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MR Imaging of Systemic Lupus Erythematosus Involving the Brain

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Focal lesions were demonstrated on magnetic resonance (MR) imaging in eight patients with systemic lupus erythematosus and recent onset of neuropsychiatric symptoms. Corresponding findings were visible in only two of seven patients who had computed tomographic (CT) scans. Three patterns of disease were observed on MR when it was performed with a pulse repetition rate of 2000 msec. The first pattern was that of cerebral infarction, with a relatively large area of increased intensity. The second pattern, multiple small areas of increased intensity, may have been secondary to microinfarctions. The third pattern was that of focal areas of increased intensity, predominantly in the cerebral gray matter. In two of three patients with the third pattern, partial or complete resolution was observed on follow-up MR images obtained several weeks after the initial studies.

Involvement of the brain is a major cause of morbidity and mortality in systemic lupus erythematosus (SLE). Both the treatment of cerebral SLE and research into new forms of therapy are severely hampered by the current lack of an accurate method of diagnosing and following the condition. The manifestations of cerebral SLE are varied and include psychosis, organic brain syndromes, seizures, migrainous phenomena, visual disturbances, cranial neuropathies, and focal neurologic findings [1–3]. Differential diagnosis, dependent on the findings in a given patient, can include drug-induced phenomena (particularly due to steroids), infection, uremia, hypertension, electrolyte abnormalities, and psychiatric disorders [1]. We describe the magnetic resonance (MR) imaging findings in eight patients with clinical evidence of SLE of the brain, and suggest that this recently developed technique may well be the method of choice in the diagnosis and clinical follow-up of patients suspected of having cerebral SLE.

Subjects and Methods

As part of ongoing research into the role of MR in the evaluation of patients with SLE, about 25 patients have undergone MR of the brain. Informed consent was obtained from all patients. SLE was diagnosed in accordance with the revised standards of the American Rheumatism Association [4]. Eight patients with positive MR scans (excluding atrophy and minimal nonspecific periventricular abnormalities) and clinical findings suggestive of acute (<1 month) cerebral involvement were selected for inclusion in the current study. It was possible to obtain follow-up MR images in four of the patients; a prior MR examination was available in two. Detailed chart reviews and correlation with available cranial computed tomographic (CT) scans were performed for the eight patients.

MR imaging was performed using a Diasonics MT/S system (Diasonics, South San Francisco, CA) with a 3.5 kG superconducting magnet. Spin-echo (SE) pulse sequences were used exclusively. Repetition times (TRs) of 1500 or 2000 msec were used in all studies; additional images with a TR of 1000 msec were collected for most cases. Images with echo delays (TEs) of both 28 and 56 msec were obtained at all levels. Coronal images with a 2000 msec TR were obtained in several cases. CT was performed with an EMI 1010 scanner (four

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TABLE 1: **Clinical, MR, and CT Findings in Systemic Lupus Erythematosus**

Case No. (age, gender)	Clinical Status	MR Findings	CT Findings (Scanner/Contrast or No Contrast)
1 (29,F)	Overlap syndrome meeting criteria for SLE, last 10 yr; presented with aphasia, which improved markedly later	Lesion in L frontal gray matter, almost resolved 2 weeks later	Negative (EMI/C)
2 (51,F)	SLE with behavior abnormalities for 20 yr, worse last 5 days; bitemporal seizure activity on EEG 1 month later	Multiple small, bilateral white-matter lesions, not present on MR 6 months earlier and unchanged on MR 1 month later	CT at time of MR negative (GE/NC)
3 (33,M)	SLE for 1 yr; presented with mild L hemiparesis, which resolved during next 2 months; seizures	Lesion in R anterior parietal gray matter, unchanged on MR 2 and 3½ months later; previously normal MR	CT not done
4 (33,F)	SLE for 2–3 yr; presented with R-sided weakness	Small L frontoparietal lesion	Negative (GE/C)
5 (20,F)	SLE for 3 yr; presented with seizure, hypertension, and metabolic abnormalities	L frontal, predominantly gray-matter lesion that resolved 1 month later	Atrophy only (GE/NC)
6 (41,F)	16 yr history of SLE or overlap syndrome; altered mentation and seizures last month	Multiple white-matter lesions	Large ventricles only (GE/NC)
7 (36,F)	SLE for 20 yr; previous R middle-cerebral-artery-area infarct; presented with seizures, vasculitis, and hypoxia	Large lesion in R frontoparietal area, parts of which suggested loss of substance	Large corresponding area of hypodensity (EMI/NC)
8 (33, F)	SLE for 20 yr; previous stroke; presented with altered vision	R parietal white-matter lesion	Corresponding area of decreased density (EMI/C)

Note.—All lesions showed increased signal on long TR SE images, except in case 7, in which there was a mixed-signal lesion. SLE = systemic lupus erythematosus; yr = year(s); R = right; L = left; EEG = electroencephalogram; GE = GE CT/T 8800; EMI = EMI 1010; C = contrast enhanced; NC = not contrast enhanced.

patients), a General Electric 8800 CT/T scanner (three patients), or both (one patient); one patient did not undergo CT.

