

Spectroscopic Metabolic Abnormalities in mTLE with and without MRI Evidence for Mesial Temporal Sclerosis Using Hippocampal Short TE MRSI

UCSF



S.G. Mueller¹, K.D. Laxer², R.C. Lopez¹, M.W. Weiner¹

University of California, San Francisco, Dept. of Radiology, MR-spectroscopy VAMC, San Francisco, CA USA¹; University of California, San Francisco, Dept. of Neurology, San Francisco, CA USA²

Introduction

In mTLE with MRI evidence of mesial temporal sclerosis (TLE-MTS) spectroscopy using long echo times (TE) (135-272 ms) reliably identifies the epileptogenic hippocampus by a reduced N-acetyl-aspartate (NAA) compared to the non-epileptogenic hippocampus or to healthy controls. Short TE (25 ms-36 ms) detects Myo-inositol (MI) in addition to NAA, Cr and Cho. However, short TE measurements are prone to artefacts due to contribution of macromolecules/lipids and magnetic field inhomogeneities. Therefore, the aims of this study were to evaluate 1. Whether short TE MRSI could lateralize MTLE in a fashion similar to long TE MRSI. 2. The value of MI for identification of the epileptogenic hippocampus in mTLE with and without evidence for MTS.

Patients and Methods

We studied 24 patients with mTLE (age 36.9 ± 9.3 years) and 14 controls (age 29.9 ± 6.1 years). 16 patients had evidence for MTS on MRI (TLE-MTS: right mTLE: 8; left mTLE: 8) and 8 had normal MRI (TLE-no: right mTLE: 3; left mTLE: 5).

All studies were performed on a 1.5 T Magnetom Vision™ MR system (Siemens Inc., Iselin, NJ). The following images were acquired: 1. T1-weighted FLASH with TR/TE = 500/14 ms, parallel to the long axis of the hippocampus. 2. Double spin echo TR/TE1/TE2 = 2500/20/80 ms. 3. Volumetric magnetization-prepared rapid gradient echo (MPRAGE), TR/TE/TI = 13.5/7/300 ms, 1.0x1.0 mm² in-plane resolution, slice thickness 1.4 mm. DSE and MPRAGE were used for segmentation. The hippocampus was manually marked on the MPRAGE using a commercially available brain mapping tool (Surgical Navigator Technology Inc., Boulder CO) and this information implemented into the segmented image. Spectroscopic measurements were done with a 2D MRSI sequence (TR/TE = 1800/25 ms) using PRESS volume pre-selection (15 mm axial, 60 mm left-right, 100 anterior-posterior) with 24x24 phase encoding steps and a 210 mm² FOV angulated along the long axis of the hippocampus, covering both hippocampi. The gray/white matter contributions to each voxel were computed by convolving each tissue map of the segmented images with the discrete transform of the MRSI spatial response function. Metabolite concentrations were corrected for CSF contribution and expressed in arbitrary units. Nominal voxels of good spectral quality containing at least 15% hippocampal tissue contribution were selected from each hippocampus (2-8 of each side). Statistical analysis was done with two-tailed t-tests.

Results

In TLE-MTS, but not TLE-no, NAA was decreased in the ipsilateral hippocampus compared to contralateral ($p=0.03$) and to controls ($p=0.0001$). In TLE-MTS NAA was also decreased in the contralateral hippocampus compared to controls ($p=0.01$). Cr, Cho and MI were not different between the ipsilateral and contralateral hippocampus of TLE-MTS or TLE-no or between patients and controls (**Table 1**). 11/16 TLE-MTS and 3/8 TLE-no were correctly lateralized by a lower NAA on one side. In controls, NAA ($p=0.000002$), Cr ($p=0.001$) and Cho ($p=0.0009$) but not MI concentrations were lower in voxels from the anterior half of the hippocampus compared to voxels from the posterior half. In TLE-MTS, NAA ($p=0.0003$), Cr ($p=0.0006$), Cho ($p=0.001$) were lower in voxels from the anterior part of the hippocampus compared to the posterior ipsilaterally but not contralaterally. The small number of voxels with good quality spectra from the anterior part of the hippocampus of TLE-no did not allow a statistical analysis in this group.

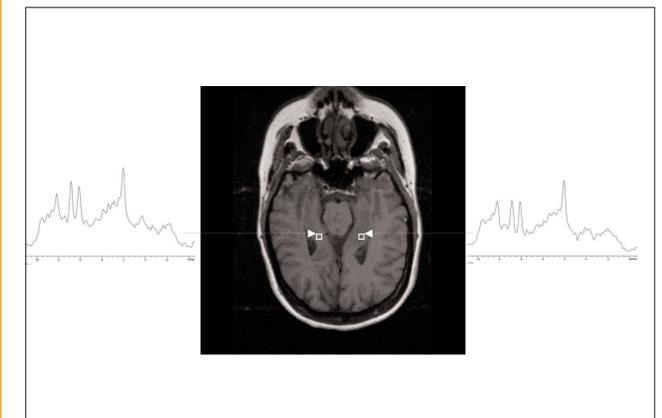
Table 1: Hippocampal Metabolite Concentrations in Controls, TLE-MTS and TLE-no

Group	Side	NAA	Cr	Cho	MI
TLE-MTS	I	2.5±0.3*	2.1±0.3	0.6±0.06	1.9±0.3
	C	2.7±0.3*	2.1±0.3	0.6±0.05	2.0±0.4
TLE-no	I	2.7±0.4	2.3±0.3	0.5±0.09	2.0±0.3
	C	2.6±0.4	2.2±0.4	0.6±0.1	2.0±0.3
Control	L	3.0±0.6	2.2±0.3	0.6±0.14	2.1±0.2
	R	3.0±0.4	2.2±0.4	0.6±0.1	2.0±0.2

I, ipsilateral; C, contralateral; L, left; R, right, TLE-MTS, temporal lobe epilepsy with mesial temporal sclerosis; TLE-no, temporal lobe epilepsy without mesial temporal sclerosis, NAA, N-acetyl aspartate; Cr, creatine and phosphocreatine; Cho, choline compounds. Concentrations are given in arbitrary units.

* $p<0.05$ compared to contralateral
° $p<0.05$ compared to controls

Figure 1



Hippocampal spectra of a TLE-MTS patient with seizures starting in the right hippocampus.

Conclusion

In TLE-MTS, hippocampal short TE MRSI replicated the results of previous long TE MRSI studies, i.e. reduction of NAA but not Cr or Cho in the ipsilateral hippocampus compared to contralateral or controls and an anterior-posterior metabolite gradient (1). MI in TLE-MTS or TLE-no was not different from controls and did not provide any additional information for identification of the diseased hippocampus. In agreement with previous short TE studies done in other laboratories (2) TLE-no were not different from controls concerning NAA, Cr, Cho and MI concentrations. The results for the TLE-no group contrast with a previous long TE study done in this laboratory (3) where spectroscopy allowed to identify two subgroups and predicted surgical outcome. The fact that in the short TE study about 50% of all voxels - most of them in the anterior hippocampus - were lost due to poor spectral quality could explain this difference. **Therefore we conclude that hippocampal short TE MRSI has no advantage over hippocampal long TE MRSI in mTLE and is more affected by artefacts leading to the loss of information from the most diseased region of the hippocampus in mTLE.**

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References:

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