

Case report

Acute transverse myelitis – A rare clinical manifestation of Lyme neuroborreliosis

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ABSTRACT

Acute transverse myelitis (ATM) is a rare, potentially devastating neurological syndrome that has variety of causes, infectious being one of them. Lyme disease (LD) is the most common vector borne zoonosis in the United States (U.S.). While neurologic complications of LD are common, acute transverse myelitis is an exceedingly rare complication. We present a case of a previously healthy 25-year-old man who presented with secondary erythema migrans, aseptic meningitis and clinical features of transverse myelitis including bilateral lower extremity motor and sensory deficits manifesting as weakness and numbness, urinary retention and constipation. Despite negative serum antibodies against *Borrelia burgdorferi*, cerebrospinal fluid (CSF) was positive for *Borrelia burgdorferi* PCR. Following treatment with methylprednisolone and ceftriaxone, he attained complete recovery apart from neurogenic bladder necessitating intermittent self-catheterization. We report rare manifestation of a common disease and emphasize the importance of considering LD in the differential diagnosis of acute transverse myelitis, particularly in residents of endemic areas.

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Introduction

ATM is a rare neurological syndrome characterized by a combination of varying degrees of bilateral motor, sensory, and autonomic dysfunction, that progresses over hours to days, exhibits nadir within days to weeks, and leaves one third of patients with severe neurological disability [1,2]. It is typically classified as either idiopathic or disease-associated transverse myelitis. Disease-associated transverse myelitis occurs in the context of systemic inflammatory diseases (sarcoidosis, Bechet's disease, systemic lupus erythematosus, Sjögren syndrome and ankylosing spondylitis), infection (human immunodeficiency virus, enteroviruses, West Nile virus, Herpes simplex viruses, cytomegalovirus, mycoplasma, Lyme borreliosis, tuberculosis), and central nervous system (CNS) multifocal diseases (multiple

sclerosis, neuromyelitis optica, and acute disseminated encephalomyelitis). The latter group is excluded from ATM according to some authors [2] due to being part of well-established CNS syndromes with specific treatment options. Idiopathic ATM is considered to be an autoimmune process triggered by previous infection and/or vaccination, but the diagnosis of idiopathic ATM is made only after extensive search for an underlying condition and etiology remains elusive.

LD is a multisystem, multistage disease that is the most common tick-borne infection in the U.S. [3,4]. Most human infections are caused by 3 members of *Borrelia burgdorferi* sensu lato (Bbsl) complex - *B.afzelii* and *B.garinii* in Europe and Asia and *B. burgdorferi* in North America [5] *B.mayonii*, a new member of the Bbsl complex was recently identified in the upper midwestern U.S. as an uncommon cause of LD with unusually high spirochetemia levels [6]. LD is transmitted to humans by the bite of infected *Ixodes scapularis* ticks. Early localized stage of LD is confined to the skin in the form of erythema migrans. Left untreated, LD progresses to early disseminated and late stage, which may affect joints, heart and/or the nervous system. Among neurological manifestations of

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LD, peripheral nerve palsy, aseptic meningitis and painful meningoradiculitis are the most commonly encountered [4,7]. ATM is an extremely rare neurologic complication of LD [8–13].

Case presentation

A previously healthy 25-year-old man presented with inability to urinate and frequent falls associated with bilateral lower extremity weakness and numbness. His symptoms began approximately two weeks prior to presentation when he developed a headache and noticed a red circumferential rash approximately 10 cm in diameter on his left thigh. The headache was frontal in location, mild, intermittent and did not increase in severity over the next two weeks. The rash was red, circular, non-pruritic and disappeared approximately a week before his admission to hospital. At around the same time as the rash disappeared, he developed neck stiffness and subjective fevers with chills but did not have photophobia, nausea or vomiting. Five days prior to admission, he developed urinary retention as well as progressive numbness and weakness in his lower extremities. Sensory deficit initially manifested as left foot numbness and progressed bilaterally in ascending, band-like fashion up to the upper thorax right below the nipple line anteriorly and below the shoulder blades posteriorly. Additionally, he also developed constipation and had been unable to defecate for a few days prior to admission. As a result of bilateral leg weakness and gait instability, he sustained a few falls without major injuries. He denied any joint pain or swelling.

