White matter changes in *B burgdorferi* encephalitis

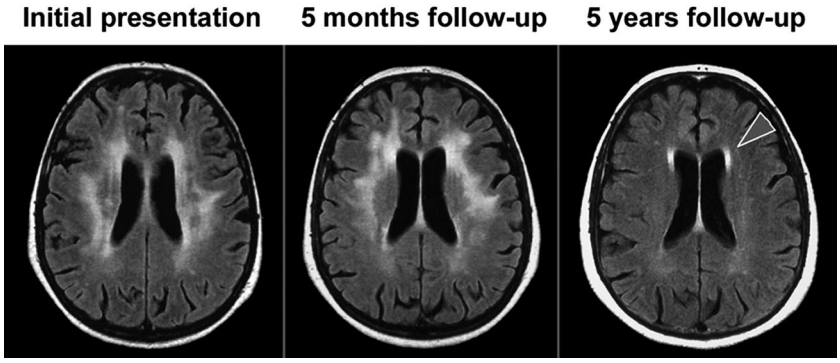
This case report by Steinbach et al. demonstrates that neuroborreliosis-associated white matter changes on MRI may resolve many years after successful antibiotic therapy.

see page 758

Borrelia burgdorferi, the brain and biologic plausibility

Commentary by John J. Halperin, MD, and Eric L. Logigian, MD

When is a single case study useful? In this issue of *Neurology*, Steinbach et al. describe a patient with severe *B burgdorferi* encephalomyelitis, detailing her clinical course, serial radiologic findings, and response to therapy. Although an extreme and unusual presentation of neuroborreliosis, this one case provides valuable insights into its pathogenesis and treatment.



How do we know this is neuroborreliosis? Despite the absence of erythema migrans or other classic elements, her rapidly progressive cognitive and gait disturbances, spasticity, and myoclonus were accompanied by inflammatory CSF, parenchymal white matter lesions on brain MRI (figure), positive Lyme serology, and intrathecal synthesis of anti-*B burgdorferi* antibody. She received 3 weeks of ceftriaxone and rapidly improved clinically and radiologically.

It bears emphasis that this patient's devastating encephalomyelitis resolved with a conventional course of antimicrobial therapy! What does this suggest about patients with less severe CNS Lyme disease? If the most damaging form of brain involvement is cured with simple treatment, it is unlikely that milder forms require more complex or prolonged regimens, a conclusion supported by recent studies of prolonged antibiotic treatment by Klempner et al.[1] and by Krupp et al.[2]

With respect to pathogenesis of CNS Lyme disease, in vitro studies have suggested possible roles both for direct CNS infection (with affinity of *B burgdorferi* for oligodendroglia) and for immune-mediated mechanisms. The fact that this patient improved dramatically within 3 weeks of treatment initiation, with gradual resolution of white matter lesions on MRI, argues for an essential role of active infection, rather than a purely para-infectious process. Given the difficulty of demonstrating spirochetes in neuroborreliosis by microbiologic or histologic techniques, substantial immune amplification is presumably necessary as well.

The lessons of this case report—the rare but dramatic clinical and radiologic presentation, the diagnostic role of intrathecal antibody production, the rapid response to straightforward treatment, and the hints as to pathogenesis—all make this an important addition to the neuroborreliosis literature.

References

1. Klempner M, Hu L, Evans J, et al. Two controlled trials of antibiotic treatment in patients with persistent symptoms and a history of Lyme disease. N Engl J Med 2001;345:85–92.
2. Krupp LB, Hyman LG, Grimson R, et al. Study and treatment of post Lyme disease (STOP-LD): a randomized double masked clinical trial. Neurology 2003;60:1923–1930.

see page 758

February 22 Highlight and Commentary: Borrelia burgdorferi, the brain and biologic plausibility

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