

Could Lyme disease be another infection associated with the onset of Guillain-Barre Syndrome?

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However, in their case study *Clinical association: Lyme disease and Guillain-Barre Syndrome*, the authors highlight "*Borrelia burgdorferi* as an important antecedent infection associated with the development of GBS," [1] and describe a 31-year-old man diagnosed with both Lyme disease and GBS. The case raises the question: Could Lyme disease be an underrecognized infectious disease triggering or contributing to the onset of Guillain-Barre Syndrome?

Eight months prior to admission, the man reported having a dime-sized lesion on his left arm. He later developed progressive numbness and weakness in both his hands and feet, along with areflexia. The numbness and burning in his feet progressed to his bilateral upper extremities, and he developed new weakness in all extremities. He also had blurry vision with decreased sensation and numbness in his tongue, and a right temporal headache that worsened with light and sound.

The exam revealed "decreased sensation to pinprick with a distal to proximal gradient up to proximal thigh," [according to Patel from SUNY Upstate Medical University](#), Syracuse, New York. "Other significant findings were 4/5 weakness in all extremities along with areflexia in biceps, triceps, patellar and achilles."

Diagnostic testing revealed a mildly high white count of 12,800 WBC/?L, with mildly elevated ESR of 17 mm/h, a spinal tap revealing an elevated protein of 190 mg/dL, and pleocytosis of 10mm³. "An electromyography (EMG) was done showing absent F waves in bilateral tibial and peroneal motor responses consistent evidence of acute, acquired polyradiculoneuropathy with active denervation," according to Patel. "A clinical picture with ascending neuropathy and EMG findings of isolated absence of F waves favor a diagnosis of GBS."

A Lyme disease diagnosis was confirmed with immunoblots positive for IgM p23 and p41, as well as IgG p18, p23, p30, p39 and p41. Elevated protein and pleocytosis have been described in neurologic Lyme disease. [2]

The EMG findings, clinical picture and laboratory results were compatible with a diagnosis of Lyme disease and GBS. And the man was prescribed a combination of intravenous immunoglobulin 0.4 g/kg daily, plasma exchange therapy, and intravenous ceftriaxone 2 g. daily. Within 7 days, his symptoms had resolved.

Guillain-Barre Syndrome, the authors point out, "is an immune-mediated polyneuropathy characterized by acute, generalized, ascending peripheral neuropathic weakness with demyelination being the main electrophysiological and pathological feature." [1] Lyme disease is also associated with demyelination [2]

and immune-mediated.

“The actual mechanism between the relationship of GBS and Lyme disease remains unclear,” explains Patel. “However, there is evidence of immune responses associated with tick-borne pathogens. It is possible that these immune complexes in some individuals result in the development of antiganglioside antibodies causing GBS.”

References:

1. Patel K, Shah S, Subedi D. Clinical association: Lyme disease and Guillain-Barre syndrome. Am J Emerg Med. 2017.
2. Logigian EL, Kaplan RF, Steere AC. Chronic neurologic manifestations of Lyme disease. N Engl J Med. 1990;323(21):1438-1444.

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