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A RARE PRESENTATION OF LYME NEUROBORRELIOSIS: MULTIFOCAL CERVICAL AND THORACIC TRANSVERSE MYELITIS-A CASE REPORT

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ABSTRACT

Background: Spinal cord involvement in Lyme disease is an uncommon manifestation of neuroborreliosis, with myelitis representing a rare clinical entity. Multifocal transverse, non-contiguous spinal cord involvement remains particularly rare and is insufficiently characterized in the existing literature.

Case report: We report the case of a 40-year-old previously healthy woman who presented with rapidly progressive paraparesis, ascending sensory deficits, and bladder and bowel dysfunction consistent with acute-subacute inflammatory myelopathy. Spinal magnetic resonance imaging (MRI) demonstrated multifocal non-contiguous inflammatory lesions involving the cervical and thoracic spinal cord. Extensive microbiological and serological investigations excluded alternative infectious aetiologies. Brain MRI showed no evidence of demyelinating disease. Despite clinically significant spinal cord inflammation, cerebrospinal fluid (CSF) analysis revealed neither pleocytosis nor elevated protein concentration. However, Borrelia-specific IgM antibodies were detected in both serum and CSF, and a positive Borrelia-specific antibody index (AI 3.07) confirmed intrathecal anti-Borrelia antibody synthesis, establishing the diagnosis of Lyme neuroborreliosis-associated myelitis. The patient subsequently demonstrated marked neurological improvement following targeted treatment with intravenous ceftriaxone combined with corticosteroids and supportive therapy.

Conclusion: This case significantly expands the recognized spectrum of spinal cord involvement in Lyme disease–associated neuroborreliosis by delineating an extremely rare multifocal presentation, thereby emphasizing the critical importance of recognizing atypical manifestations in the diagnostic evaluation of inflammatory myelopathies.

KEYWORDS

Transverse myelitis, Lyme disease, neuroborreliosis, multifocal spinal cord lesions

LEARNING POINTS

- Multifocal non-contiguous transverse myelitis is an unusual manifestation of Lyme neuroborreliosis.
- Lyme-associated inflammatory myelitis may occur even in the absence of classical cerebrospinal fluid abnormalities or documented tick exposure.
- Early recognition and antimicrobial treatment improve neurological recovery.

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INTRODUCTION

Lyme disease (borreliosis) is one of the most common vector-borne diseases worldwide which is caused by Borrelia burgdorferi sensu lato. Lyme disease clinical manifestations may be divided into three stages: (i) early localized stage (erythema migrans in the tick bite site), (ii) early disseminated stage (multiple erythema migrans lesions, borrelial lymphocytoma, Lyme neuroborreliosis, carditis, or arthritis) and (iii) the late disseminated stage (acrodermatitis chronica atrophicans, Lyme arthritis and neurological symptoms)[1]. The most common neurologic complications of Lyme disease are cranial neuritis, meningitis, and radiculoneuritis/mononeuropathy multiplex[2]. Myelopathy encompasses a broad spectrum of pathological processes affecting the spinal cord, including degenerative, vascular, neoplastic, and inflammatory aetiologies, of which myelitis constitutes the inflammatory subset. Myelitis represents an exceptionally rare manifestation of Lyme disease, with fewer than 60 cases reported worldwide[3]. Spinal cord involvement in neuroborreliosis demonstrates considerable clinical and radiological heterogeneity, ranging from focal and segmental to longitudinally extensive patterns; however, multifocal transverse involvement remains distinctly uncommon[4]. In this context, we present an extremely rare case of multifocal transverse myelitis associated with Lyme disease, thereby contributing to the limited and evolving body of evidence on this atypical presentation. This is the first reported case of Lyme neuroborreliosis-associated transverse myelitis in Kosovo.

CASE DESCRIPTION

A 40-year-old previously healthy woman presented to the Neurology Department at the University Clinical Centre of Kosovo with a rapidly progressive myelopathy characterized by paraparesis, ascending sensory deficits to the umbilical level, and sphincter dysfunction with loss of bladder and bowel control. Neurological symptoms began two days before admission with right lower-limb monoparesis and associated hypoesthesia, followed by rapid progression to bilateral lower-extremity involvement. A flu-like prodromal illness was reported several days before symptom onset. No documented history of tick bite or erythema migrans was identified.

