



Providing Choice & Value
Generic CT and MRI Contrast Agents

**FRESENIUS
KABI**

CONTACT REP

AJNR

Biopsy-confirmed CNS Lyme disease: MR appearance at 1.5 T.

S E Rafto, W J Milton, S L Galetta and R I Grossman

AJNR Am J Neuroradiol 1990, 11 (3) 482-484

<http://www.ajnr.org/content/11/3/482.citation>

This information is current as
of July 29, 2025.

Biopsy-Confirmed CNS Lyme Disease: MR Appearance at 1.5 T

Stein E. Rafto,¹ Walter J. Milton,¹ Steven L. Galetta,² and Robert I. Grossman¹

Lyme disease is a spirochetal infection caused by *Borrelia burgdorferi* found worldwide in deer, mice, raccoons, and birds, with incidental transmission to humans by infected ixodid ticks [1–6]. Initially described in Europe in 1909 [3], additional manifestations have been recognized in the last decade in which at least a tenfold increase in reported cases has occurred in the United States [2]. Although neurologic manifestations are frequent and protean, this case report is among the first in the radiologic literature to demonstrate CT and MR abnormalities in biopsy-confirmed CNS Lyme disease.

Case Report

A healthy 41-year-old wife of a deer hunter initially complained of a red ulcerated anterior chest wall lesion. The lesion was dissected and yielded a tick, with subsequent resolution of the lesion. Approximately 3 months later she noted flulike symptoms and an unsteady gait. These symptoms resolved after a 2-week course of meclizine and a tetracycline-derivative antibiotic. A 6-month symptom-free interval was then followed by recurrent unsteady gait and left hemibody seizures. MR imaging of the brain demonstrated multiple parenchymal lesions suggesting a demyelinating or inflammatory process. The patient's neurologic status continued to decline despite a second course of oral tetracycline, and she was referred to our institution for further evaluation.

Serum Lyme indirect immunofluorescence assay (IFA) was positive with an IgG titer of 1:256, and Lyme enzyme-linked immunosorbent assay (ELISA) was elevated at 1.23 (normal range <0.9). A rapid plasma reagin and *Treponema pallidum* hemagglutination assay were nonreactive. Serial CSF examinations were unrevealing except for the presence of oligoclonal bands. The patient was ataxic and was oriented only to person at the time of our initial high-field (1.5-T) MR examination (G.E. Medical Systems, Milwaukee). This demonstrated multiple lesions (high signal on long TR/TE [3500/30,90/2 TR/TE/excitations] images; larger lesions visible as low signal on short TR/TE [600/20/2] images) scattered throughout the brain parenchyma but principally involving white matter, ranging from punctate to 2.5 cm in size with irregular margins (Fig. 1A). Virtually all lesions revealed a ring-enhancing pattern following the IV administration of 0.1 mmol/kg Gd-DTPA (Berlex Laboratories, Cedar Knolls, NJ) (Fig. 1B). The patient became lethargic and dysphasic despite IV antibiotics, and a second MR study 18 days later showed new white matter lesions (Fig. 2) as well as a new 5-mm left thalamic lesion. Post-gadolinium

images also demonstrated enhancement in the interpeduncular cistern consistent with leptomeningeal inflammation (Fig. 3). Because of the patient's declining mental status, stereotaxic CT-guided biopsy of a contrast-enhancing nodule in the posterior left frontal lobe was performed (Fig. 4). Histopathology examination reported spongiform-like change with reactive astrocytes and areas of necrosis (Fig. 5A). Silver stain from a paraffin squash preparation displayed two spirochetes morphologically compatible with *Borrelia burgdorferi* (Fig. 5B). A third MR examination, although severely degraded by motion due to the patient's declining mental status, showed progressive enlargement of the multiple lesions. This patient has subsequently been treated with several courses of IV ceftriaxone and corticosteroid therapy with dramatic improvement in her neurologic status. She is stable 1 year later; residual deficits include moderate cognitive impairment, focal motor seizures, and gait ataxia. A recent follow-up MR examination 13 months after presentation revealed no abnormal enhancement in the parenchyma or meninges, and there was improvement or resolution of many lesions on long TR/TE images.