The patient did not report any prior illnesses and was not taking any medication or supplements. He lived in Wisconsin, in a house with very close proximity to the woods and had significant outdoor exposure in the form of hiking, camping and fishing. He had two healthy dogs and denied any dog bites. He did not report being bitten by ticks in the recent past. Family history was negative for autoimmune or neurological diseases. A social drinker, he smoked cigarettes and rarely smoked marijuana (THC). The patient's vaccinations were up to date but he had not received any vaccination within the last year. He was in a monogamous relationship with his girlfriend and denied high risk sexual practice. He was born and raised in Wisconsin and lived there all his life with no travel outside Wisconsin over the last year.

On admission, the patient was afebrile and had stable vitals with blood pressure of 134/75 mmHg, a regular heart rate at 62 beats per minute and respiratory rate of 16 breaths per minute. His oxygenation was 98 percent on ambient air. Skin exam showed few erythematous lesions on the left lateral surface of neck that were painless, non-itchy and circumferential, 5 cm in diameter and were in a resolving stage. There was no cervical lymphadenopathy. Neck veins were flat. Lungs were clear bilaterally and abdomen was non-tender with decreased bowel sounds. Neurological exam showed pupils that were equal, round and reactive bilaterally. Extra ocular movements were intact through all planes. Facial muscle strength was full and symmetric bilaterally. Tongue protrusion was midline. Speech pattern was appropriate without dysarthria. Motor testing demonstrated no rigidity or spasticity in the upper limbs, however, 1+ spasticity was noted at both knees. While upper extremity strength was normal, there was symmetrical, pyramidal pattern of weakness noted in both lower extremities at 4 out of 5, along with diffuse hyperreflexia that was more pronounced in the lower limbs. Plantar reflex elicited an abnormal Babinski's sign on the left and a normal response on the right. There was diminished sensation to light touch circumferentially affecting bilateral lower extremities as well as the torso up to roughly the T5 dermatomal level. There was no limb dysmetria or ataxia. Gait evaluation was deferred secondary to fall risk.

Laboratory data on admission showed normal complete blood cell count (CBC), however white blood cell (WBC) differential showed mild neutrophilia ($7.27 \times 10^9/L$) and monocytosis ($1.3 \times 10^9/L$). Liver and renal functions were normal. C-reactive protein (CRP) was elevated at 7.6 mg/dL. Blood alcohol level was undetectable. Serum vitamin B12 and folic acid levels were within normal range. Lactic acid was 1.5 mmol / L. Urinalysis showed no pyuria and drug screen was positive only to THC. Blood cultures were drawn on admission and remained negative after 5 days of incubation. Magnetic resonance imaging (MRI) of the brain with and without contrast was negative for space occupying lesion, demyelinating lesion, hemorrhagic or ischemic infarct. However, MRI of the cervical and thoracic spine revealed T2 signal hyperintensity in the central spinal cord gray matter at C5, C6 and T3 to T9 levels suggestive of myelitis (Figs. 1 and 2).

CSF findings were highly suggestive of an inflammatory process. CSF cell count was 316 with lymphocytic predominance (76 percent). CSF protein was slightly elevated to 54 mg/dL and glucose was normal at 61 mg/dL. CSF gram stain showed many white blood cell (WBC) but no organisms. CSF cultures remained negative. *Borrelia burgdorferi* serum antibodies were negative but PCR from CSF returned positive. The Lyme disease molecular detection testing in CSF was done at Mayo Medical Laboratories (MML) wherein DNA was extracted from the CSF specimen using MagNA Pure Instrument (Roche) and tested for *B.burgdorferi* sensu lato genogroup by a real-time PCR assay that targeted the plasminogen-binding protein (OppA2) gene by using hybridization probes [6].

Initially, empiric treatment was started with ceftriaxone 2 g intravenously (IV) daily and acyclovir 10 mg/kg IV every 8 h. He also received methylprednisolone 1 g IV daily for 3 days. Acyclovir was discontinued on day 2 after CSF *B. burgdorferi* PCR returned



Fig. 1. This is T2 weighted sagittal image demonstrating intramedullary T2 signal abnormality over a long segment of the thoracic spinal cord (red arrows) without significant cord expansion.