Results

Images obtained with 2000 msec TRs were almost always most useful diagnostically. Although images with 28 msec TEs had signal-to-noise ratios superior to those with 56 msec TEs, pathology was generally demonstrated better in the latter. We have since studied one SLE patient with a lesion well demonstrated on a 500 msec TR image, just visible on a SE 2000/28 scan, and nearly invisible on a SE 2000/56 scan. The coronal images, when available, were frequently useful in verifying equivocal findings noted on the axial images. MR and relevant clinical and CT findings are summarized in table 1.

In our limited number of patients, three distinct patterns of disease emerged. The first pattern consisted of large areas of increased MR intensity, which involved mostly white matter. This pattern was present in cases 7 (fig. 1A) and 8. In both, CT revealed corresponding areas of hypodensity consistent with previous infarction (fig. 1B), a diagnosis supported in both cases by the clinical history.

The second pattern demonstrated small, sometimes multiple, focal areas of increased MR intensity in the cerebral white matter. Present in cases 2, 4 (fig. 2), and 6, these lesions were not visible on CT, even in retrospect (though contrast-enhanced scans were not available in two of these three patients). The lesions may well represent the microinfarctions of cerebral SLE described pathologically [2], but we have as yet no pathologic data, and a follow-up MR study is available on only one of these patients.

The third pattern consisted of focal areas of increased MR intensity seen predominantly in cortical gray matter. The lesions, present in cases 1, 3, and 5, were solitary. CT was performed in two of the patients and did not demonstrate corresponding abnormalities. Follow-up MR scans were available in all three patients. In two of the three, there was complete or nearly complete resolution of the MR findings after 2 and 3 weeks (figs. 3 and 4); in the third, there may have been marginal improvement. All three patients had substantial improvement in their clinical symptoms over the period of several weeks.

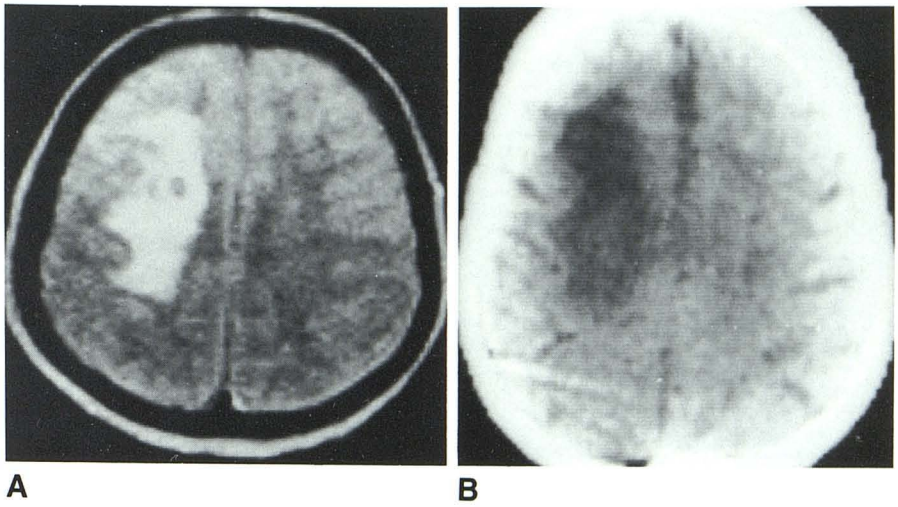

Fig. 1.—Case 7. **A**, SE 2000/56. Large area of increased intensity. **B**, Nonenhanced CT scan. Hypodensity in same location. CT findings are consistent with previous infarction.