Discussion

Like other spirochetal infections, that caused by *B. burgdorferi* has several clinical stages, and patients typically experience remissions and exacerbations [7]. Lyme disease usually begins with the characteristic rash, erythema chronicum migrans, which may be accompanied by headache, stiff neck, myalgia, or neuralgia (stage 1) [8, 9]. CSF is usually normal in this stage. Weeks to months later patients may experience recurrent or new neurologic manifestations, usually in the form of overt meningitis, radiculoneuropathy, or cardiac abnormalities (stage 2) [7]. Weeks to years later the patient may develop arthritis or CNS dysfunction (stage 3) [8]. These stages may overlap or occur alone, frequently manifesting in the summer or fall following outdoor activities in endemic areas [7].

Neurologic manifestations are extremely varied [10] and symptoms may simulate demyelinating diseases, such as multiple sclerosis [7, 11]. Pathophysiology includes direct spirochetal invasion of affected tissues and may also involve immunologic mechanisms or vasculitic processes [10, 12, 13]. Treatment is primarily by antibiotics although there may be some, as yet unresolved, role for corticosteroids [10, 14–

Received October 17, 1989; revision requested December 11, 1989; revision received January 5, 1990; accepted January 8, 1990.

¹ Department of Radiology, Neuroradiology Section, Hospital of the University of Pennsylvania, 3400 Spruce St., Philadelphia, PA 19104. Address reprint requests to R. I. Grossman.

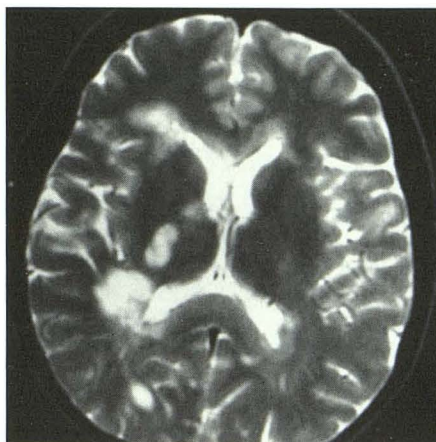
² Department of Neurology, Hospital of the University of Pennsylvania, Philadelphia, PA 19104.

AJNR 11:482–484, May/June 1990 0195–6108/90/1103–0482 © American Society of Neuroradiology

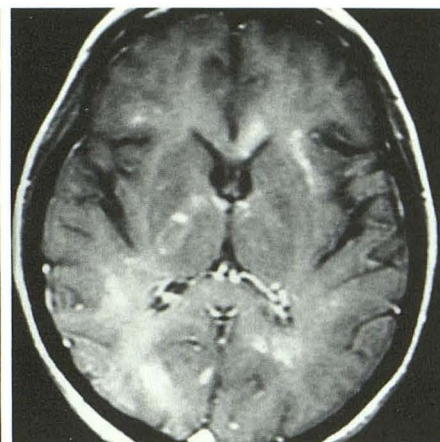
Fig. 1.—MR images at level of foramen of Monro.

A, 3500/90 image shows multiple areas of abnormally increased signal located predominantly in the white matter.

B, 600/20 image after Gd-DTPA at similar location to A shows ring-enhancing lesion in posterior limb of right internal capsule. Most of the other areas of enhancement were also shown to be ring-enhancing on adjacent slices.



A



B

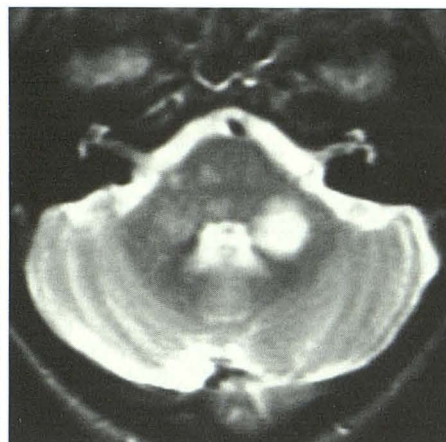
Fig. 2.—MR images through posterior fossa.

A, 3500/90 image from our initial MR study shows increased signal in cerebellar peduncles.

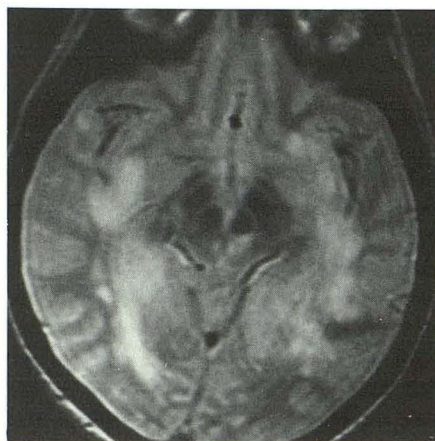
B, Follow-up examination 18 days later shows significant increase in size of left cerebellar peduncle lesion.