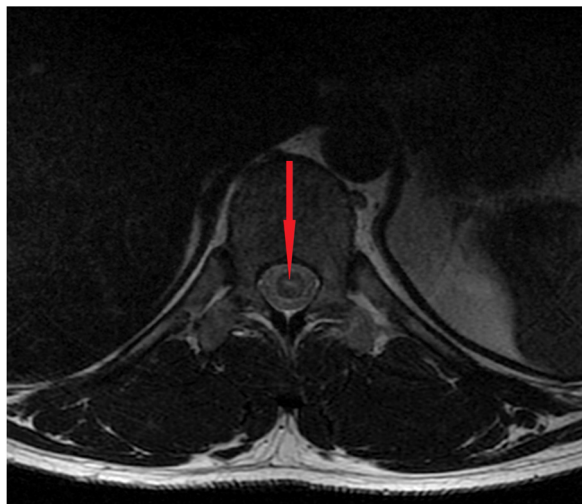


Fig. 2. This is an axial T2 weighted image which shows non-expansile T2 signal in the center of the thoracic spinal cord (red arrow).

positive. Patient received IV ceftriaxone for 28 days. He showed gradual improvement in gait, motor and sensory functions of his lower extremities along with resolution of neurogenic bowel. However, he continues to need intermittent self-catheterization for neurogenic bladder. Convalescent testing for *B.burgdorferi* serology was not performed because it would not alter the management and patient had already improved on treatment.

Discussion

In about 64 percent of cases, ATM is classified as idiopathic, while 36 percent of cases have a clear etiology categorizing these patients as disease-associated ATM [14]. Disease-associated ATM can be classified into three groups: systemic inflammatory disease, infectious diseases, and multifocal CNS disease. Multifocal CNS diseases are well established diagnostic categories and ATM is frequently a component of a broader clinical syndrome with well-established treatment protocols. Hence, it is important to obtain MRI of the brain in patients with ATM - if brain lesions are present, then the chance of patients meeting clinical criteria for multiple sclerosis (MS) is 84%, whereas it decreases significantly to 11% if the brain MRI does not show lesions [15].

Infectious myelitis is identified in about 12% of patients and can be due to viral, bacterial, fungal and/or parasitic infections [16–18]. Despite being a rare etiology of transverse myelitis, infection needs to be considered among the top differential diagnosis, given the importance of timely recognition and initiation of therapy. Our patient presented with typical symptoms and signs of ATM, had normal MRI brain, and he was found to have CSF PCR positive for *B. burgdorferi*, which classifies his clinical presentation as disease-associated ATM with infectious etiology.

Neurologic manifestations of LD are termed Lyme neuroborreliosis (LNB) and may occur in early and late disseminated stages of disease. LNB occurs more often during the early disseminated stage of LD (10–15% of cases) [19]. Aseptic meningitis, peripheral nerve palsy (cranial nerve VII being most common) and radicular neuropathy are the most frequent clinical presentations of early LNB [19,20]. Bannwarth syndrome (painful meningo-radicularitis), the most common manifestation of early LNB in Europe, is uncommon in North America. However, a recent report of a cluster of 5 patients with Bannwarth syndrome in the Midwest U.S. suggests that this syndrome may be under-recognized in North America [5]. Late LNB is defined as persistence of neurologic symptoms for more than six months and can occur months to years

following initial infection. It can manifest as progressive encephalitis or encephalomyelitis, cerebral vasculitis or chronic meningitis [20,21]. While acute transverse myelitis is an infrequently encountered complication of LD, it can have devastating consequences if not recognized and treated early.

Our patient presented with typical constellation of symptoms and signs of early disseminated stage of LD including multiple secondary erythema migrans and aseptic meningitis. However, his clinical presentation was consistent with spinal cord involvement which was unusual. We tested him for the most common infectious diseases, autoimmune and paraneoplastic diseases as described above (see Table 1). By ruling out other more common etiologies of ATM and by confirming the presence of *B. burgdorferi* in CSF by molecular testing, we confidently concluded that ATM was, indeed, a manifestation of LNB. Additionally, our diagnosis was confirmed by demonstrating significant improvement in neurologic deficit following four weeks of intravenous therapy with ceftriaxone.

We performed a literature search on PubMed database using the following words alone or in combination: Lyme disease, acute transverse myelitis, neuroborreliosis, myelitis, neurologic manifestation of Lyme disease. We excluded pediatric cases and articles published in languages other than English. Our search yielded 6 cases of LNB related ATM (Table 2). Interestingly, only one case was reported from the U.S. and the rest were from Europe. One explanation for the higher rate of LNB in Europe may be due to the higher neuro-tropism of the agents of European borreliosis, *B. afzelii* and *B. garinii* when compared to *B. burgdorferi*. The patients in the published reports were younger (median age 40.5 years, range 19–50) generally healthy and without significant gender preference. Similar to our patient, two out of five reported cases (40%) also exhibited significant autonomic dysfunction, manifesting as urinary or bowel incontinence or retention. Interestingly, this observation is in contrast to findings from a retrospective study from Germany [22] in which none of the five patients who had Lyme myelitis exhibited autonomic dysfunction. As in these five cases, our patient too showed significant clinical improvement after appropriate treatment.