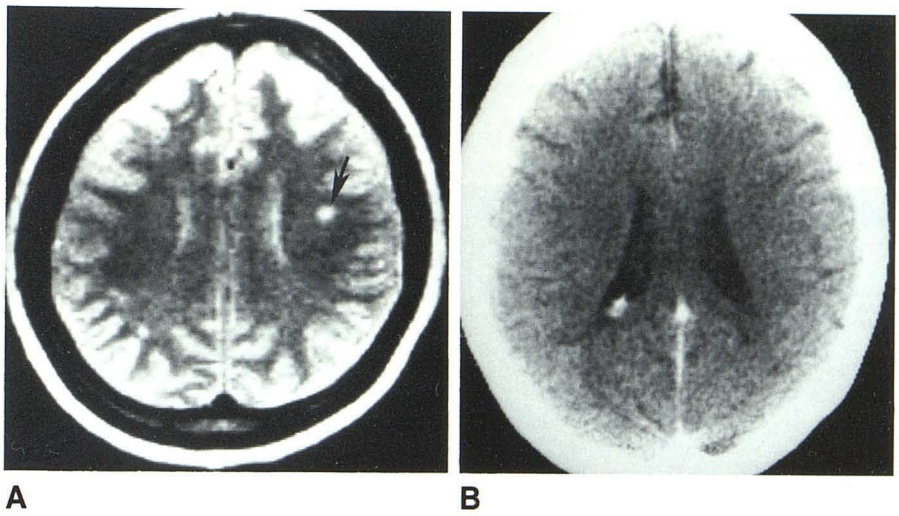

Fig. 2.—Case 4. **A**, SE 2000/56. Focal area of frontoparietal increased intensity in left-cerebral-hemisphere white matter (*arrow*). **B**, Contrast-enhanced CT scan. No abnormality.

Discussion

SLE is a multisystem autoimmune disease of imperfectly understood pathogenesis. The assessment of involvement of the brain has been hampered by the lack of a reliable means of diagnosis, but cerebral involvement has been reported in as many as 75% of SLE patients [3]. The pathologic findings are varied and include vasculopathy, infarction, and hemorrhage, as well as infection. Both large and microscopic infarctions are common [2]. In the past, however, reliance on autopsy series for clinicopathologic correlation (necessitated by the lack of a suitable diagnostic imaging technique) has resulted in quite limited data on morphologic correlates of clinical events.

The diagnosis of SLE of the brain can be difficult. In addition to physical examination, cerebrospinal fluid analysis, electroencephalography, and evoked-response studies can all be useful, but none is generally definitive.

Angiography is useful in selected cases [5]. However, before the introduction of MR, CT was the most useful imaging technique. Unrelated or secondary etiologies such as tumor or infection can often be diagnosed or excluded. Hemorrhage and infarction, particularly when large, can be detected.

Atrophy has been reported as the most common CT finding in SLE of the brain [6], though steroid treatment has been reported to produce a similar pattern [7], and there is controversy as to whether this CT finding is related to the disease process (e.g., repeated microinfarction) or is a consequence of steroid therapy [8, 9]. Of direct relevance to the present study is a case report of thalamic and internal capsular CT hypodensity in a patient with SLE who developed sudden onset of severe hypertension, convulsions, and stupor; both clinical and CT findings resolved after 1 week. The authors hypothesized that edema was a possible etiology of the CT findings [10].

One report on the MR findings in SLE of the brain has appeared in the literature [11]. Nine patients were included in the report; all had CT examinations also. All lesions seen on CT were also visible on MR; however, MR proved to be

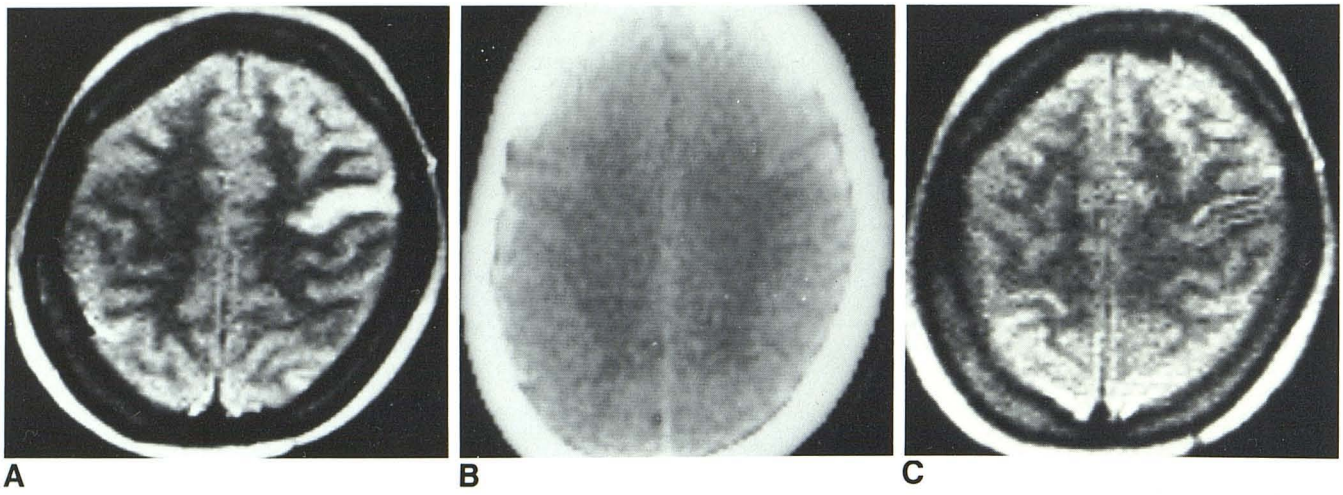

Fig. 3.—Case 1. **A**, SE 2000/56. Increased intensity in part of left frontal gray matter, near motor cortex. **B**, Contrast-enhanced CT scan. No abnormality. **C**, SE 2000/56 image 2 weeks later. Nearly complete resolution of high-intensity signal.