On admission, the patient was alert, fully oriented, afebrile, and hemodynamically stable. Neurological examination demonstrated a well-defined sensory level at umbilicus, bilateral hypotonia and hyporeflexia in the lower extremities, and bilateral extensor plantar responses. No involuntary movements or meningeal signs were present. Initial investigations for infectious aetiologies did not identify a causative pathogen. Serological investigations for brucellosis (including Wright and rapid agglutination assays) were negative. Additional testing was negative for toxoplasmosis and herpes simplex virus 2 (IgG and IgM), while cytomegalovirus and rubella serologies were consistent with remote prior exposure rather than active infection. Magnetic resonance imaging (MRI) of the spinal cord obtained on the first hospital day demonstrated a central T2-weighted hyperintense lesion at the C4–C5 level and an additional posterior peripheral T2 hyperintensity at the T2–T3 level *(Fig. 1)*. The distribution and signal characteristics of these lesions were considered consistent with inflammatory myelopathy in keeping with transverse myelitis and were concordant with the patient's clinical presentation and sensory level. Brain MRI showed no pathological abnormalities, including no evidence suggestive of demyelinating disease. In the absence of an established aetiology at presentation, the patient was empirically treated for presumed inflammatory myelitis with high-dose corticosteroids, empirical broad-spectrum antimicrobial therapy, and anticoagulant prophylaxis in accordance with standard therapeutic approaches, alongside standard supportive management. After 19 days, the patient was discharged in stable clinical condition with continuation of oral corticosteroids and supportive therapy.

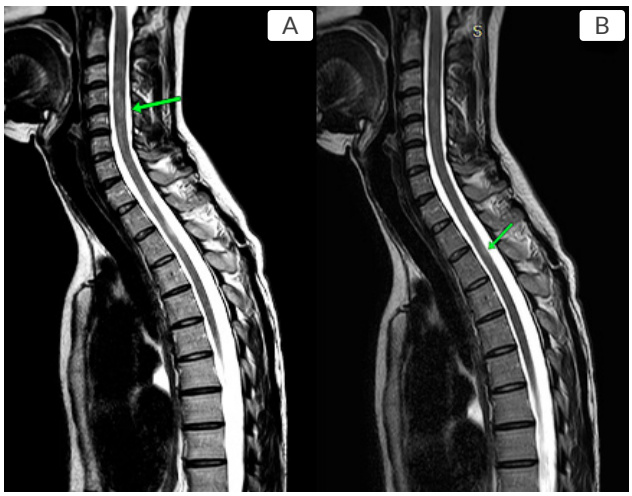


Figure 1. *Sagittal T2-weighted magnetic resonance imaging of the cervicothoracic spinal cord demonstrating two non-contiguous intramedullary T2-hyperintense lesions: A) a centrally located lesion at C4–C5 (arrow) and B) a smaller posterior peripheral lesion at T2–T3. The distribution and morphology of the lesions were considered most consistent with inflammatory myelopathy in the appropriate clinicoradiological context.*

Approximately one week after discharge, extended serological evaluation demonstrated serum anti-Borrelia burgdorferi IgM antibodies confirmed by Western blot, while IgG antibodies remained negative. In light of these findings, the patient was referred to the Infectious Diseases Department for further evaluation. At re-admission, the patient continued to report persistent lower-limb sensory deficits, more pronounced on the left side, as well as ongoing urinary dysfunction. Routine laboratory studies were largely unremarkable aside from mild leucocytosis (white blood cell count 14.0 $\times 10^9$/l) with neutrophilia. Blood cultures were negative. Cerebrospinal fluid analysis demonstrated normal glucose concentration (4.06 mmol/l; reference range 2.2–4.4 mmol/l) and protein concentration

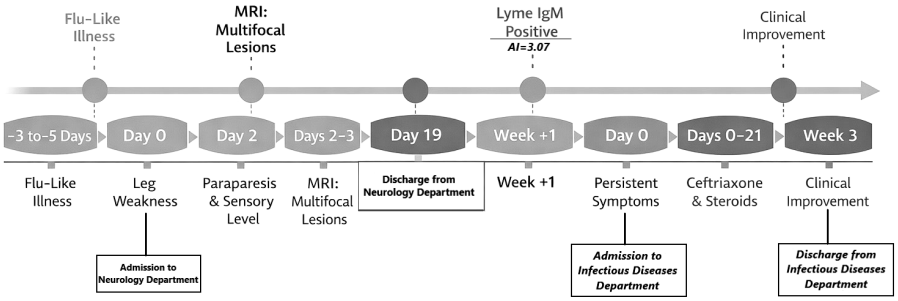


Figure 2. *Graphical timeline summarizing prodrome, symptom onset, investigations, interdepartmental transfer and therapeutic interventions.*
Abbreviation: MRI, magnetic resonance imaging.

