

Lyme Neuroborreliosis Mimics Stroke: A Case Report

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ABSTRACT. Zhang Y, Lafontant G, Bonner FJ Jr. Lyme neuroborreliosis mimics stroke: a case report. *Arch Phys Med Rehabil* 2000;81:519-21.

Lyme neuroborreliosis is diagnostically challenging because of its diverse manifestations. The well-documented neurologic spectrum includes lymphocytic meningitis, cranial neuropathy, and radiculoneuritis in the early disseminated stage; and peripheral neuropathy, chronic encephalomyelitis, and mild encephalopathy in the late persistent stage. This case report describes a 74-year-old man who developed progressive left hemiparesis and facial palsy. The patient was hospitalized to rule out a cerebral vascular accident. The diagnosis of Lyme borreliosis was established with serologic studies. The patient was treated with intravenous ceftriaxone and responded with rapid clinical and functional recovery. Lyme neuroborreliosis presenting as hemiparesis has rarely been reported. Prompt diagnosis and treatment appear to facilitate symptomatic relief and prevent persistent neurologic deficits.

Key Words: Lyme neuroborreliosis; Stroke; Rehabilitation.

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LYME DISEASE is a multisystem infectious syndrome of considerable public concern, both because of its prevalence and because of the availability of diagnostic and therapeutic measures to combat it. It is now the most common arthropod-borne infection in the United States, occurring in 48 of the 50 states at rates of up to 12,000 cases annually.¹ The causative organism, *Borrelia burgdorferi*, is a flagellated spirochete transmitted from small-mammal reservoirs to human beings through bites from infected ticks of the *Ixodes* species (commonly known as deer ticks).

It has been estimated that 10% to 40% of persons afflicted will develop neurologic abnormalities.²⁻⁴ The clinical manifestations of Lyme neuroborreliosis are markedly diverse and include lymphocytic meningitis, cranial neuropathy, and radiculoneuritis in the early disseminated stage, and peripheral neuropathy (mainly sensory), chronic encephalomyelitis, and mild encephalopathy in the late persistent stage.^{2,4-8}

Only rarely do the neurologic abnormalities of Lyme disease simulate progressive stroke.^{4,9,10} The case described here is one of Lyme neuroborreliosis presenting as hemiparesis.

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Submitted for publication January 15, 1999. Accepted in revised form September 23, 1999.

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit upon the authors or upon any organization with which the authors are associated.

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0003-9993/00/8104-5391\$3.00/0

doi:10.1053/mr.2000.4431

CASE REPORT

A 74-year-old Russian-born man had long-standing limited ability to comfortably express himself in English. He was right-handed, and described progressive left-sided weakness and paresthesiae involving the left upper extremity (UE) more than the left lower extremity (LE) for a week before he was hospitalized. The day before he was first seen, he noted left facial drooping. There was no limb pain, dysarthria, dysphagia, visual disturbance, headache, seizure, or sphincter dysfunction. On physical examination, the patient was afebrile, with a blood pressure of 140/70mmHg. There was an obvious left central facial palsy with slowed blink reflex but no tongue deviation. The muscle strength in the left UE was markedly decreased, 4/5 proximally and 4⁻/5 distally, whereas the muscle strength in the left LE was mildly decreased, 4⁺/5. There was no muscle tone abnormality. Deep tendon reflexes were symmetrically reduced. There were no objective sensory deficits or signs of cerebellar dysfunction.

His history disclosed long-standing hypertension, and there was a vague history of "vasculitis" of the right LE 8 years earlier that responded to a short course of corticosteroids. A retired house painter, he was independent in ambulation and activities of daily living (ADL) and enjoyed gardening and local traveling. On repeated, careful questioning, he had no recollection of tick bite or erythema migrans.

The patient was hospitalized to determine if he had had a cerebral vascular accident. Cranial computed tomography, carotid duplex ultrasonography, and magnetic resonance angiography confirmed that the carotid and cerebral vasculature were unremarkable. Magnetic resonance imaging of the brain revealed several small infarcts bilaterally in the parietal lobes, but no recent events. Echocardiography showed mild to moderate left atrial enlargement without thrombi. A complete blood count showed an elevated white blood cell count of 12,300/ μ L, with 58% neutrophils and 35% lymphocytes. Routine blood chemistries were normal.

The patient was treated initially with aspirin. He started an inpatient rehabilitation program on hospital day 3. At that time, muscle strength of the distal left UE was 2/5, and there was decreased sensation to light touch. The remainder of the neurologic examination remained unchanged. He needed set-up for feeding and minimum assistance for sit-to-stand transfer. He ambulated 50 feet with close supervision, and mild steppage gait on the left LE was noted. Routine neurologic rehabilitation techniques (such as range of motion, transferring, ADL and gait training, strengthening) were employed.

Because the patient exhibited atypical characteristics of stroke, the etiology was further investigated. An electrodiagnostic study of the left UE revealed prolonged left median and ulnar motor distal latencies, with reduced amplitudes and slowed conduction velocities, and a markedly delayed left ulnar sensory distal latency, with reduced amplitude. A left median sensory nerve action potential and an ulnar F-wave were unevokable. Needle electromyography did not reveal any abnormal spontaneous activities (ie, fibrillation potentials or positive sharp waves) in the left first dorsal interossei, abductor pollicis brevis, pronator teres, biceps brachii, and deltoid muscles. The motor unit action potentials were normal. The above findings were suggestive of a neuropathic process involving

both motor and sensory fibers. Other laboratory tests included erythrocyte sedimentation rate, 78mm/h; creatine kinase 75U/L; rapid plasma reagin, nonreactive; antinuclear antibody and antineutrophil cytoplasmic antibodies, negative.

