

Study of Neuropsychiatric systemic lupus erythematosus with DTI and FAIR

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Introduction:

Neuropsychiatric systemic lupus erythematosus (NPSLE) is a special disease that may occur in approximately 15% -75% of patients with systemic lupus erythematosus (SLE) ¹. The neuroimaging abnormalities have been described in a high percentage of patients with SLE and clinical evidence of CNS involvement (NPSLE) on brain CT, MRI, SPECT and PET². Conventional morphologic neuroimaging (i.e. CT and MRI) cannot help us either to differentiate between past (chronic) and recent (acute) lesions or to improve in our understanding of SLE pathogenesis because of the high probability of a NPSLE event during the course of the disease. The functional MR techniques including diffusion tensor imaging (DTI), which demonstrate the vectors corresponding to the strength and direction of the molecular water movement in the brain tissue, and arterial spin labeling (ASL) MRI with flow-sensitive alternating inversion recovery (FAIR) sequence, which can be used for measurement of relative cerebral blood flow (rCBF) changes, may help to further assess the possible abnormality in neuronal and neural vascular reactivity in the NPSLE patients. The aim of this study was to investigate whether the quantitative DWI analysis can depict cerebral abnormalities in patients with symptoms of NPSLE and if significant differences in measured apparent diffusion coefficient (ADC), fractional anisotropy (FA) value, and fair perfusion between these patients and normal controls exist.

Methods and Materials

Twelve female NPSLE patients (aged 16-56 years, mean 34.5 years) and 12 age-matched controls (female aged 20-55 years, mean 36.5 years) without psychiatric and neurological disorders were studied. All MR image, DTI and FAIR studies were performed on a 1.5T GE scanner with 8-channel NV coil. The MRI protocol included the following scans: 1) fast T1-weighted localizer; 2) whole-brain conventional T1-and T2-wighted anatomical scans; 3) Diffusion Tensor image was scanned by SE-EPI sequence with diffusion-weighted imaging scans of 15 different diffusion-weighting directions at $b=1000s/mm^2$, 2 NEX, 5 mm thickness without gap; 4) ASL MRI data were acquired using FAIR sequence with echoplanar readout at minimum TE, TR/ TI =900/1200ms, FA=90°, matrix 128×96, 5mm thickness with 1.5mm gap and 48 multi-phase. The total slices are 3 including corpus callosum and centrum semioval. ADC, FA and rCBF were measured respectively in bilateral splenium of corpus callosum, cingulated gyrus cortex, frontal white matter, partial white matter, and dorsolateral prefrontal cortex. For quantitative assessment, a 50 mm² ROI was equally used for measurements and the rCBF of that places were divided by the rCBF of veins in superior sagittal sinus. Statistical analysis was performed by SPSS 9.0.

Results

The conventional MR images of four patients are normal. The others show high signal on T2WI and low signal on T1WI. The measured values of ADC, FA and rCBF were shown in table 1. All of these three characters demonstrate significant difference between diagnostic patients group and control group ($p<0.05$), with a increase of ADC, decrease of FA and rCBF.

Discussion

Our study demonstrates alterations in patients with NPSLE by means of abnormal ADC, FA, rCBF, even though in normal-appearing gray and white matter brain parenchyma. These alterations may be based on loss of tissue integrity because of facilitating motility of free-water protons, small vessel degenerative vasculopathy and vasculitides. It is possible that DTI and FAIR could help in the diagnosis of NPSLE in the future. A combination of DTI and FAIR could highly increase the diagnosis accuracy in the earlier pathologic changes of NPSLE.

Table 1: FA, ADC and fair perfusion (mean ± SD) in control and patient group

| | ADC($\times 10^{-3}mm^2/s\pm SD$) | | | FA | | | rCBF/ rCBF of veins | | |
|--------------------------------|-------------------------------------|-----------|----------|-----------|-----------|----------|---------------------|-----------|----------|
| | NPSLE | Control | <i>p</i> | NPSLE | Control | <i>p</i> | NPSLE | Control | <i>p</i> |
| Splenium of corpus callosum | 0.95±0.07 | 0.85±0.08 | 0.002 | 0.68±0.06 | 0.75±0.03 | 0.005 | 0.13±0.04 | 0.16±0.03 | 0.009 |
| Cingulated gyrus cortex | 0.88±0.06 | 0.83±0.04 | 0.022 | 0.17±0.06 | 0.20±0.04 | 0.036 | 0.16±0.03 | 0.20±0.03 | 0.045 |
| Frontal white matter | 0.75±0.05 | 0.70±0.04 | 0.019 | 0.52±0.04 | 0.55±0.03 | 0.019 | 0.12±0.02 | 0.14±0.02 | 0.011 |
| Partial white matter | 0.76±0.07 | 0.72±0.05 | 0.032 | 0.51±0.05 | 0.58±0.02 | 0.046 | 0.12±0.03 | 0.16±0.02 | 0.009 |
| Dorsolateral prefrontal cortex | 0.85±0.07 | 0.80±0.03 | 0.018 | 0.20±0.05 | 0.27±0.04 | 0.038 | 0.15±0.02 | 0.17±0.03 | 0.026 |

Reference

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2. Castellino G, Govoni M, Giacuzzo S, F. et al. Optimizing clinical monitoring of central nervous system involvement in SLE. *Autoimmunity Reviews* 2008; 7: 297–304