Characterization of gliomas using MRI and short echo 1-H MRSI at 7 Tesla

Yan Li[1], Peder Larson[1], Albert Chen[2], Douglas Kelley[1,3], Susan Chang[4], and Sarah J Nelson[1,5]

[1]*Department of Radiology and Biomedical Imaging, University of California, San Francisco, California, United States,* [2]*GE Healthcare, Toronto, Ontario, Canada,* [3]*GE Healthcare, San Francisco, California, United States,* [4]*Department of Neurosurgery, University of California, San Francisco, California, United States,* [5]*Department of Bioengineering and Therapeutic sciences, University of California, San Francisco, California, United States*

Introduction

Patients with glioma have a variable prognosis, ranging from a median survival of 15 months for the most malignant lesions to decades in the case of low grade oligodendrogliomas. The standard criteria for assessing tumor progression and response to therapy are based upon changes in cross sectional diameter of the Gadolinium enhancing lesion. For cases where there is no enhancement the changes in the T2 lesion provide the only means of assessing the tumor, but this may also include regions of edema or reactive gliosis. Ex vivo MRS data have suggested mI/Cho can be used to differentiate between tumor and gliosis. Ultra high field (7 Tesla) MR systems offer advantages in higher SNR and improved spectral resolution; but have not yet been widely used in the clinical setting. The purpose of this study was to evaluate the metabolic signature of lesions in patients who were undergoing treatment for glioma using our 7 Tesla whole body scanner.

Methods

Five patients with gliomas (ID, A-E) and three healthy volunteers (ID, 1-3) were studied. Patient characteristics and treatments are summarized in Table 1. All MR studies were performed using a 32-channel receive-only array with a volume transmit head coil on a GE 7 Tesla scanner (GE Healthcare, Waukesha, WI). Anatomical imaging consisted of a T1-weighted sagittal scout, T2*-weighted gradient recalled echo (GRE), T2-weighted fast spin echo (FSE) and sagittal CUBE fluid attenuated inversion recovery (FLAIR) images. The 2D or 3D H-1 MRSI was localized using CHESS water suppression, 8 VSS outer volume suppression, including 2 fixed VSS around the prescribed slab in the S/I direction and 6 graphic prescribed VSS pulses, spin echo slice selection [1] with TE/TR=30/2000ms, spectra array=20x22 or 20x22x8. To further eliminate signal from subcutaneous fat, a spectrally-selective adiabatic inversion recovery pulse was applied prior to the CHESS pulses (TI=516ms) with an inversion bandwidth of 400Hz (Figure 1). An interleaved flyback echo-planar trajectory with a spatial resolution of 1 cm were designed for 7 Tesla MRSI and applied in A/P direction with a FOV of 22 cm to shorten the total acquisition time. Each acquisition comprised 552 readout lobes with a spectral-bandwidth of 958 Hz after reconstruction using the flat portion of the flyback lobes. The second acquisition was temporally shifted by 1/(2xspectral-bandwidth). Thus, the combined data would have a bandwidth of 1916 Hz. The 32 channels of data were combined and processed as described previously [2], and quantified using LCModel. Metabolite signals for the basis set, which included Cho, Cr, NAA, Glu, Gln, mI, Gly, GABA, GSH, PE, Tau and Asp, were generated using GAMMA simulations with prior knowledge of chemical shift and J-coupling. For the cases B, C and D, where there were a large number of voxels in the T2 lesion, ranksum tests were utilized to compare metabolite ratios in the T2 lesions for individual patients with values in the white matter of the volunteers.

Results

Figure 2 illustrates MRI and H-1 MRSI data acquired from patient A. The T2 lesion from the FSE image that was obtained at 7 Tesla was close to that at 3 Tesla. Noted that the baseline was not removed in the spectra and the water signal is smaller than NAA from the selected grey matter (blue) and white matter (pink) voxels. Metabolite ratios are summarized in Table 2, and boxplots of Cho/NAA and mI/Cho in Figure 3. Metabolite ratios in the T2 lesion for patients A and E were similar to those in the white matter for the volunteers. Patient B had significantly lower Cho/Cr (p, 0.0007), NAA/Cr (0.0005), Cho/NAA (0.0243) and mI/Cr (0.0236); Patient D had significantly higher Cho/NAA (0.001), lower NAA/Cr (0.0032), higher mI/Cr (<0.0001) and mI/Cho (<0.0001); while patient C had significantly higher Cho/Cr (<0.0001), lower NAA/Cr (<0.0001), higher Cho/NAA (<0.0001) and higher mI/Cr (0.007) (Figure 4).

Discussion

This study has successfully demonstrated the application of MRI and H-1 MRSI to volunteers and patients with glioma. Lower Cho/Cr, NAA/Cr, Cho/Cr and mI/Cr in patient B could be due to edema, higher Cho/Cr and lower NAA/Cr in patient C to active tumor, while the lower NAA/Cr and higher mI/Cho may indicate treatment effects. It is anticipated that using the combination of metabolite ratios would be helpful in interpretation of response to therapy. We are continuing to follow these subjects and will apply similar methods to a larger cohort of patients with glioma in order to further evaluate the role of 7 Tesla in assessing response to therapy.

