E-ISSN: 2707-8353
P-ISSN: 2707-8345
IJCRO 2022; 4(2): 21-23
Received: 16-04-2022
Accepted: 20-05-2022

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A rare case of transverse myelitis in a patient with Lyme disease

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DOI: https://doi.org/10.22271/27078345.2022.v4.i2a.118

Abstract
Acute transverse myelitis (ATM) is an inflammatory disease of the spinal cord, characterized by rapid onset of bilateral neurological symptoms. The term myelitis refers to inflammation of the spinal cord, which often leads to demyelination. The clinical finding of a pattern of altered sensation is often a horizontal band-like sensation at the dermatomal level of the lesion, with sensory changes below. The symptoms usually develop over hours to days; they typically present as muscle weakness, ascending paralysis, and autonomic dysfunction. Recovery is variable, but often prolonged over many months, and can lead to a wide range of deficits.[1] Our report reviews a unique case of transverse myelitis in a patient with Lyme disease. A 27 year old male presented to the emergency department with past medical history of diabetes mellitus and hyperlipidemia with a week long history of fever to 102°F, non-productive cough, nausea, and decreased motor function and sensation in the bilateral lower extremities. Strength was intact in bilateral biceps, triceps, and wrists, but diminished in bilateral hips, knees, and ankles. Sensation to light touch was diminished from the level of T8 and below, fine touch sensation diminished from the level of T10 and below, and there was diminished proprioception in bilateral toes. There was intact pain sensation in all extremities and hyperreflexia in bilateral upper extremities. An MRI was obtrained which demonstrated cervical syrinx with expansion of the spinal cord and resultant cervical stenosis with cord compression indicating need for surgical intervention. A posterior cervical decompressive laminectomy at levels C4-C7 and posterior thoracic decompressive laminectomy at level T1 were scheduled, with a subsequent lumbar puncture. Serum findings were significant for elevated Lyme disease antibody titer (7.89) and antibody reaction to 8 borrelial proteins. After detection of Lyme disease, the patient was started on antibiotics. Over the course of the hospital stay, motor function continually improved with the aid of physical therapy. Sensation also significantly improved. Lyme disease may not always be identifiable with CSF collection from lumbar puncture, and as a result the etiology of ATM may go unknown. Guillain-Barre syndrome is a common differential which has a similar presentation. However, the treatment consists of plasmapheresis and immunoglobulin. This is an important distinction in order to to provide the appropriate treatment modalities to the patient.

Keywords: Transverse myelitis, Lyme disease, cervical stenosis, Guillain-Barre syndrome, laminectomy

Introduction
Acute transverse myelitis (ATM) is an inflammatory disease of the spinal cord, characterized by rapid onset of bilateral neurological symptoms. The term myelitis refers to inflammation of the spinal cord, which often leads to demyelination. The clinical finding of a pattern of altered sensation is often a horizontal band-like sensation at the dermatomal level of the lesion, with sensory changes below. The symptoms usually develop over hours to days; they typically present as muscle weakness, ascending paralysis, and autonomic dysfunction. Recovery is variable, but often prolonged over many months, and can lead to a wide range of deficits [1].

Causes of this condition include idiopathic causes, immune system disorders such as Sjogren syndrome, infection with pathogens such as Syphilis, Toxoplasmosis or Aspergillosis, and Herpes Simplex Virus, and central nervous system disease processes [2]. Very rarely, infection with Borrelia burgdorferi can cause Lyme disease that progresses to ATM. Lyme disease is a multi-system, multi-stage disease that is the most common tick-borne infection in the United States. It is transmitted to humans by the Ixodes scapularis tick. Early symptoms present with erythema migrans; however, if left untreated, it may progress to affect the joints, heart, and nervous system [3].

Risk factors for Lyme disease include spending time in wooded or grassy areas in Northeast and Midwest US, having exposed skin, and failure to remove ticks promptly. Lyme disease is a very rare cause of ATM and may prove difficult for clinicians to initially identify. However, early identification of this disease as the underlying cause could lead to better patient outcomes. We present a case focused on a 27-year-old male with a rare presentation of ATM.

Case Report

A 27-year-old male presented to the emergency department with past medical history of diabetes mellitus and hyperlipidemia with a week-long history of fever to 102°F, non-productive cough, nausea, and decreased motor function and sensation in the bilateral lower extremities. Patient reported that he tested negative for COVID 3 days ago. Patient was oriented to person, place, and time with intact CN 2-12, strength was intact in bilateral biceps, triceps, and wrists, but diminished in bilateral hips, knees, and ankles. Sensation to light touch was diminished from the level of T8 and below, fine touch sensation diminished from the level of T10 and below, and there was diminished proprioception in bilateral toes. There was intact pain sensation in all extremities and hyperreflexia in bilateral upper extremities. Patient was subsequently admitted to the hospital for further workup.