A diagnosis of Lyme neuroborreliosis was confirmed thereafter by positive serologic findings on both enzyme-linked immunosorbent assay (ELISA) and Western blot. The patient refused lumbar puncture for cerebrospinal fluid analysis. He was started on an intravenous ceftriaxone regimen on hospital day 6. His clinical condition and functional status promptly improved. At discharge on hospital day 10, he needed close supervision for ADL and transfer, and ambulated 250 feet with improved gait. The patient had regained full left UE strength and functional independence at the end of the 4-week antibiotic therapy.

DISCUSSION

Lyme disease occurs in one of three stages, depending on organ system involvement and duration of infection: (1) early localized disease, which occurs in the first month after an infected tick bite; (2) disseminated disease, which occurs 1 to 4 months after a bite; and (3) late disease, which generally occurs 4 months to years after a bite.^{5,11} The hallmark of early localized disease is erythema migrans, which is seen in 50% to 70% of patients.¹¹ Although the history of tick bite and erythema migrans are valuable clues for early diagnosis, they are not always present, as in the described case.

Lyme neuroborreliosis can develop at any time within the disseminated or late stages and both the central and peripheral nervous systems can be involved.^{2,6,7} In the stage of disseminated disease stage, the classic triad includes lymphocytic meningitis, cranial neuropathy (most frequently the seventh cranial nerve), and motor or sensory radiculoneuritis. These manifestations occur singly or in combination. Less commonly, acute myelitis, encephalitis, mononeuritis multiplex, or ataxia may be the most prominent features of this stage. Rarely, there may be hydrocephalus, chorea, or a Guillain-Barré-like syndrome. In the late stages of the disease, peripheral neuropathy (predominantly sensory), chronic encephalomyelitis, and chronic encephalopathy with disturbances of memory, mood, and sleep have been described. Because of the diverse manifestations of Lyme neuroborreliosis, it should be included in the differential diagnosis of many neurologic disorders.

Lyme Disease and Stroke

Strokelike manifestations of Lyme neuroborreliosis have been reported,^{9,10,12} with clinical characteristics simulating transient ischemic attacks and cerebral infarction. Despite widespread uncertainty, some authors suggest that the strokelike manifestations are more likely in the 3rd stage.⁹

Whether Lyme neuroborreliosis causes stroke is equally inconclusive, although a few case reports imply that possibility.^{9,12-14} The proposed pathogenesis is that localized vasculitis may cause focal brain symptoms as part of a more generalized vasculopathy.^{10,12} Angiography has demonstrated changes in cerebral vessels compatible with vasculitis in a few cases of Lyme neuroborreliosis with strokelike symptoms.^{13,14}

Interestingly, our patient had "vasculitis" in the right lower extremity 8 years earlier. However, with no history of recurrence, any relationship to current Lyme neuroborreliosis was tenuous.

Diagnosis

A diagnosis of Lyme neuroborreliosis is based on characteristic clinical findings and appropriate laboratory testing. Knowl-

edge of the neurologic spectrum that the disease can present and a high level of suspicion are essential. However, even though Lyme neuroborreliosis may occasionally simulate stroke, screening all patients with stroke seems to be of little value. Hammers-Berggren and colleagues¹⁰ found only one patient with stroke-like symptoms caused by Lyme neuroborreliosis in a *B burgdorferi*-endemic area.

The Centers for Disease Control (CDC) recommends that serologic testing for Lyme disease should be considered positive only when both ELISA and Western blot analysis are positive.¹⁵ This two-test approach minimizes the possibility of false-positive tests. If central nervous system involvement is suspected, cerebrospinal fluid can be assessed for intrathecal antibody production, or demonstration of organisms by culture, histopathology, or polymerase chain reaction (PCR). Electrodiagnostic studies are important in investigating peripheral nervous system involvement. Polyneuropathy is primarily axonal, but demyelination has also been reported.^{2,16,17} It may be too early to detect any denervation changes in our patient. A more extensive follow-up study might be warranted. Although the Lyme-associated polyneuropathy slowly improves without treatment, its course is abbreviated by antibiotic therapy.^{2,17}

Medical Management

Lyme neuroborreliosis can be treated effectively with antibiotics. Commonly used agents include ceftriaxone, cefotaxime, and penicillin. They are usually administered intravenously, for 2 to 4 weeks.¹² Neurologic symptoms resolve with antibiotic regimens in most patients,² including our case. Accurate and prompt diagnosis is important because effective antibiotic therapy may prevent the persistence of neurologic deficits.⁹

Rehabilitation Management

At present, neurologic deficits from Lyme neuroborreliosis are managed in rehabilitation in a manner similar to management of the same deficits resulting from other causes. However, two questions arise from this case report that may have future ramifications: (1) How can rehabilitation professionals contribute to the prompt diagnosis of the disease?; and (2) What role can rehabilitation techniques play in improving the functional outcomes in these patients?

With the trend toward early transfer of patients with stroke and other neurologic disorders to rehabilitation units, physiatrists will be accepting many patients without complete workups and without definitive etiology established for their patients' problems. Maintaining a high level of suspicion suggests that we include Lyme neuroborreliosis as one of the differential diagnoses of patients with somewhat atypical stroke presentations. Appropriate serologic studies should be ordered in the selected cases.

Although rehabilitative techniques and strategies now used have been adapted from those commonly used in other neurologic disorders, the efficacy of these methods in Lyme neuroborreliosis remains to be evaluated. Compounding factors, such as the natural history of the disease and the effects of antibiotic therapy, must be considered. Further, for those manifestations of Lyme neuroborreliosis such as radiculoneuritis that may have a protracted course of recovery,^{2,7} further investigation is needed to develop new, more effective therapeutic methods.

Acknowledgment: We thank Dr. Lenore R. Zohman for critically reviewing this manuscript.

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