References 1. Li, Y., et al. ISMRM 2010; 2. Nelson, SJ. Magn Reson Med, 2001;46(2):228-39.

Acknowledgements Funding from the University of CA and GE Healthcare, as well as a St. Louis Fall Festival Committee American Brain Tumor Association Basic Research Fellowship.

ID	Cho/Cr	NAA/Cr	Glu/Cr	mI/Cr
A	0.27±0.06 (4)	1.54±0.47 (4)	1.41±0.73 (2)	0.66+0.00 (2)
B	**0.22±0.13 (24)*****	**1.31±0.27 (24)*****	1.19±1.11 (17)	**0.61±0.24 (20)***
C	**0.52±0.21 (30)*****	**0.7±0.32 (30)*****	1.25±1.00 (17)	**1.10±0.56 (22)*****
D	0.26±0.05 (13)	**1.23±0.30 (13)****	0.89±0.44 (8)	**1.42±0.41 (12)*****
E	0.26±0.07 (4)	1.35±0.30 (4)	1.91±1.20 (3)	0.70±0.12 (3)
1	0.22±0.03 (20)	1.49±0.16 (20)	0.96±0.33 (14)	0.55±0.07 (19)
2	0.28±0.04 (36)	1.51±0.21 (36)	1.25±0.39 (32)	0.78±0.15 (36)
3	0.22±0.05 (13)	1.48±0.24 (13)	1.39±0.12 (3)	0.64±0.17 (11)

Table 2. Metabolite ratios (mean±std, N) *p<0.05; **p<0.001; ***p<0.001

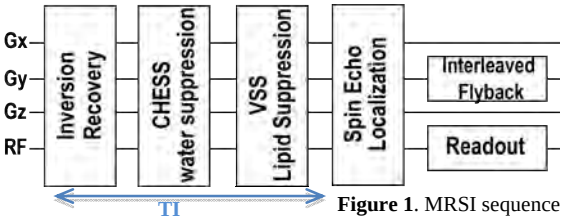

Figure 1. MRSI sequence

ID	Age	Sex	Type	Treatment	Last Visit
A	32	F	AA	Sx+RT+Avastin+TMZ	Stable
B	61	M	OG2	Sx+RT+PCV	Suspected
C	39	M	OG2	Sx+RT	Suspected
D	65	F	OG2	Sx	Stable
E	37	M	A2	Sx	Stable

Table 1. Characteristics and treatments of patients

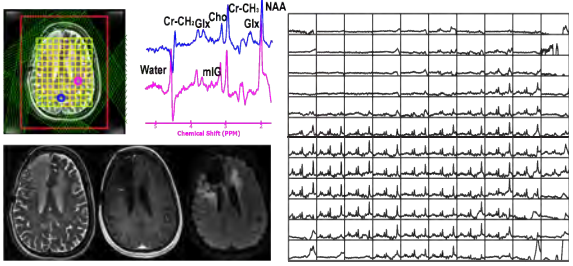

Figure 2. MRSI data (with baseline) corresponding to the location from patient A and a panel of MRI images composing of FSE at 7T, T1-weighted post contrast and FLAIR at 3T.

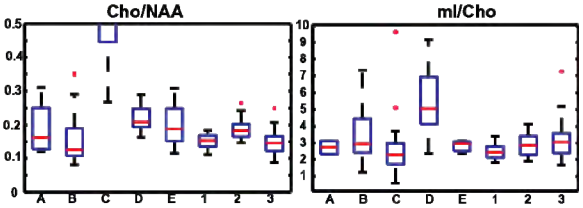

Figure 3. Boxplots of Cho/NAA and mI/Cho

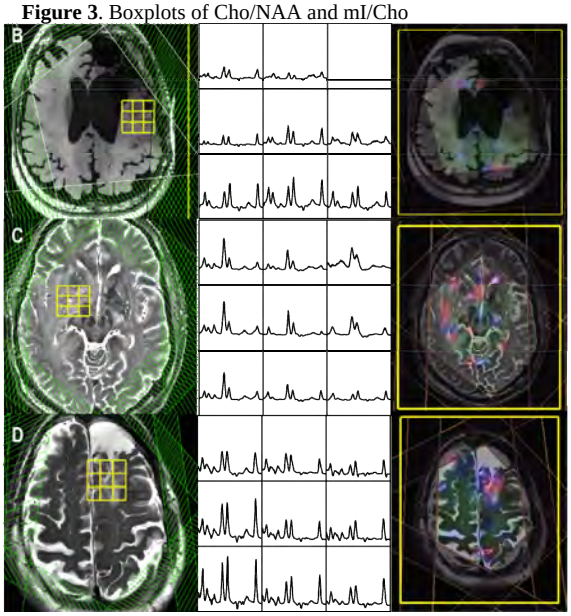

Figure 4. MRSI from patient B, C and D along with mI/Cr maps (0-1.6) quantified using LCModel