MRI findings in neuropsychiatric lupus

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Introduction

MRI studies in neuropsychiatric systemic lupus erythematosus (NPSLE) patients have reported varying prevalences of abnormalities, probably due to the lack of a classification system for NP syndromes until 1999[1,2]. Aim of this study was to assess the range of MRI abnormalities in a group of SLE patients meeting the NP case definitions developed by the American College of Rheumatology (ACR) in 1999[3].

Materials and methods

Forty-nine SLE patients were selected fulfilling the 1982 revised ACR criteria for SLE[4] and at least one of the 1999 NPSLE case definitions[3] and who had had ≥1 MRI of the brain in an active state of the disease between 1989-2003 (45♀, 12-69 years, mean 37). All MRI's were acquired on a 0.5T or 1.5T MR scanner. In total, 166 MRI's were reviewed, including 160 T1/T2-weighted (fast) spin-echo images and 112 fluid-attenuated inversion recovery (FLAIR) images. For every patient, T2 or FLAIR images were available for evaluation of hyperintensities. Presence or absence of brain abnormalities on all MRI examinations were scored by a neuroradiologist blinded for the clinical status of each patient (table 1).

Results

In 65% of NPSLE patients, one or more abnormalities were found (table 1). Aspecific white matter hyperintensities were present in ± ⅔ of patients, whereas ± ⅓ of patients exhibited gray matter hyperintensities. Most striking was the finding of cortical gray matter hyperintensities in 29% of patients (fig. 1). In 79% of these cortical gray matter hyperintensities, involvement of the underlying white matter was observed. Furthermore, focal atrophy was found in 44% of cortical gray matter hyperintensities.

Conclusion

This is the first study to show presence of cortical gray matter hyperintensities in almost ⅓ of NPSLE patients. Cortical gray matter hyperintensities were very often associated with hyperintensity of the underlying white matter and focal atrophy. Furthermore, a high number of isolated white matter hyperintensities was found, which is in accordance with other studies[2]. White matter hyperintensities are non-specific and part of them might be unrelated to the disease. However, the new finding of specific cortical gray matter hyperintensities with white matter involvement and focal atrophy in a significant number of NPSLE patients sheds new light on the pathogenesis of this disease. A quantitative neuroimaging study in NPSLE has recently suggested abnormalities in the gray matter not visible on conventional MRI[5]. The gray matter findings in this conventional MRI study may result from the same pathologic process, suggesting cortical gray matter damage as an important etiologic factor in NPSLE.

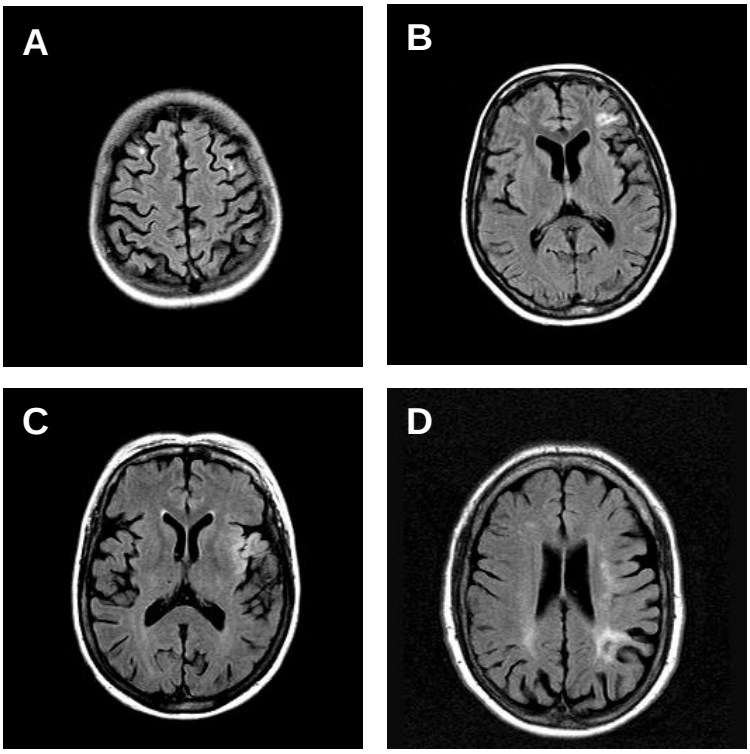

Figure 1.
FLAIR images showing hyperintensities of (A) frontal cortical gray matter, (B) frontal cortical gray matter with involvement of the subcortical white matter, (C) left insular gray matter and (D) left parietal cortical gray matter with involvement of the subcortical white matter and focal cortical atrophy

Table 1.

Cross-sectional MRI findings (n=49)	***n***	***%***
White matter hyperintensity	**31**	**63**
Periventricular	6	12
Deep white matter	31	63
Deep white matter <3mm	15	31
Deep white matter >3mm	28	57
White matter parenchymal loss	**8**	**16**
Loss of volume	2	4
Defect	6	12
Gray matter hyperintensity cortex	**14**	**29**
T1 hyperintensity	3	6
T2 hyperintensity	14	29
Gray matter parenchymal loss	**9**	**18**
Loss of volume	8	16
Defect	3	6
Basal ganglia hyperintensity	**3**	**6**
Basal ganglia defect	**5**	**10**
Infratentorial hyperintensity	**4**	**8**
Cerebellum	2	4
Brainstem	4	8
Infratentorial defect	**8**	**16**
No abnormalities	**17**	**35**

References

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