Assessment of hepatic lipid content by MRS in patients on home parenteral nutrition

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Introduction: Patients with severe chronic intestinal failure depend on parenteral nutrition, which implies that all macro- and micronutrient requirements are administered into a large-bore central vein [1]. Often these patient receive this nutrition at home, which is called home parenteral nutrition (HPN). Patients receiving HPN are at risk for developing hepatic dysfunctioning due to steatosis. In addition, their hepatic iron and manganese content may increase. Previously the condition of the liver could only be assessed by taking a biopsy, but presently non-invasive techniques like MRS can be used [2]. The **aim of the present study** was to determine hepatic lipid content in patients receiving HPN with taking into account the possibility of altered relaxation behavior as result of increased manganese or iron content.

Subjects and Methods: Liver fat content was studied in 13 patients (5 male, 8 female) with a mean age of 46 yrs (range 20-64 yrs), who have been on HPN (mean 5.7 times per week) for 8.2 yrs (range 2 -36 yrs). MR measurements were performed on a clinical 3T whole body MR system (Siemens Magnetom Tim Trio) using the bodycoil for excitation and two phased-array surface coils positioned at the liver and the abdomen for signal reception. After the acquisition of localizers in three orthogonal directions during breath holding, single voxel proton MR spectra were acquired from a volume of $30x30x30mm^3$ positioned in the center of a liver lobe avoiding large vessel structures. A STEAM localization sequence without water suppression was used for data acquisition. To allow for correction for relaxation effects on signal intensity both long repetition time (TR = 3s) and four different echo times (TE = 20, 30, 40 and 50 msec) were used. Six averages were obtained during breath holding for 15 seconds. For each TE, the MRS measurement was performed in duplo.

Post-processing consisted of time-domain fitting of the water signal resonating at 4.7 ppm and the methylene lipid peak at 1.3 ppm (Fig. 1) using the AMARES routine in jMRUI [3]. To correct for T2 relaxation the ratio (lipid intensity (L)/water intensity (W)) was plotted as function of echo time (Fig. 2) and an exponential function $\{L/W=(L/W)_0 \exp(TE*K) \text{ with } K=(1/T2_W-1/T2_L)\}$ was fitted to the data to obtain the value for TE = 0 msec. From this ratio the hepatic lipid content was calculated by the formula $(100 \times (L/W)_0)/(1 + (L/W)_0)$, which equals $100 \times (\text{lipid} - \text{CH}_2- \text{intensity}) / (H_2O \text{intensity} + \text{lipid} - \text{CH}_2- \text{intensity})$ at TE=0 msec.

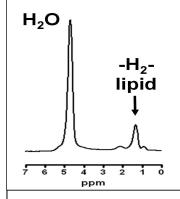


Fig. 1 MR spectrum of the liver (TE = 20 ms) of patient with 8.5% fat content.

Results: Proton MR spectra of the liver of good quality were obtained (example in Fig.1). Five patients had a hepatic lipid content above the upper limit of 5% for healthy volunteers [4] with an average value of $12.8\pm3.5\%$ (mean \pm SD), while the other eight had normal liver fat concentration (1.1 \pm 0.6%) (Fig. 2). Figure 3 shows for two patients the intensity ratio L/W as a function of echo time. K-values were obtained in the range between 0.0148 and 0.0330 msec⁻¹ (0.0216 \pm 0.0056 msec⁻¹). The lowest value agrees very well with a reference K-value of 0.0147 msec⁻¹ calculated from literature values for T2_W and T2_L of 34 and 68 msec, respectively [5]. The rest of the patients showed higher K-values, caused by increased T2 relaxation behavior probably due to elevated hepatic iron or manganese content. The two highest K-values of 0.0330 and 0.0305 msec⁻¹ were obtained for a patient with sclerosing peritonitis receiving HPN for 5 yrs and a patient with short bowel due to mesenteric thrombosis receiving HPN for already 36 yrs, respectively.

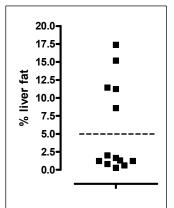


Fig. 2 Liver fat content in patients on HPN. The dashed line indicates the upper level of normal hepatic lipid content.

Discussion: As no multi-echo MRS sequence was available, the MR spectra at various echo times were collected during different breathholds and the overall intensities of the spectra showed some variation hampering determination of individual T2 values of the water and lipid signals. Therefore, the ratio L/W as function of TE was used to determine (L/W)₀ to calcultate % hepatic lipid content, as described above. The advantage of this procedure was that spectra obtained during different periods of breath-holding could be combined. However, the disadvantage was that only K-values and no absolute T2-values for hepatic water and lipid signals could be obtained. Therefore, future experiments will be performed with a multi-echo MRS sequence using shortened repetition time to allow collection of data points of a complete T2 relaxation decay within one breath-hold, both to improve patient comfort and to allow measurements of absolute T2-values of all MR signals present in the spectrum.

Conclusion: The present study shows that elevated hepatic lipid content of patients receiving HPN can be non-invasively determined by MRS. In particular in these patient group, possible increased T2 relaxation behavior has to be taken into account to obtain reliable hepatic lipid content.

References: [1] Wanten GJ and Calder PC, Am J Clin Nutr. 2007;85:1171-1184; [2] Woodward JM, et al., J Parenter Enteral Nutr. 2009;33:669-676; [3] http://sermn02.uab.es/mrui/; [4] Szczepaniak LS, et al., Am J Physiol Endocrinol Metab. 2005;288:E462-468; [5] de Bazelaire CM, et al., Radiology 2004; 230: 652–659.

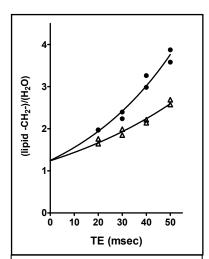


Fig. 3 Lipid/water intensities as a function of TE with an exponential fit. Data of two patient with similar lipid content but different relaxation behavior. Δ K=0.0148 msec⁻¹, and \bullet K = 0.0221 msec⁻¹.