Preliminary Study of the Brain in Patients with Amyotrophic Lateral Sclerosis by Using Susceptibility-weighted Imaging

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Hypointense signal is often observed in bilateral precentral gyri in patients with amyotrophic lateral sclerosis (ALS) on T2 FLAIR sequences, known as "motor dark line", and the low signal occurred more frequently in ALS patients than in normal subjects [1]. Susceptibility-weighted imaging (SWI) is very sensitive to the susceptibility of various brain tissues, and has been widely used in the field of neuroimaging research [2]. We assumed that more prominent signal differences may be detected in the brain, especially in precentral gyri, in ALS patients on SWI, so a comparative study between ALS patients and normal subjects was prospectively conducted by using SWI.

Methods:
Twenty patients (13 males and 7 females, age ranged from 17 to 75 years, mean age 46.1 years; duration ranged from 4 to 192 months, mean duration 41.2 months) with definite or probable ALS (defined by the diagnostic criteria of El Escorial World Federation of Neurology) and 19 age and gender matched normal controls (11 males and 8 females, age ranged from 14 to 62 years, mean age 43.8 years) were enrolled.
MRI was performed using a 3.0 T MR scanner. Parameters for SWI were as follows: TR=32 ms, TE=19 ms, FA=20, slice thickness=2 mm, FOV=24×24 cm, matrix=448×384, NEX=0.75, and a total of 56 slices were acquired.
Phase shifts on corrected phase images were blindly measured by two experienced neuroradiologists, including bilateral precentral gyrus (PG), frontal cortex (FC), caudate nucleus (CA), globus pallidus (GP), and putamen (PU). For the measurement of phase shift value at PG, region of interest (ROI) was from 15 to 25 pixels, and ROI measurement was conducted for three times in each side, and the lowest value was used (Fig.1). For the measurement of phase shift values at FC (Fig.2. ROI: 15-25 pixels), GP (Fig.3. ROI: 25-35 pixels), PU (Fig.4. ROI: 25-35 pixels), and CA (Fig.5. ROI: shape of caudate nucleus), ROI was measured for only one time in each side. The phase shift values were then statistically compared between the two groups. In ALS patients, possible correlation was also evaluated between phase shift values at PG and disease duration, Norris Scale, and Amyotrophic Lateral Sclerosis Functional Rating Score (ALSFRS), respectively.

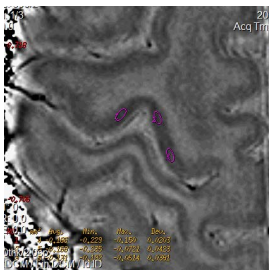
Fig.1. 3 ROIs at PG

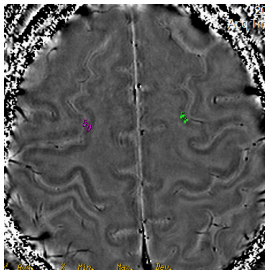
Fig.2. ROI at FC

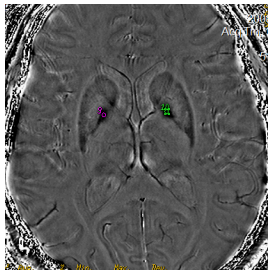
Fig.3. ROI at GP

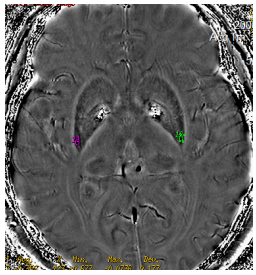
Fig.4. ROI at PU

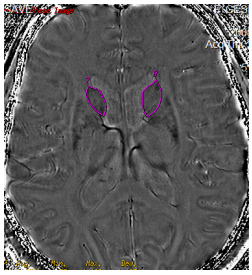
Fig.5. ROI at CA

Results: At the region of PG, FC, CA, GP, and PU, mean phase shift value were -0.176±0.050, -0.089±0.023, -0.065±0.016, -0.336±0.191, and -0.227±0.101, respectively in ALS group, and the values were -0.119±0.016, -0.885±0.015, -0.079±0.018, -0.329±0.136, and -0.229±0.083, respectively in control group. The phase shift value was significantly lower in ALS than in control only at PG (P=0.000), but no statistical differences between the two groups were shown in other regions (Fig.6). The phase shift value distribution with age also showed decreased values at PG in ALS patients (Fig.7). But no correlation was found between phase shift values at PG and disease duration, Norris Scale, or ALSFRS in ALS patients (P>0.05).

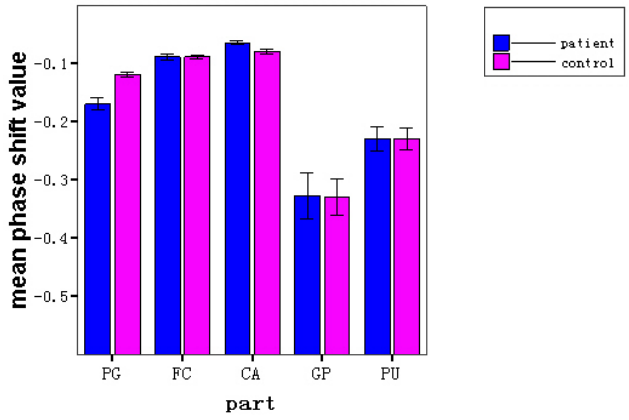

Fig.6. Mean phase shift value at different regions in ALS group and control group. Note the decreased value at PG in ALS

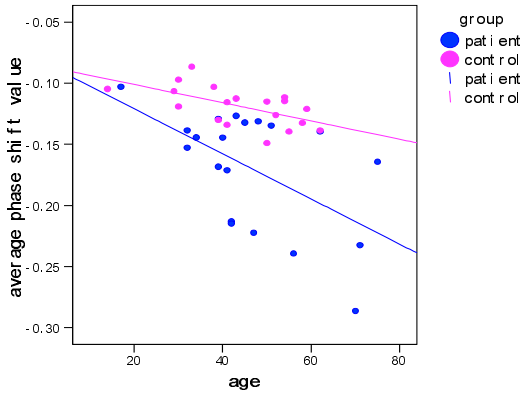

Fig.7. Dreased phase shift value distribution with age in ALS patients than in controls at PG

Discussion: As we expected, a marked lower phase shift value (darker signal) was seen at PG in ALS patients on SWI, yet the exact reason for this still remains poorly understood. The present result indicated that certain kinds of trace elements, probably including iron, with stronger susceptibility are accumulated in PG along with the process of neuronal loss and gliosis in the development of ALS. Further pathological studies and evidence are needed to elucidate the mechanism. SWI with quantitative measurement, however, is a potential sequence of choice in the study of neurodegenerative diseases.

References:
[1] Zhang L, et al. J Magn Reson Imaging, 2003, 17: 521-527.
[2] Sehgal V, et al. J Magn reson Imaging, 2005, 22: 439-450.