

## Lyme Disease of the CNS: MR Imaging Findings in 14 Cases

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The MR images of 14 patients with clinical diagnoses of Lyme disease, CNS complaints, and positive Lyme titers were reviewed. MR examinations were abnormal in 43%. Areas of abnormal signal were identified within the cerebral white matter as well as within the brainstem.

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Lyme disease is a multisystem inflammatory disease caused by the spirochete *Borrelia burgdorferi*. It is transmitted by the ixodid ticks and has been reported in 32 states [1]. The most common ixodid tick vector is the deer tick (*Ixodes dammini*), although other ixodid ticks may also be vectors. In addition to the white-tail deer, the white-footed mouse may also serve as a reservoir [2]. Affected ticks have been found not only in woodlands but also in suburban lawns [1].

### Materials and Methods

A retrospective study was undertaken to identify those patients who had been referred to our institution for MR examination of the brain and who either had a history of diagnosed Lyme disease or were thought to have Lyme disease. We identified 17 patients who had been referred over a 14-month period. Of these 17 patients, 14 had positive serum Lyme titer tests. The other three patients were not included in the study even though patients with early Lyme or patients treated early with antibiotics may have the disease with a negative titer [3]. The MR images of the remaining 14 patients were then reviewed by a senior neuroradiologist. The imaging examinations had been performed on a 1.5-T unit (General Electric, Milwaukee, WI) with a spin-echo technique. T1-weighted images had been obtained in both axial and sagittal planes using 600/20 (TR/TE), and T2-weighted axial images had been obtained using 2000/80. Contrast material was not given. The patients ranged in age from 7 to 73 years old (average age 29; mean, 32).

### Results

Six of the 14 patients examined had MR abnormalities. These patients had been referred for complaints or diagnoses of headache, blurred vision, hearing loss, radiculopathy, or transverse myelitis. The MR images showed focal areas of abnormal signal intensity, which were mildly hypointense on T1-weighted images and markedly hyperintense on T2-weighted images. The number of lesions ranged from one to 27 (Table 1). The lesions in patient 5 (a 73-year-old woman) are not discussed here because, at her age, some lesions may have been caused by microvascular disease rather than Lyme. The remaining five patients had a total of 41 lesions. The size of lesions ranged from 12 mm (in the patient with a single brainstem lesion, Fig. 1) to 2 mm. Most lesions were about 2-3 mm. No mass effect was identified for any lesion. The lesions could be identified on both the T1 and T2 sequences, but they were visualized better on the T2 sequence. Titers

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ranged from mildly to markedly positive. There was no correlation between the duration of disease and the number of lesions.

### Discussion

Lyme disease has been divided into three clinical stages. Stage I disease includes constitutional flu-like symptoms and a characteristic expanding skin lesion (erythema chronicum migrans). Stage II disease is characterized by cardiac and neurologic manifestations. Stage III disease is characterized by arthritis and chronic neurologic syndromes. There may, however, be considerable overlap of the stages, and a patient may present with stage III disease [4]. Lyme disease is now the most commonly reported tick-borne illness in the United States [5]. Between 10% and 15% of patients with Lyme disease develop CNS abnormalities [6, 7]. The neurologic syndromes are many and may occur alone or in combination; they include peripheral neuropathies, radiculopathies, myelopathies, encephalitides, meningitides, pain syndromes, cognitive disorders, and movement disorders (chorea, ataxia) [8–11]. Treatment is with antibiotics, and varies depending on the duration of the disease and the age of the patient [3]. The exact mechanism for CNS manifestations of Lyme disease is

not known; both direct brain invasion and vasculopathy have been postulated as mechanisms [12].

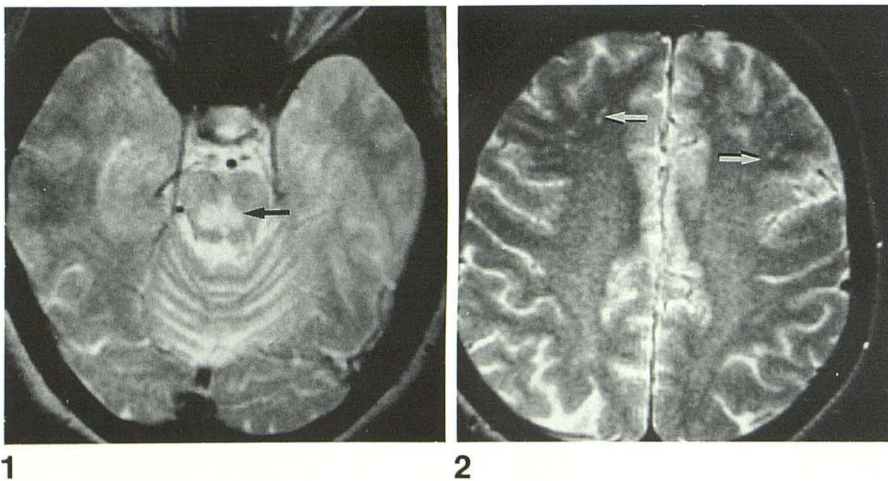
Our series is small, making it difficult to draw any conclusions about distribution; however, it does demonstrate that multiple areas of the brain can be affected. There also appears to be a predilection for involvement of the subcortical white matter of the frontal and (to a lesser extent) parietal lobes (Fig. 2). Some of these subcortical lesions looked like high-convexity, dilated Virchow-Robin spaces, which have been reported in normal aging brain. They are relatively rare and show a strong association with age (they become more common with advancing age) [13]. In our series, all four patients less than 40 years old had subcortical lesions; one of these had 26 foci of abnormality (see Table 1). These foci in younger patients represent disease and not dilated Virchow-Robin spaces. They may represent dilated perivascular spaces; however, we think it is more likely that they represent foci of perivascular inflammation or demyelination. Other subcortical foci were larger and showed no similarity to dilated Virchow-Robin spaces (Fig. 3).