Table 1

Summary of negative diagnostic tests that were done in our patient to rule out autoimmune, inflammatory, paraneoplastic and other infectious etiologies of acute transverse myelitis.

Tests done on serum	Tests done on CSF
<i>B.burgdorferi</i> Ab	HSV-1 PCR
<i>A.phagocytophilum</i> Ab	HSV-2 PCR
<i>E.chaffeensis</i> Ab	VZV PCR
<i>B.dermatitidis</i> Ab	EBV PCR
<i>M. pneumoniae</i> Ab	CMV PCR
<i>C.neoformans</i> Ag	Enterovirus PCR
Quantiferon TB GOLD	Powassan virus PCR
RPR	NMO Ab
West nile virus PCR	AQP-4 Ab
CMV Ab	
EBV Ab	
HIV Ab	
HBV Ab	
HCV Ab	
ANA	
RF	
SSA Ab	
SSB Ab	

Ag- antigen, Ab- antibody, ANA- Anti-nuclear antibody, AQP-4- Aquaporin-4, CMV- Cytomegalovirus, EBV- Epstein Barr virus, HBV- Hepatitis B virus, HCV- Hepatitis C virus, HIV- Human immunodeficiency virus, HSV- Herpes simplex virus, NMO- Neuromyelitis optica, PCR -Polymerase chain reaction, RPR - Rapid plasma reagin, RF- Rheumatoid factor, SSA- Anti-Sjogren's syndrome A, SSB- Anti-Sjogren's syndrome B, VZV- Varicella zoster virus.

Table 2 This table summarize all adult cases of neuroborreliosis related transverse myelitis reported in English literature. It includes patients' demographics, country of origin, most common symptoms and signs as well as treatment and outcome of the diseases.

Case (Ref)	Year	Age (y/o)	Sex	Country	Systemic illnesses	Time between symptom onset and diagnosis	Diagnostics	Oligoclonal bands	Erythema migrans	Autonomic dysfunction	Sensory level	Images	Therapy	Outcome
1 ([8])	1986	19	F	Belgium	none	2 weeks	CSF WBC-10, 90% lymphocytes; + Serum antibodies (IgG)	Positive	Yes	None	T4	No	Penicillin G for 2 weeks	Improved
2 ([9])	1995	42	M	UK	none	5 days	LP not done (patient declined) Serum antibodies positive (not specified IgM or IgG)	N/A	Yes	None	C5-6	MRI	Cefotaxime 6 g/day for 1 week then 8 weeks of doxycycline	Improved
3 ([10])	2002	40	M	France	none	1 week	CSF WBC-205, 90% lymphocytes, CSF antibodies IgM and IgG positive	Yes	Yes	Urinary and bowel incontinence	C2	MRI	Ceftriaxone 2 g/day for 3 weeks.	Improved
4 ([11])	2005	50	M	France	none	4 weeks	CSF WBC-16, 100% lymphocytes; Intrathecal <i>B.burgdorferi</i> Ab production + Serum and CSF PCR+	Yes	No	None	Not reported	MRI	Ceftriaxone 4 weeks, doxycycline 4 weeks and IV steroids	Improved
5 ([12])	2008	46	F	Georgia, USA	none	3 weeks	Positive antibodies in blood (IgM and IgG)	Negative	No	Urinary retention, constipation	T6	MRI	Ceftriaxone for 1 week plus doxycycline for 3 weeks plus IV Methylprednisolone	Improved
6 ([13])	2009	21	F	Turkey	none	3 months	CSF WBC-20, 86% lymphocytes, CSF and serum antibodies + (IgM and IgG)	Negative	No	None	T11	MRI	Ceftriaxone 4 g/day for 28 days plus (200 mg/day of doxycycline	Improved
7 Our case	2018	25	M	Wisconsin, USA	none	2 weeks	CSF WBC 316, 76% lymphocytes, + CSF <i>B.burgdorferi</i> PCR	Negative	Yes	Urinary retention, constipation	T5	MRI	IV ceftriaxone (28 days) and IV Methylprednisolone (3 days)	Improved

