# Could Lyme disease have led to a spinal cord lesion?

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In their article "Case report: Dueling etiologies: Longitudinally extensive spinal cord lesion mimicking spinal cord infarct with simultaneous positive Lyme serology and amphiphysin antibody,"[1] Kalaszi and colleagues discuss the case of a patient who presented with a Bull's-eye rash 6 months prior to his illness and whose testing was suggestive of Lyme disease as a possible cause of the spinal cord lesion.

The man "presented with chest pain, followed by right lower limb weakness, preceded by 2 weeks of constipation and voiding dysfunction," wrote the authors.

He developed bilateral severe flaccid paresis of the lower limbs, bilaterally absent knee and ankle reflexes with upgoing plantar responses, patchy impairment of the sensory exam, urinary retention and reduced anal tone.

The spinal MRI showed extensive disease. "Magnetic resonance imaging (MRI) of the brain and the whole spine with contrast revealed an abnormal, increased T2 signal in the anterior aspect of the spinal cord beginning at the T4 level and extending to the conus without associated edema or contrast enhancement," wrote the authors.

The doctors considered several possibilities. "The imaging findings were consistent with a longitudinally extensive transverse myelitis (LETM) of potential autoimmune, paraneoplastic, or infective etiology."

Symptoms of spinal cord infarction depend on the effected spinal cord region and often appear suddenly. "Around 70% of patients report acute back, chest, neck, or limb pain before the neurological deficit," the authors point out.

**The patient reported having a Bull's-eye rash 6 months prior to the onset of symptoms. In addition, he complained of autonomic dysfunction 2 weeks before his illness.**

Finally, his blood and spinal tap findings were remarkable for the presence of intrathecal immunoglobulin G (IgG) against two specific Borrelia antigens, p21 and VlsE. He was treated with IV ceftriaxone.

Testing also revealed the presence of the amphiphysin antibody. (Transverse myelitis secondary to Lyme disease has been described.)

Amphiphysin antibodies are seen in paraneoplastic stiff-person syndrome and in a variety of neurological manifestations. "The most common neurological manifestation was limbic encephalitis (n=10), followed by dysautonomia (n=9), and cerebellar dysfunction (n=6)," wrote Moon and colleagues.[2]

Testing, including a PET scan, did not reveal a malignancy. There was no evidence of an occult malignancy.

The patient responded to steroids and 3 weeks of intravenous ceftriaxone.

“After 6 weeks, he was able to walk unaided with a stick, and bowel and bladder functions were fully recovered,” wrote the authors.

**Related Articles:**

Podcast: Transverse myelitis and Lyme disease

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Six cases of neuroinvasive Lyme disease

**References:**

1. Kalaszi M, Donlon E, Ahmad MW, Mohamed AS, Boers P. Case report: Dueling etiologies: Longitudinally extensive spinal cord lesion mimicking spinal cord infarct with simultaneous positive Lyme serology and amphiphysin antibody. Front Neurol. 2022;13:905283. doi:10.3389/fneur.2022.905283
2. Moon J, Lee ST, Shin JW, et al. Non-stiff anti-amphiphysin syndrome: clinical manifestations and outcome after immunotherapy. J Neuroimmunol. Sep 15 2014;274(1-2):209-14. doi:10.1016/j.jneuroim.2014.07.011

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