Proton Magnetic Resonance Spectroscopy (1H-MRS) Findings in Patients with Hepatic encephalopathy with and without Parkinsonian Signs

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Introduction

Hepatic encephalopathy (HE) is a reversible neuropsychiatric disorder common in patients with cirrhosis. In some HE patients, parkinsonian signs suggestive of extrapyramidal impairment points toward basal ganglia (BG) alterations, clinical manifestations of which consist of asterixis, tremor, rigidity and bradykinesia[1]. Conventional MR imaging showed signal hyperintensities in the globus pallidus on T1WI[2], but there were no obvious differences in signal intensities between the patients with and without parkinsonism. This work quantatively evaluates the metabolic changes in both patients with and without parkinsonism by using magnetic resonance spectroscopy (MRS) [3].

Methods

Twenty-eight patients (22 males, 6 females, the age ranged from 29 to 62 years, mean age 43 years) with definite liver cirrhosis were studied. 15 patients were classified as having parkinsonian signs evaluated by Unified Parkinson's Disease Rating Scale (UPDRS) test. The patients had no history of cerebrovascular disease, hypertension, diabetes mellitus, or chronic pulmonary disease.15 patients had obvious portosystemic shunting. 18 age-matched healthy volunteers (13 males, 5 females, the age ranged from 24 to 51 years, mean age 38 years) underwent MRS for comparison.

A superoconductive 1.5T MR scanner (Twin Speed, GEMS, Milwaukee) was employed and the head coil was used. Single voxel point-resolved (PRESS) sequence was performed in the basal ganglia (BG) with following parameters: TR=1500ms, TE=35ms, total number of scans=64, voxel=2.5cm×2.5cm×2cm. The MRS data were post-processed with Spectroscopic Analysis GE (SAGE) software. The following resonances were studied: myoinositol (mI), choline-containing compounds (Cho), N-acetylaspartate (NAA), and creatine (Cr). Results were expressed as ratios with Cr as an internal reference. UPDRS tests were evaluated by two neurologists blinded to diagnosis. The statistical analysis was performed using ANOVA test.

Results

The values of NAA/Cr, Cho/Cr, mI/Cr were showed in Table 1. NAA/Cr levels in patients with and without parkinsonism were close to the value observed in the control group (P>0.05). The values of mI /Cr and Cho/Cr showed no differences between the control group (Fig.1) and the patients without parkinsonism (Fig.2) (P>0.05). The mI /Cr and Cho/Cr are significantly different in patients without and with parkinsonism (Fig.3) (P<0.001). The mI /Cr and Cho/Cr ratios measured in the BG of patients correlated significantly to the UPDRS used for the assessment of parkinsonian signs.

Table 1: Metabolite ratios in Basal ganglia in ^{1}H MR spectroscopy in healthy volunteers, patients with and without Parkinsonism

Metabolite ratios	NAA/Cr	Cho/Cr	mI/Cr
Control group□n=18□	1.44±0.01	0.80±0.02	0.61±0.02
Patients without Prkinsonism□n=13□	1.40±0.03	0.82±0.03	0.63±0.01
Patient with Parkinsonism□n=15□	1.35±0.03	0.68±0.01#	0.39±0.02#

P values are given in brackets for the comparison. #sig nificant difference

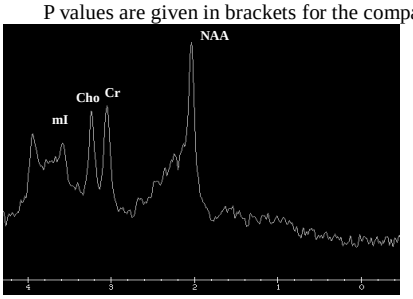

Fig.1. MRS of normal volunteer in BG

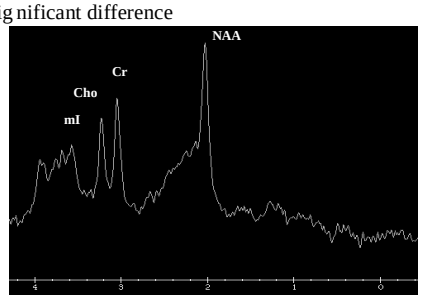

Fig. 2. MRS of the patient without Parkinsonism, no apparent change of metabolites

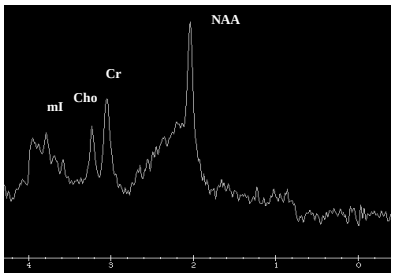

Fig. 3. MRS of the patient with Parkinsonism, marked decrease of the myoinositol and choline peaks.

Discussion

In our study, decrease of the mI /Cr and Cho/Cr ratios in the BG of HE patients with parkinsonism was found. This result is consistent with the previous report[2]. But a new finding is that the decrease of the mI/Cr and Cho/Cr ratios is directly correlated with the parkinsonian signs, meanwhile, there is no decrease of those ratios in patients without parkinsonism. The reason for these changes is that excessive manganese (Mn) is primarily cleared by the liver, while the portosystemic collaterals and impaired liver function may result in Mn deposition and damage to the pallidum selectively. Choline may be associated with altered cellular membranes and mI has an osmoregulative function in the glial cells. Thus, it is suggested that the metabolic alterations in the cell may be responsible for astrocyte swelling and consecutive altered brain function. In this study, positive correlations between brain MRS abnormalities and parkinsonian signs are observed in patients with cirrhosis. MRS reflects the BG metabolic alterations directly and quantatively in HE patients.

References

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