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# Lyme disease presenting as subacute transverse myelitis

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*Abstract*

*Lyme disease (borreliosis) is a systemic illness resulting from infection with the spirochete* Borrelia burgdorferi. *It is transmitted to humans by the bites of infected ticks belonging to several species of the genus* Ixodes. *After the bacteria enter the body via the dermis, most patients develop the early, localised form of Lyme disease, which is characterised by erythema migrans and influenza-like symptoms. This disease may also affect the heart, nervous system and joints. The neurological findings of this disease may include peripheral and central nervous system signs. A 21-year-old woman attended a family medicine outpatient clinic complaining of unexplained pain and muscle power loss in her lower extremities. The problem had started in her right leg 3 months earlier and worsened in the last week. She had a neurology consultation and was hospitalised. Her neurological examination revealed bilateral facial paralysis and sensory impairment. Immunoglobulin M antibody to* B. burgdorferi *was positive on Western blotting in both serum and cerebrospinal* fluid. The patient was diagnosed with subacute neuroborreliosis and treated.

*Key words*: Subacute transverse myelitis; facial paralyses; Lyme disease.

## Introduction

Transverse myelitis is an acute or subacute inflammatory disease of the spinal medulla that may present with weakness in the upper or lower extremities, autonomic nervous problems, such as disturbed bladder and bowel sphincter control, and sensory impairment below the lesion level (Kaplin *et al.*, 2005; Hammerstedt *et al.*, 2005). It has a multifactorial etiopathology, including viral infections, vaccinations, connective tissue disorders, demyelinating disorders and occasionally Lyme disease.

Lyme disease (borreliosis) is a tick-borne systemic infection caused by the spirochete *Borrelia burgdorferi*. The bacterium is introduced into the skin via a bite from an infected *Ixodes* tick. The early clinical manifestations of Lyme disease are typically characterised by erythema migrans and influenza-like symptoms. The most common symptoms include skin and musculoskeletal involvement. Untreated infection can result in advanced disease involving the heart, nervous system or joints. This paper presents a case of transverse myelitis secondary to borreliosis, which is a rare clinical manifestation.

## Case report

A 21-year-old college student who lived out of town attended a family medicine outpatient clinic complaining of unexplained pain, itching and muscle power loss in her lower extremities. She stated that the problem had started in her right leg 3 months earlier and had worsened in the last week. She also reported hypersensitivity to touch in the lower extremities. She had no other symptoms at that time. A dermatologist had diagnosed it as herpes zoster 2 months earlier and she had taken valacyclovir 1,000 mg three times daily for 7 days and acyclovir cream two times daily for 5 days. In the last week, she had noticed increasing difficulty walking. She had no history of systemic disease and her family history was unremarkable. She did not use alcohol, cigarettes or illicit drugs. Her family history was negative for neurologic disease. On systemic investigation, she also reported dysuria, dyspepsia, nausea and vomiting for the previous 3 days. Otherwise, the systems review was negative.

On physical examination, her temperature was 36.5°C, pulse 82/min, respiratory rate 18/min, blood pressure 121/69 mm Hg and body mass index 19 kg/m$^2$. The neurological examination revealed facial diplegia/paraparesis syndrome (hip flexion 3/5, knee flexion 3/5, knee extension 4/5, foot dorsiflexion 1/5, plantar flexion 2/5). Sensory testing

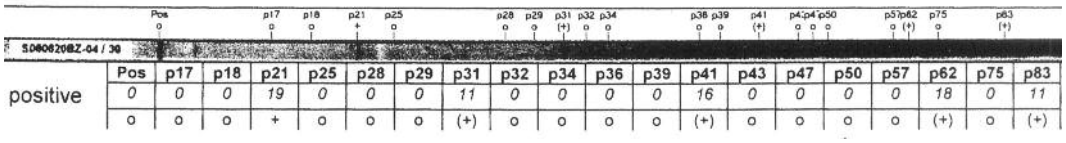


FIG. 1. — Borrelia burgdorferi IgM (Western Blot) was positive (p21, p31 and, p83 rerpectively) in patient's serum

revealed allodynia with light touch on the limbs and pinprick below T11. Cerebellar tests were difficult to administer due to the muscle weakness. The right patellar and left Achilles reflexes were absent, the other deep tendon reflexes were hypoactive, and Babinski's sign was equivocal bilaterally. Vibratory sense was reduced at the lower extremities. The examination of the other systems was unremarkable.

She consulted the neurology department and was hospitalised for further investigation and treatment that day.