The method of choice for laboratory diagnosis of non- neuro-invasive LD is serology. This involves a two-step approach where serum samples that are positive or equivocal for antibodies against LD-causing *Borrelia* species by enzyme linked immunosorbent assay (ELISA) assay are tested with a confirmatory immunoblot assay [4,23]. It is important to note that serology may be negative in early LD, as seen in our patient. Definitive laboratory diagnostic criteria for LNB are not as well-established as those for non-neuroinvasive LD. The diagnostic specificity of serum antibodies for LNB is low owing to baseline seropositivity of normal population in endemic areas and persistence of antibodies for years after successful therapy for LD. Of note, the diagnosis of LNB related ATM was not confirmed in 3 of the 6 reported cases in Table 2 as their diagnosis was based only on positive serology. As noted above, another disadvantage of using serum antibodies as a diagnostic test for early LNB is a lower sensitivity in patients with less than 6 weeks of symptoms. Intra-theal production of antibodies against Bbsl is considered diagnostic of LNB [23]. However, the detection of anti-Bbsl IgM and IgG antibodies in CSF is not definitive evidence of intra-theal antibody synthesis as serum antibodies may be present in the CSF due to blood contamination in a traumatic lumbar puncture or by passive diffusion of serum antibodies across the blood-brain barrier [5]. A LD antibody index (AI) calculates the ratio of IgG in CSF to serum after normalization for total IgG and albumin in both specimen sources. An AI ratio of ≥ 1.5 corresponds to true, intra-theal antibody synthesis and has a diagnostic sensitivity of 80% and 100% for early and late LNB [5]. Our patient presented with very early LNB and had tested negative for serum antibodies against Bbsl. Hence, we used PCR in CSF to establish the diagnosis of LNB. Even though PCR on CSF samples may aid in the diagnosis of very early LNB (prior to intra-theal antibody synthesis), it is not recommended as the test of choice for LNB because of its low diagnostic sensitivity. PCR testing for LD in CSF has a median sensitivity of 10–30% for the diagnosis of early LNB and even lower sensitivity for those with chronic symptoms or late LNB [23]. Among the 6 previously reported cases of ATM related to LNB (Table 2), only one patient had a positive CSF PCR for *B.burgdorferi*. There are novel promising tests such as B-cell attracting chemokine CXCL13 which is increased in CSF of patients with early LNB. However, these newer tests are not currently recommended for routine diagnostic or follow up testing in patients with LNB [23].

LNB should be treated with ceftriaxone for four weeks [4,25]. The treatment in reported cases greatly varied, however all patients improved. We identified two reported cases in whom steroids were used in combination with antimicrobial therapy for LNB. In one [11], steroid was started empirically for possible demyelinating or autoimmune disease and was stopped after *B. burgdorferi* PCR in CSF was reported. In the second case report [12] of severe LNB causing encephalomyelitis and axonal polyneuropathy, clinical improvement was reported with the administration of two antimicrobials along with steroid therapy. Similar to these two cases we initially treated our patient with methylprednisolone for three days. This treatment was started empirically for possible demyelinating or autoimmune disease and was stopped after *B. burgdorferi* PCR in CSF was reported. Additionally, steroids were chosen to limit symptomatic spinal cord edema associated with the infection. The role of immunosuppression is not clear in patients diagnosed with ATM caused by infection. Ramesh et al [24] showed that inflammation has causative role in rhesus macaques' model of neuroborreliosis and their data suggest that dexamethasone inhibited inflammation-related lesions in brain, spinal cord, and nerve roots, and dorsal root ganglion. Translating beneficial effects of dexamethasone in the above noted animal study into clinical practice is still controversial. One retrospective study of patients with LNB-associated facial palsy showed that

patients treated with steroids did worse [25]. At this time, the question whether steroids might be beneficial in the treatment of LNB is unresolved and there is no clear evidence to support their use as of yet.

Conclusion

We describe the second case of ATM as a manifestation of LNB in a patient in the U.S. ATM is an exceedingly rare but potentially devastating complication of LD. In spite of its dramatic presentation, the majority of patients completely recover following appropriate therapy. Clinicians, particularly those in LD endemic areas, should remain cognizant about this rare clinical presentation of LNB in order to be able to diagnose and treat their patients effectively. We recommend testing for LNB in patients presenting with inflammatory acute transverse myelitis in endemic areas.

Author's contribution

Igor Dumic wrote the manuscript, searched the literature, revised manuscript throughout the peer review process and took care in clinical care of the patient.

Danilo Vitorovic and Scot Spritzer edited ATM part of the manuscript and provided relevant literature. Scot Spritzer took part in clinical care of the patient.

Erik Sviggum provided the images and their description and reviewed the final version of the manuscript

Janki Patel edited manuscript for intellectual content and took care in clinical care of the patients.

Poornima Ramanan search the literature, wrote parts of the manuscript and edited manuscript throughout the review process as well as reviewed it for intellectual content

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