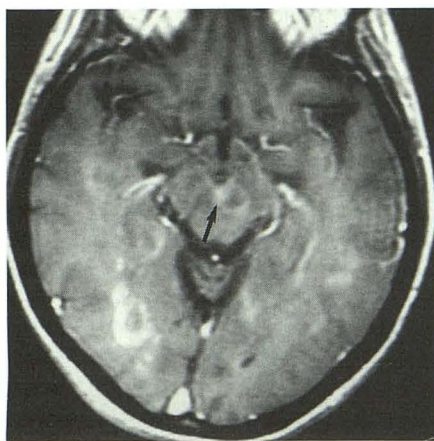
A



B



A



B

Fig. 3.—MR images at level of interpeduncular cistern.

A, 3500/30 image shows multiple parenchymal abnormalities.

B, 600/20 image after Gd-DTPA shows meningeal enhancement in interpeduncular cistern (arrow) along with multiple enhancing intraaxial lesions.

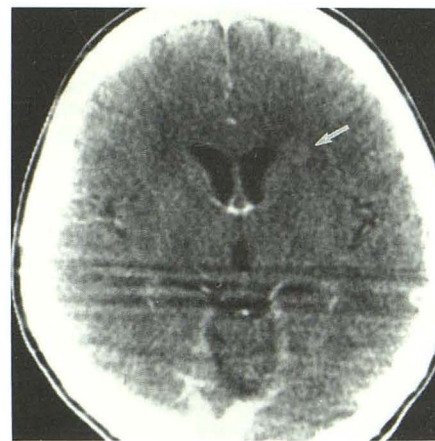


Fig. 4.—Contrast-enhanced CT scan shows subtle nodular enhancement adjacent to frontal horn of left lateral ventricle (arrow). Streak artifacts are from stereotaxic holder. Lesion was successfully biopsied immediately after this image was obtained.

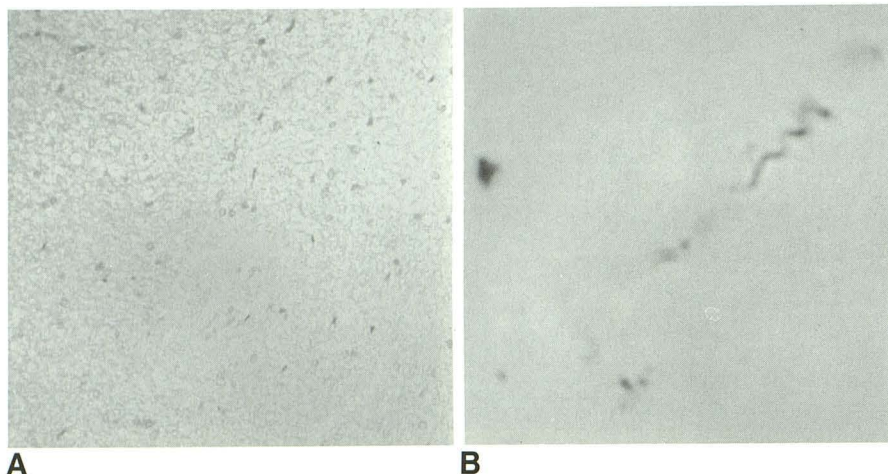


Fig. 5.—Photomicrographs from stereotaxic biopsy of lesion shown in Fig. 4.

A, H & E stain shows spongiform change and reactive astrocytes.

B, Silver stain and higher magnification shows spirochetal organism with the morphologic characteristics of *Borrelia burgdorferi*.

16]. Our patient with multiple lesions continued to deteriorate clinically and radiologically despite two courses of IV ceftriaxone therapy. The addition of corticosteroid therapy provided a dramatic improvement in the patient's aphasia and ability to ambulate.