On laboratory examination, the complete blood cell count, blood biochemistry, electrocardiogram, chest X-ray, echocardiogram and abdominal ultrasonography were normal. The erythrocyte sedimentation rate was 24 mm/h. She had a lumbar puncture and cerebrospinal fluid (CSF) studies done, which revealed a normal opening pressure (140 mm $H_2O$); the CSF glucose levels were normal. Total protein level was mildly elevated (52.75 mg/dL; after treatment: 33.45 mg/dL) CSF white cell count was 20/mm$^3$ (86% lymphocytes and 14% neutrophil; after treatment: 1 lymphocyte/mm$^3$). No oligoclonal bands were detected and IgG index was normal. Herpes simplex, mycoplasma, coxiella, cryptococcus, brucellosis were all negative. Antiviral serology VDRL, TPHA, HIV and serum antinuclear factor were negative, her CSF was positive for immunoglobulin M (IgM) antibody to *B. burgdorferi*, and her serum Lyme antibody titer was positive on Western blotting (Fig. 1). The IgM antibody to *B. burgdorferi* was negative but positive for IgG antibody in Elisa test and electromyoneurography was normal. Somatosensory evoked potential studies showed right fasciculus gracilis involvement. Visual evoked potential studies was normal. Thoracic, abdominal and pelvic computed tomography (CT) were normal.

Cerebellar contrast magnetic resonance imaging (MRI) was normal. Spinal MRI revealed lesions showing heterogenic signal gain with contrast, causing swelling along the intramedullary and caudal fibres from the C5–T2 and T6–T12 levels to the medullary cone (Figs. 2 and 3A, B). Leptomeningeal lesions showed signal gain with contrast along the caudal fibres secondary to infection.

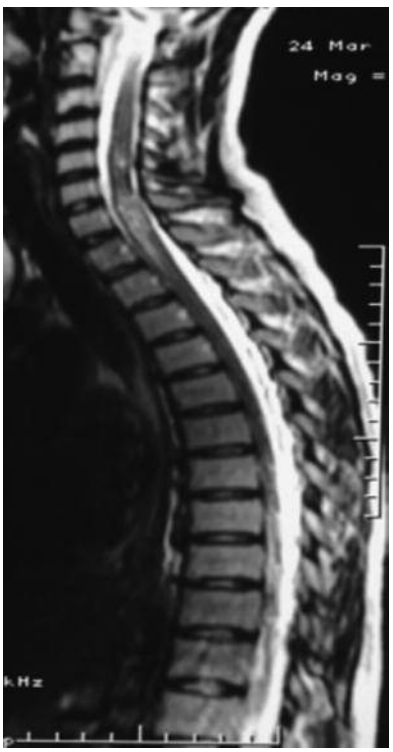


FIG. 2. — Sagittal T2 weighted FSE (TR:4420 ms; ef TE:85,8 ms, ETL: 23) cervicothoracic spinal MRI shows intramedullary inhomogeneous hyperintense lesions causing mild expansion of the spinal cord at the C5–T2 levels.

The patient was diagnosed with Lyme disease and treated with 4 g/day ceftriaxone (IV) for 28 days and then with 1200 mg/day doxycycline for 3 weeks. She was also given pentoxifyllin 2 g/day and carbamazepine 400 mg/day for pain. Her symptoms improved. The patient was hospitalised for 30 days and discharged in stable condition.

At her one -month follow-up, the right patella reflex was absent and the Achilles reflex was hypoactive bilaterally. Otherwise, her neurological examination was normal. At her fifteen- months follow-up, neurological examination was the same. IgG antibody to *B. burgdorferi* was still positive after treatment by Elisa.

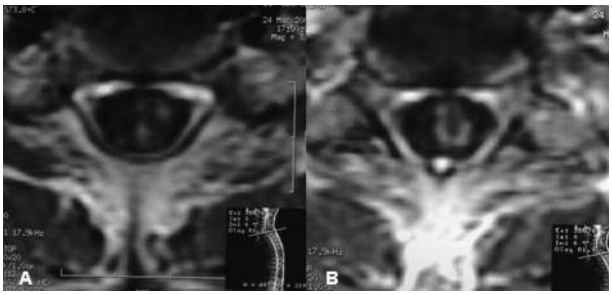


FIG. 3. — Axial postgadolinium T1 weighted SE (TR: 500 ms; TE: 13 ms) MRI shows enhancing intramedullary lesions (A, B).