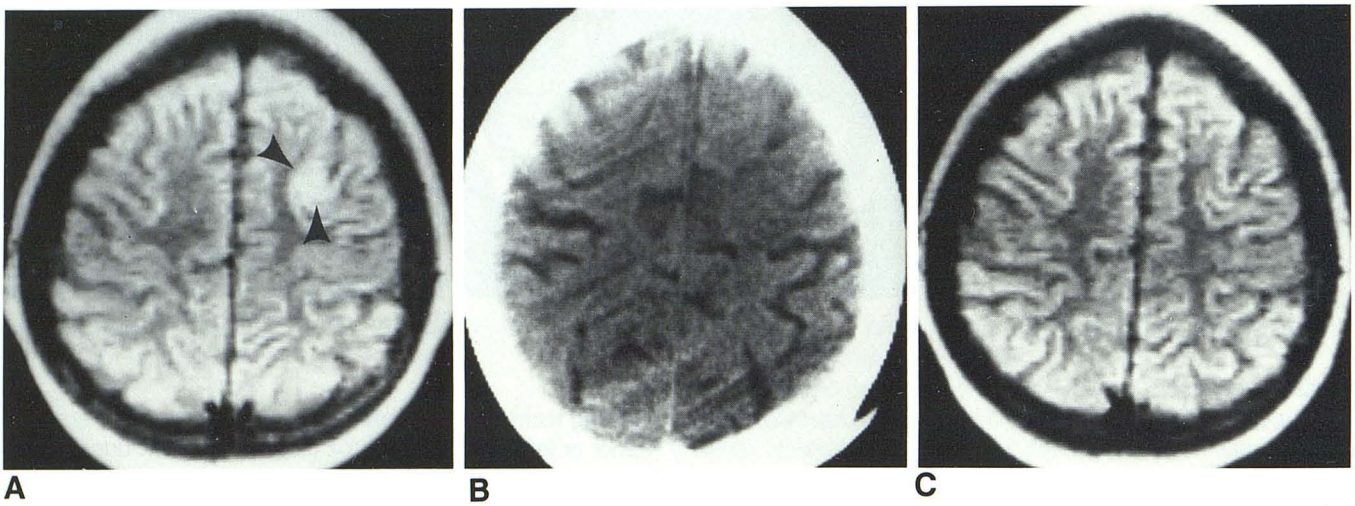

Fig. 4.—Case 5. **A**, SE 2000/28. Focal area of increased intensity in left frontal lobe, predominantly gray matter (*arrowheads*). **B**, Nonenhanced CT scan. No abnormality. **C**, SE 2000/28 image 3 weeks later. Essentially complete resolution.

substantially more sensitive. Many lesions not visible on CT were noted on MR, and in general, MR demonstrated the abnormalities with greater clarity. MR showed areas of increased T1 and T2 relaxation times, presumably secondary to edema.

Our own experience supports these MR and CT findings. Several of our patients displayed either large or small areas of increased MR intensity on scans obtained some time after the onset of clinical symptoms. These findings were consistent with the MR abnormalities, and most likely represented regions of large or small infarction. In addition, in two of our patients, MR abnormalities were seen but largely disappeared over the course of several weeks. We suggest these represented a reversible and perhaps noninfarctive process, at least in some cases. Indeed, the clinical course in patients with cerebral SLE is often variable, with resolution occurring in some patients, but not in others.

MR is of demonstrated utility in the evaluation of patients with clinical evidence of SLE of the brain. It seems reasonable to propose that the presence of focal findings on MR images implies true cerebral disease, which is an important point in differentiating drug effects or primary psychiatric illness from cerebral involvement in SLE patients with nonspecific symptomatology. Our limited experience suggests it is not yet

possible to distinguish prospectively irreversible from reversible brain injury with MR imaging. A more specific diagnosis may be possible in the future. Gray-matter lesions may prove to have a different prognosis than white-matter abnormalities. Though both reversible and irreversible processes can result in edema, the exact nature of the biochemical alteration may vary, and it may be possible reliably to differentiate the two processes as imaging equipment and expertise improve. Differences in relaxation properties of different types of edema have been reported in vitro [12], and early efforts to apply this to in vivo MR have been described [13].

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