Differentiation of acute and subacute demyelinating lesions with short echo time Chemical Shift Imaging

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Introduction:

Acute encephalomyelitis is an immune-mediated, inflammatory demyelinating disease which can be monophasic (i.e. ADEM) or multiphasic (i.e. MS, MDEM). Until now identification of acute demyelinating lesions was limited to contrast-enhanced MR images. Proton MR spectroscopy with metabolite-nulling has shown elevated lipids/macromolecules in acute demyelinating lesions [1]. We present a patient in whom ¹H-MRSI helped to differentiate areas of active and subacute demyelination.

Methods:

A 20-year old man who had well recovered under corticosteroids from an episode of acute encephalomyelitis with aphasia and right-sided hemiparesis one year ago presented with acute recurrence of the right-sided hemiparesis. MRI was performed on a 1.5 T MR scanner (MAGNETOM Avanto, Siemens Medical Solutions, Erlangen, Germany). Images revealed a single large mass-like demyelinating lesion in the left-sided supratentorial white matter. Standard MR images (Flair, T1, T2, T1 with Gd-DTPA) could not clearly differentiate between the active and subacute demyelinating parts of the lesion (Fig. 1a).

For spectroscopic investigation we applied a Chemical Shift Imaging (CSI) sequence with PRESS localization of the Volume of Interest (VOI). 512 data points were acquired with a bandwidth of 1000 Hz. The matrix size of 16 x16 was acquired by sampling elliptically and weighted with 4 averages in the center of k-space. With TR = 1.5 s the acquisition time was 7 min for each CSI experiment. The nominal resolution was 10 x 10 x 15 mm³. The body coil was used for excitation and a 12 channel head coil for signal reception. The signals of the individual coil elements were weighted for the signal to noise of each element and coherently combined. Additionally, the head array coil profile was homogenized realizing the sensitivity profile of the body coil. CSI measurements were performed with long (135 ms) and short (30 ms) TE in a transversal plane containing the lesion.

Results:

CSI measurements revealed strong differences between the rostral and the dorsal part of the lesion. With TE of 135 ms NAA was strongly diminished in the rostral and slightly diminished in the dorsal part (Fig. 1b). No lactate signal was detected. In the rostral part of the lesion, CSI maps with TE of 30 ms showed strongly increased myo-Inositol corresponding to a high glial content (Fig. 1c) and only slightly elevated lipids/macromolecules at 1.3 ppm. The dorsal part of the lesion, however, presented a very high lipid/macromolecule signal at 1.3 ppm (Fig. 1d) and a slightly diminished myo-Inositol resembling active demyelination. The corresponding short echo time CSI spectra (Fig. 2) were of good quality, so that all metabolite images could be rendered automatically.

Conclusion:

Short TE ¹H-CSI spectra have reached high quality in *vivo*. CSI including maps for myo-Inositol and lipids/macromolecules can be helpful to differentiate acute and subacute demyelinating lesions in cases without contrast enhancement on T1-weighted images.

References:

[1] Mader I, Seeger U, Weissert R, Klose U, Naegele T, Melms A, Grodd W.

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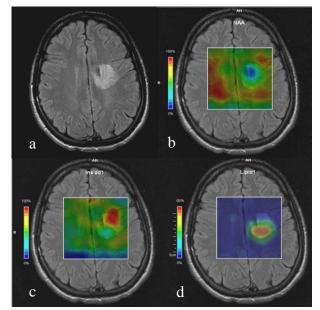


Fig. 1 a) TSE-FLAIR (TI = 2.5 s): the acute and subacute lesion is enhanced; b) interpolated NAA metabolite image calculated from the spectra acquired with TE = 135 ms; c) myo-Inositol and d) Lipid/macromolecule metabolite image calculated from the spectra acquired with TE = 30 ms. The white box illustrates the excited VOI of 8 x 8 cm².

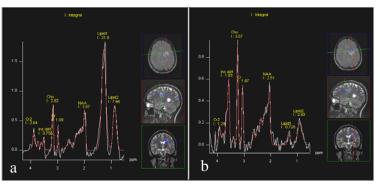


Fig. 2 Short TE spectra from the the rostral and the dorsal part of the lesion: a) rostral: very high lipid/macromolecule signal and slightly decreased myo-Inositol signal

b) dorsal: strongly increased myo-Inositol