Previous reports have shown lesions within the frontal lobes, parietal lobes, temporal lobes, thalami, occipital lobes, corpus callosum, and pons [11, 14, 15]. These previously reported lesions had been identified by CT and/or MR imaging. In our series, the patient with the highest number of lesions (patient 1) had had a negative CT examination 3 days before her MR images were obtained. MR imaging is considered to be significantly more sensitive than CT in detecting CNS Lyme. Six-month follow-up MR images were obtained in one patient (patient 6), and showed persistence of three of the four lesions; the other lesion was small and may have been missed because of scan section. Although we did not administer a contrast agent, previous contrast enhancement with CT examination has been reported [12, 14] and would be expected with MR.

Lyme disease can be difficult to diagnose because its symptoms are many and the presentation may show various combinations of them. The disease may also be cyclical. Lyme disease may therefore be confused clinically with a variety of other disease processes, including multiple sclerosis [16].

**TABLE 1: Distribution of Lesions in Patients with CNS Lyme Disease**

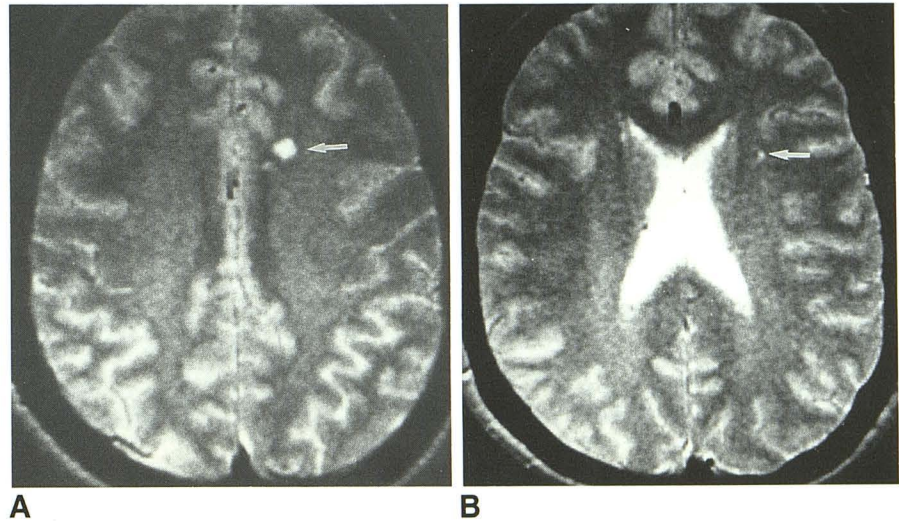
Patient No.	Age	No. of Lesions	Distribution of Lesions
1	37	27	25 subcortical frontal, 1 external capsule, 1 subcortical parietal
2	42	1	1 upper pons and lower brainstem
3	16	3	1 medulla, 1 subcortical frontal, 1 periventricular frontal
4	39	6	6 subcortical frontal
5	73	20	1 thalamus, 1 subcortical insula, 12 subcortical frontal, 3 subcortical parietal, 3 periventricular parietal
6	41	4	2 subcortical frontal, 1 subcortical parietal, 1 periventricular parietal



**Fig. 1.**—Patient 2: 42-year-old woman with transverse myelitis. Abnormal signal intensity (arrow) is shown in the pons on this T2-weighted MR image (2000/80).

**Fig. 2.**—Patient 1: 37-year-old woman, 2 months after tick bite. Multiple foci of abnormal signal (arrows) are seen in subcortical white matter on this T2-weighted image (2000/80).

Fig. 3.—Patient 6: 41-year-old woman.  
 A, MR image (2000/80) shows larger subcortical lesion (arrow) that does not look like a dilated Virchow-Robin space.  
 B, Lower axial MR image (2000/80) shows periventricular lesion (arrow).



The MR appearance of some lesions in CNS Lyme disease may be periventricular (Fig. 3).

In summary, a significant number of patients with CNS Lyme disease have positive MR examinations. While a few case reports have appeared in the pediatric and neurologic literature, little has been published in the radiologic literature [14, 16]. We believe that it is important to consider CNS Lyme in the differential diagnosis of white matter lesions. Awareness of the MR findings of Lyme disease is important because this disorder is treatable even though it is difficult to diagnose. MR imaging may therefore play an important role in suggesting the diagnosis and in differentiating Lyme disease from other white matter diseases.

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