BORRELLA BURGDORFERI ANTIBODIES AND AMYOTROPHIC LATERAL SCLEROSIS

Several neurological involvement in Lyme disease usually takes the form of symptoms of meningitis, meningitis-like disease, radiculoneuritis, and/or cranial nerve involvement developing within months of primary infection with Borrelia burgdorferi. Recently, however, long-term sequelae have been described; these include a nonfatal/radioning neurological syndrome (simulating multiple sclerosis), focal encephalitis, and psychiatric disease. 

B. burgdorferi antibody titers were determined in sera from Wisconsin and Illinois patients. Antibody titers in serum from Wisconsin State Laboratory of Hygiene were significant (256 or greater). B. burgdorferi antibody titers were determined in sera from Wisconsin and Illinois patients. Antibody titers in serum from Wisconsin State Laboratory of Hygiene were significant (256 or greater).

A patient's query as to whether her amyotrophic lateral sclerosis (ALS) could be caused by Lyme disease led us to test a series of ALS patients for B burgdorferi antibodies in serum. Of 54 patients from Wisconsin and Illinois, antibodies at titers thought to be significant (256 or greater) were found in sera from Wisconsin State Laboratory of Hygiene to be significant (256 or greater).

Case 1—A 55-year-old woman had slurred speech 3 months after a generalised rash while on holiday in Oxford, Wisconsin, 6 months later she saw a neurologist for progressive weakness, dysarthria, and dyspnea. Arthropathy, fasciculations, and brisk reflexes were observed on examination. An electromyogram (EMG) demonstrated widespread denervation. Her B burgdorferi antibody titer in her serum was 512. She died of respiratory failure.

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years after onset. Her spinal fluid was never examined. Anterior horn cell loss and cortical spinal tract degeneration were demonstrated at necropsy. Conventional stains for central nervous system (CNS) involvement were negative.

Case 2—A 42-year-old woman who had been on holiday in northern Wisconsin had arm weakness followed by dysarthria and weakness in all four limbs. There was no history of rash or arthritis. Arthropathy and fasciculations were noted on examination. B burgdorferi antibody titers ranged from 1:64 to 1:4096 in this patient. Her CSF was normal. In view of the rise in titre while she was getting progressively worse intravenous ceftriaxone was administered. Her disease seemed to have stabilised.

Case 3—A 35-year-old man had left median nerve palsy and sensory disturbance of both arms in September, 1985. He had previously been on holiday in northern Wisconsin. These findings progressed into hand weakness, generalised muscle fasciculations, and weakness of the arms. The serum anti-B burgdorferi titre was 512. He died of respiratory failure. No necropsy was done.

The finding of cases of ALS with high titers of B burgdorferi antibodies should be viewed in the context of other CNS diseases as diverse as dementia and multiple sclerosis for which similar antibodies have been reported. This could mean that the cross-reactivity potential of B burgdorferi antigen is high—or that this spirochaete causes a wider diversity of common CNS syndromes than is generally recognised. Since there is no treatment for ALS and there is for chronic Lyme disease clinicians will ask if patients with ALS who have high-titre anti-Borrelia antibodies should be treated empirically with ceftriaxone, one of the antibiotics of choice for chronic B burgdorferi infections. At the least, it seems reasonable to find out if a patient with ALS does have B burgdorferi antibodies.

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