## Discussion

The causative agent of Lyme disease is *B. burgdorferi*, a tick-borne spirochete. This zoonotic disease was first recognised in the United States in 1970. After it was seen frequently in the vicinity of Lyme, Connecticut, it was named Lyme disease in 1975 (Steere *et al.*, 1977). In 1982, Willy Burgdorferi discovered that *B. burgdorferi* was the agent of Lyme disease (Burgdorfer *et al.,* 1982 ). The disease occurs worldwide and it is most frequent between May and July. Lyme disease occurs in both genders and at all ages, but is more frequent in children and adults between the ages of 30 and 59 than in other age groups. Lyme disease is a multisystemic illness that can affect the dermis, nervous system, heart and joints. The disease has three clinical stages: early localised infection, with erythema migrans, fever, malaise, fatigue, headache, myalgias and arthralgias; early disseminated infection with neurologic, musculoskeletal or cardiovascular symptoms; and late disseminated infection with arthritis (Bratton *et al.*, 2008). About 80% of all borreliosis cases have skin manifestations (Mullegger, 2004). Psychiatric symptoms can also occur, such as panic attacks, anxiety and depression (Lopez *et al.*, 2008; Meurs *et al.*, 2004; Lesca *et al.*, 2002). Neurological symptoms occur in 15% of the patients and usually manifest as unilateral, rarely bilaterally, facial paralysis, acute hemiparesis, meningitis, encephalitis, transverse myelitis, headache and radiculoneuropathy (Lopez *et al.*, 2008; Meurs *et al.*, 2004; Lesca *et al.*, 2002). Transverse myelitis occurs in less than 5% of all cases of neuroborreliosis in large series (Hansen *et al.*, 1992). In most cases, the symptoms of spinal cord involvement occur 3 months after the tick bite and usually present with radicular pain. In our patient, the clinical symptoms started with pain and were followed by leg weakness that worsened subacutely. Her neurological examination showed bilateral facial diplegia, paraparesis, localised sensory impairment, signs of proprioceptive involvement and allodynia.

The first symptom of Lyme disease is usually a red rash known as erythema migrans, which starts as a small red spot at the site of the tick bite. The spot expands over a period of days or weeks, forming a circular or oval rash. The rash may resemble a bull's eye, appearing as a red ring surrounding a clear area with a red centre. The rash, which appears within a few weeks of a tick bite and usually at the site of the bite, can range in size from 1 cm to the width of the back. As the infection spreads, rashes can appear at different sites on the body. Erythema migrans is often accompanied by symptoms such as fever, headache, a stiff neck, body aches and fatigue. Although these flu-like symptoms may resemble those of common viral infections, Lyme disease symptoms tend to persist or may come and go. Therefore, this disease is diagnosed easily if the patient notices the tick bite and erythema occurs in the acute stage. The patient's complaints and clinical situation are important for diagnosing the disease in the chronic stage. In some cases, the patients may not notice the tick bite and will consult different doctors, sometimes being falsely diagnosed with arthritis, fibromyalgia, chronic fatigue syndrome, infectious mononucleosis, depression or multiple sclerosis. Our patient did not report a tick bite, but on close questioning, she recalled two or three erythematous lesions on her skin that occurred before the first complaints that were initially diagnosed as zona zoster.

The early diagnosis of Lyme disease is very important. Erythema migrans can be misdiagnosed as urticaria, eczema, sunburn and insect or flea bites. The laboratory tests are important, as are the clinical symptoms. The disease can be diagnosed using an indirect immunofluorescence method or an enzyme-linked immunosorbent assay (ELISA) showing antibody to *B. burgdorferi* in the serum and CSF. Sometimes, the blood test remains negative for a few weeks after the tick bite. Western blotting is the test used most commonly and this showed *B. burgdorferi* IgM in our patient's serum and CSF.

In cases of transverse myelitis, the spinal cord lesions can be seen on neuroimaging. These lesions are probably due to a direct infectious process, accompanied by the secretion of antibodies in the CSF (Meurs *et al.*, 2002; Lesca *et al.*, 2002; Bennet *et al.*, 2008). In our case, spinal MRI showed lesions with heterogeneous contrast enhancement causing expansion of the spinal cord.

Oral tetracycline derivatives or intravenous cephalosporin antibiotics are used in the treatment. In Europe, oral doxycycline has been found to be as

effective as intravenous cephalosporin in adults with neuroborreliosis (Ljøstad *et al.*, 2008). We treated our patient with intravenous cephalosporin followed by doxycycline.

## Conclusion

Transverse myelitis has different causes, one of which is Lyme disease. In this case report, we emphasise that Lyme disease should be included in the differential diagnosis of any patient presenting with transverse myelitis symptoms, even if the patient does not report a tick bite or erythema migrans in his/her medical history.

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