Proton MR Spectroscopic Changes in Primary Motor Cortex and Supplementary Motor Area of Hemiparetic Patients

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Abstract

To determine the primary motor cortex (M1) and supplementary motor area (SMA) dysfunction on affected and unaffected hemisphere, authors performed proton magnetic resonance spectroscopy (^{1}H MRS) for the evaluation of biochemical changes in the motor cortex in hemiparesis according to axonal injury at the level of internal capsule. We found that the mean N-acetylaspartate (NAA)/ phosphocreatine (Cr) and NAA/ choline (Cho) ratios were significantly decreased in M1 on affected hemisphere of hemiparesis patients. ^{1}H MRS examinations of the motor cortex might help to differentiate distinct clinical entities of hemiparesis and to monitor pharmacological effects in therapeutic trials, providing a quantitative biological marker for motor neuron dysfunction.

Introduction

Noninvasive ^{1}H MRS offers the opportunity to investigate regional changes in the metabolite composition of different brain regions in vivo. It is feasible to define spectral changes specific to the motor cortex. For example, significant differences in NAA/ Cr and NAA/Cho in the motor cortex between controls and patients with motor neuron disease/amyotrophic lateral sclerosis (ALS) have been reported (1, 2). To our knowledge there have been no previous cerebral ^{1}H MRS studies of motor cortex in hemiparesis in humans. This was the first study to use ^{1}H MRS for the evaluation of biochemical changes in M1 and SMA on affected and unaffected hemisphere according to their axonal injury at the level of internal capsule. We analyzed the relative resonance intensities of NAA, Cho, Cr, myo-inositol (Ins), and glutamine/glutamate in the motor cortex regions of patients with hemiparesis to detect the brain metabolite ratios that best reflect the neuronal dysfunction of the motor cortex, and to prove that ^{1}H MRS could provide additional information not available from conventional MRI.

Materials and Methods

The M1 and SMA of affected hemisphere and unaffected hemisphere were studied by using ^{1}H MRS in 9 patients (4 men and 5 women; mean age 40 years, range 21-77 years) with documentable hemiparesis of varying severity. To exclude possible ^{1}H MRS spectral change, the patients who have undergone any surgical intervention or have major systemic illness such as uremia were excluded. The results were compared with ^{1}H MRS studies performed in 12 normal control volunteers (six men and six women; mean age 28 years, range 21-49 years). In vivo ^{1}H MRS study was performed on a 1.5 T MRI system (GE Signa Advantage, version 4.8) using STEAM sequence after water suppression with CHESS RF pulse and dephasing gradients. As a single-voxel technique, ^{1}H MR spectra were obtained from alert patients with definite hemiparesis in extremities contralateral to the affected hemisphere. Spectral parameters were: 20 ms TE, 2000 ms TR, 128 averages, 2500 Hz spectral width, and 2048 data points. Raw data were processed by the SAGE data analysis package (GE Medical Systems). Peak areas of NAA, Cr, Cho, Ins, and sum (Glx) of γ-Amino butyrate (GABA) and glutamate were calculated by means of fitting the spectrum to a summation of Lorentzian curves using a Marquardt algorithm. After blindly processed, we calculated the metabolite ratios of NAA/Cho, NAA/Cr, Cho/Cr, Ins/Cr, and Glx/Cr. Ratios are given as the mean ± SD. Statistical significance was determined using Student's paired t-tests between control subjects and patients with hemiparesis.

Results

Table 1 shows metabolite ratios from the M1 cortex of affected hemisphere and unaffected hemisphere according to axonal injury at the level of internal capsule. We found that the mean NAA/Cr and NAA/Cho ratios were significantly decreased in the M1 of affected hemisphere compared with their unaffected motor cortex ($p < 0.05$ and $p < 0.05$, respectively). Moreover, the NAA/Cr and NAA/Cho ratios were lower in M1 of the hemiparesis than in M1 of control subjects ($p < 0.05$ and $p < 0.05$, respectively). No differences between patients and controls were seen for any of the other metabolite peaks.

Discussion

The spectra from the M1of affected hemisphere revealed significantly lower NAA/Cr and NAA/Cho ratios compared with unaffected hemisphere in patients with hemiparesis. The decrease in the NAA/Cr and NAA/Cho ratios might be considered by reduced NAA concentrations. The decreases in the relative NAA concentrations are commonly observed in pathological processes well known to involve neuronal loss such as degenerative disorders, strokes, and glial tumors (3). Low NAA signals are also observed in other brain pathological processes in which the loss or damage to neurons and axons is less well known and less evident, even at mostmortem examination (4). Since NAA is exclusively expressed in neurons, therefore, we suggest that the reduction of NAA/Cr and NAA/Cho ratios may indicate a neuronal loss or dysfunction of the cortical motor neurons. We think that ^{1}H MRS could be used either to evaluate the different pathologic features of patients with hemiparesis, especially during disease onset, or to detect interindividual differences in the progression of the disease. Further serial studies focusing on the correlation between ^{1}H MRS findings and clinical characteristics of hemiparesis are warranted.

Table 1. Relative metabolite ratios obtained from the primary motor cortex of hemiparetic patients due to deep intracerebral hematoma.

Metabolite Ratio	Controls (N = 12)	Affected (N = 9)	Unaffected (N = 9)	*p* Value
NAA/Cr	1.34 ± 0.12	1.03 ± 0.14	1.43 ± 0.18	0.04†
Cho/Cr	0.86 ± 0.16	0.89 ± 0.12	0.88 ± 0.15	0.75
NAA/Cho	1.61 ± 0.29	1.21 ± 0.29	1.63 ± 0.33	0.01†

†Statistical significance determined by using the paired-students t-test, where $p<0.05$ was considered significant.

Reference

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