Diagnosis is based primarily on clinical findings, positive serology for *B. burgdorferi*, the exclusion of other causes of CNS abnormalities, and response of symptoms to antibiotics. Spirochetes do not grow well in vitro and the yield in culture of *B. burgdorferi* is very low [1]. There is currently no routine test available that indicates active infection with *B. burgdorferi* in the CNS. Thus, even with positive serology other causes that might explain the neurologic findings must be excluded. There are scattered reports in the neurology literature that describe head CT and MR findings in patients with Lyme disease. Many of these are reported as normal, but abnormalities have ranged from periventricular signal abnormalities simulating multiple sclerosis [10, 17] to hydrocephalus [10] to hypodense thalamic or basal ganglia lesions [10, 18]. In 10 patients with neurologic manifestations of clinical and seropositive-confirmed Lyme disease imaged at our institution in 2½ years, this was the only one with any significant abnormality on CT and MR. However, most of the other CT and MR examinations were performed in patients with stage 1 or early stage 2 disease. The image appearance in our case was somewhat nonspecific. The principal radiologic differential diagnoses in combination with the clinical findings were multiple sclerosis, other demyelinating processes (acute disseminated encephalomyelitis [ADEM]), vasculitis, and inflammatory or infectious processes. The presence of meningeal enhancement and/or the enhancement of virtually all white matter lesions would be exceedingly unusual in multiple sclerosis in our experience, but might be compatible with ADEM. Only further experience will answer questions about the typical appearance of neuroborreliosis, and the significance of parenchymal and meningeal gadolinium enhancement [19].

In summary, a negative head CT or MR finding should not deter the diagnosis of neuroborreliosis, even in the setting of focal neurologic findings. The presence of meningeal enhancement and/or single or multiple parenchymal abnormalities on MR in the appropriate clinical setting (endemic area, erythema, chronicum migrans, meningitis, cranial neuritis, radiculoneuritis) should place Lyme disease in the differential

diagnosis and prompt serologic testing to further evaluate for this treatable disease.

ACKNOWLEDGMENT

We acknowledge Paul Duray for his expertise in pathology.

REFERENCES

1. Pachner AR. Spirochetal diseases of the nervous system. *Neurol Clin* 1986;4:207–222
2. Wickelgren I. At the drop of a tick. *Sci News* 1989;135:76–82
3. Afzelius A. Report to Verhhandlungen der dermatologischen gesellschaft zu Stockholm on December 16, 1909. *Arch Dermatol Syph* 1910;101:404–406
4. Garin-Bujadoux C. Paralyse par les tiques. *J Med Lyon* 1922;3:765–767
5. Bannwarth A. Chronische lymphozytare meningitis, entzündliche polyneuritis und rheumatismus. *Arkhy Psychiatr Nervenkr* 1941;113:284–376
6. Steere AC, Malawista SE, Snyderman DR, et al. Lyme arthritis: an epidemic of oligoarthritis in children and adults in three Connecticut communities. *Arthritis Rheum* 1977;20:7–17
7. Pachner AR, Steere AC. The triad of neurologic manifestations of Lyme disease: meningitis, cranial neuritis, and radiculoneuritis. *Neurology* 1985;35:47–53
8. Steere AC, Malawista SE, Nardin JA, Ruddy S, Askenase PW, Andiman WA. Erythema chronicum migrans and Lyme arthritis: the enlarging clinical spectrum. *Ann Intern Med* 1977;86:685–698
9. Steere AC, Bartenhagen NH, Craft JE. The early clinical manifestations of Lyme disease. *Ann Intern Med* 1983;99:76–82
10. Finkel MF. Lyme disease and its neurologic complications. *Arch Neurol* 1988;45:99–104
11. Eichenfield AH. Diagnosis and management of Lyme disease. *Pediatr Ann* 1986;9:583–594
12. Duray PH, Steere AC. Clinical pathologic correlations of Lyme disease by stage. *Ann NY Acad Sci* 1988;539:65–79
13. Aberer E, Brunner C, Suchanek G, et al. Molecular mimicry and Lyme borreliosis: a shared antigenic determinant between *Borrelia burgdorferi* and human tissue. *Ann Neurol* 1989;26:732–737
14. Dattwyler RT, Halperin JJ, Volkman DJ, Luft BJ. Treatment of late Lyme disease borreliosis—randomized comparison of ceftriaxone and penicillin. *Lancet* 1988;1:1191–1194
15. Steere AC. Lyme disease. *N Engl J Med* 1989;321:586–596
16. Pachner AR. *Borrelia burgdorferi* in the nervous system: The new "great imitator." *Ann NY Acad Sci* 1988;539:56–64
17. Pachner AR, Duray P, Steere AC. Central nervous system manifestations of Lyme disease. *Arch Neurol* 1989;46:790–796
18. Wokke JHJ, vanGijn J, Elderson A, Stanek G. Chronic forms of *Borrelia burgdorferi* infection of the nervous system. *Neurology* 1987;37:1031–1034
19. Mathews VP, Kuharik MA, Edwards MK, D'Amour PG, Azzerelli B, Dressen RG. Gd-DTPA-enhanced MR imaging of experimental bacterial meningitis: evaluation and comparison with CT. *AJNR* 1988;9:1045–1050