(0.2 g/l; reference range 0.15–0.45 g/l), without pleocytosis (0 cells/mm^3; reference range 0–5 cells/mm^3). Broad microbiological evaluation of the cerebrospinal fluid (CSF), including multiplex polymerase chain reaction, GeneXpert MTB/RIF assay, and conventional culture, likewise yielded negative results. Notably, Borrelia-specific IgM antibodies were detected in both serum and CSF, with corresponding reactivity values of 80 and 1.229, respectively, while IgG antibodies remained negative. Integration of pathogen-specific antibody reactivity with total IgM concentrations in serum and CSF yielded a positive Borrelia-specific antibody index (AI 3.07), confirming intrathecal anti-Borrelia antibody synthesis. Targeted treatment with intravenous ceftriaxone was initiated in combination with corticosteroids and supportive therapy, resulting in marked and progressive neurological improvement. After three weeks, the patient was discharged in stable clinical condition on oral doxycycline and corticosteroid taper, with referral for structured neurorehabilitation and physiotherapy. Following discharge from the Infectious Diseases Department, the patient continued the same therapeutic regimen orally at half dosage for an additional two weeks. Subsequent follow-up evaluations demonstrated sustained clinical and neurological improvement, with progressive recovery of motor deficits and sensory disturbances. A graphic presentation of the course is provided in *Figure 2*.

DISCUSSION

Multifocal transverse involvement of the spinal cord is an uncommon radiological pattern in inflammatory myelopathies and has only rarely been described in association with Lyme disease[4]. Reported spinal cord involvement in neuroborreliosis has predominantly demonstrated focal or longitudinally extensive patterns[5,6]. In contrast, the presence of non-contiguous cervical and thoracic lesions in our patient highlights an unusual multifocal transverse distribution, further expanding the recognized spectrum of spinal cord involvement in Lyme-associated disease.

Clinically, the patient developed a rapidly progressive inflammatory myelopathy characterized by paraparesis, a clearly defined thoracic sensory level, and sphincter dysfunction, with strong clinicoradiological concordance between neurological findings and multifocal MRI abnormalities. The preceding flu-like prodromal illness may reflect early systemic immune activation preceding overt neurological manifestation, as previously described in infection-associated inflammatory myelitides[7,8].

An extensive diagnostic workup failed to identify an alternative infectious aetiology. Comprehensive microbiological investigations yielded negative results aside from Borrelia burgdorferi seroreactivity. Brain MRI demonstrated no demyelinating lesions or other abnormalities suggestive of multiple sclerosis. A particularly noteworthy feature of this case was the atypical CSF profile. Despite clinically and radiologically significant spinal cord inflammation, CSF analysis revealed neither pleocytosis nor elevated protein concentration. This finding may partially be explained by the delayed lumbar puncture performed after more than three weeks of high-dose corticosteroid therapy, which may have attenuated the underlying neuroinflammatory response and contributed to partial restoration of blood–brain barrier integrity, thereby normalizing conventional CSF inflammatory parameters by the time of analysis. Nevertheless, Borrelia-specific IgM antibodies remained detectable in the CSF, and a positive Borrelia-specific antibody index (AI 3.07) confirmed intrathecal anti-Borrelia antibody synthesis, substantially strengthening the diagnosis of Lyme neuroborreliosis despite otherwise unremarkable routine CSF findings. At the same time, the possibility that this case represents an intrinsically atypical form of Lyme neuroborreliosis with minimal conventional CSF inflammatory abnormalities should also be considered. While pleocytosis and elevated CSF protein are typical findings in neuroborreliosis, rare atypical near-normal CSF profiles have occasionally been reported[9].

Although no documented tick bite or erythema migrans was identified in our patient, Lyme borreliosis remained epidemiologically credible given the documented presence of *Ixodes ricinus* ticks and circulating Borrelia species in Kosovo[10]. Importantly, absence of recognized tick exposure or classical dermatological manifestations does not exclude borreliosis, as these findings may frequently remain unrecognized in clinical practice[11].

The coexistence of multifocal non-contiguous spinal cord lesions, transverse inflammatory involvement, and essentially unremarkable CSF inflammatory parameters therefore represents an exceptionally uncommon and diagnostically challenging manifestation within the spectrum of Lyme neuroborreliosis. Importantly, the

patient subsequently demonstrated marked neurological improvement following targeted antimicrobial therapy with intravenous ceftriaxone and corticosteroids, a therapeutic response consistent with previously described outcomes in Lyme-associated neuroinflammatory disease.

This case therefore not only illustrates the diagnostic complexity associated with atypical Lyme-associated myelitis but also contributes to the evolving literature by documenting a rare phenotype of multifocal, non-contiguous transverse spinal cord involvement in neuroborreliosis.

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