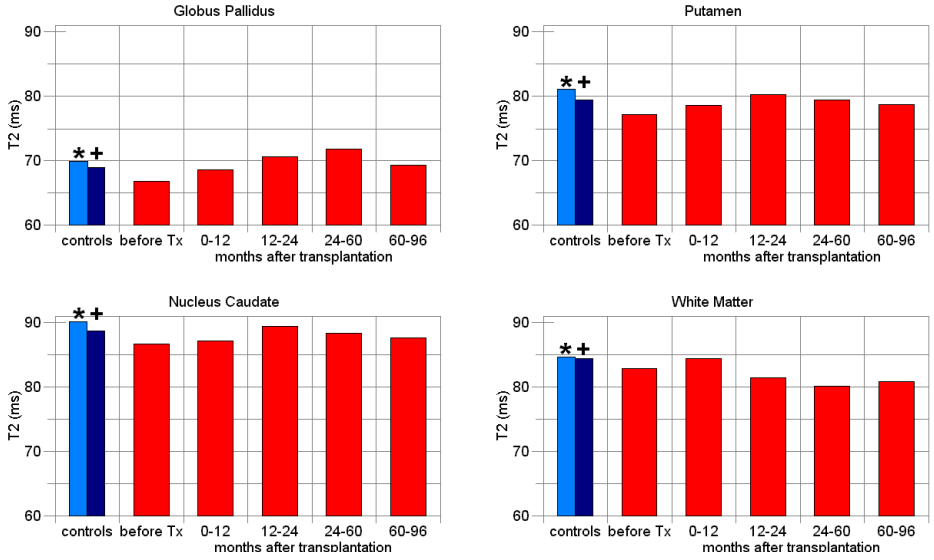

Figure: T2 relaxation times in basal ganglia and frontal white matter in patients before transplantation (Tx) and up to 96 months after. Healthy controls are matched to the age of patients at the beginning (*) and at the end of the study (+).

Discussion/Conclusion

We observed a strong drop in T2 relaxation time due to hepatic cirrhosis prior to transplantation, as previously described. Following liver transplantation, the T2 relaxation time slowly recovered and after approximately two years reached normal values. We hypothesize that deposited manganese, which is thought to be responsible for relaxation time alterations, might be shunted away from the basal ganglia after liver transplantation. The relaxation time in the basal ganglia moderately decreased after 5-8 years. Comparison to similar decrease of T2 in controls shows that for this decrease an age dependence of T2 (caused by deposition of Fe3+ particularly in the basal ganglia [4]) might be responsible. Thus we can conlude, that there is no abnormal deposition of Mn or other paramagnetic ions in BG observable within 8 years after liver transplantation. We observed a decrease of T2 in white matter after transplantation with no recovery. As the patients are dependent on immunosuppresants after transplantation, which are potentially neurotoxic and could particularly influence white matter, we hypothesize that T2 relaxation could be affected by the immunosuppression.

References

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