Based on the patient's clinical presentation, initial imaging of the complete spine with MRI with contrast was ordered. The results of the MRI showed cervical canal syrinx extending from C6-C7 to the inferior portion of T1 (Figure 1.). Imaging also showed a multilevel degeneration of the intervertebral disks of the cervical spine which was most pronounced between C4-C6. In addition, the cervical spine showed right C4-C5 foraminal narrowing along with mild central canal narrowing at this same level. MRI of thoracic and lumbar spine demonstrated a mild left paracentral disc bulge in T12-L1, as well as multilevel lumbar spine spondylosis at L3-L4 and L4-L5. There was a 1.3cm x 0.4cm Tarlov cyst on the right at the level of L1-L2 causing potential compression of the exiting L1 nerve root. After reviewing the MRI results, the assessment was cervical syrinx with expansion of the spinal cord and resultant cervical stenosis with cord compression indicating need for surgical intervention. A posterior cervical decompressive laminectomy at levels C4-C7 and posterior thoracic decompressive laminectomy at level T1 were scheduled, with a subsequent lumbar puncture to be collected for CSF analysis in the operating room.

A posterior cervical midline laminectomy of C4-C7 and T1 were performed in sequence. Visualization of the spinal cord showed severe compression. Other findings included a thickened ligamentum flavum, spinal cord compression at the level of C7/T1, and engorged veins at C6/C7 and the lateral recess, most significant on the right. Hemostasis was established using bipolar cauterization and bone wax. Upon completion of the laminectomy antibiotic saline, vancomycin powder, and two drains were placed in the epidural space. Wound closure was established with application of a sterile dressing and then the lumbar puncture was performed. Puncture obtained 2 cc of clear CSF before the fluid became increasingly blood tinged. A second attempt was made under C-arm guidance, however it also yielded blood-tinged CSF; further attempts at LP were aborted. (Figure 2)

The procedure resulted in the successful decompression of the spinal cord and exiting nerve roots. Serum findings were significant for elevated Lyme disease antibody titer (7.89) and antibody reaction to 8 borrelial proteins. Results were notably negative for ADA, HIV, cryptococcal antigen, and RPR nonreactive. The CSF analysis at the time was pending. After detection of Lyme disease, the patient was started on antibiotics per infectious disease recommendations. In addition, the patient received daily administration of pain control, pregabalin for neurogenic hand pain, and DVT prophylaxis. Over the course of the hospital stay, motor function continually improved with the aid of physical therapy. Sensation significantly improved but remained diminished from the level of T12 and below bilaterally at the time of hospital transfer. The patient was then scheduled to be transferred to another hospital for continued care, but was lost to follow up after transfer.

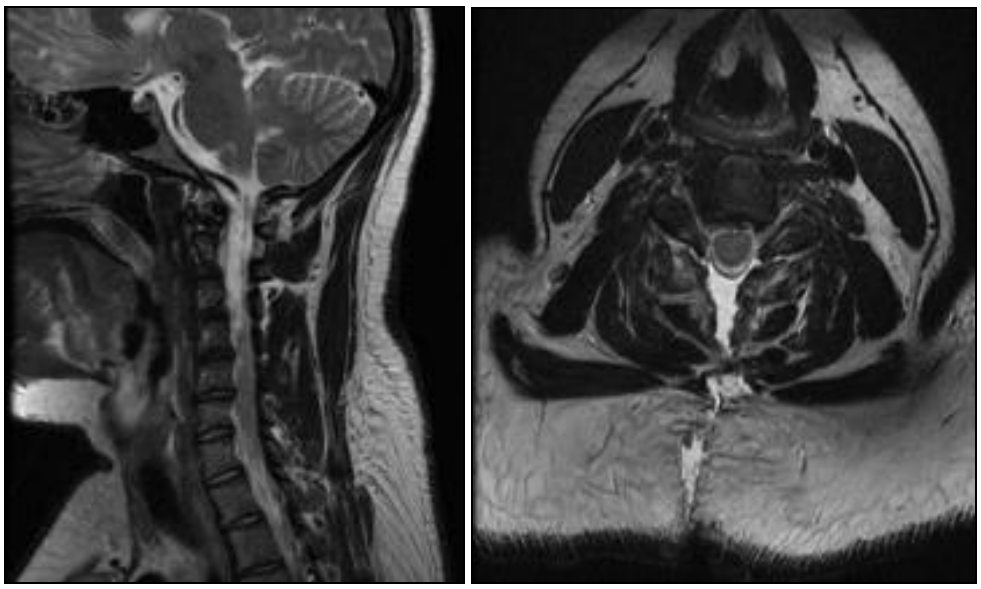

Fig 1: MRI T2 of Sagittal and axial views of the cervical spine demonstrating cervical stenosis

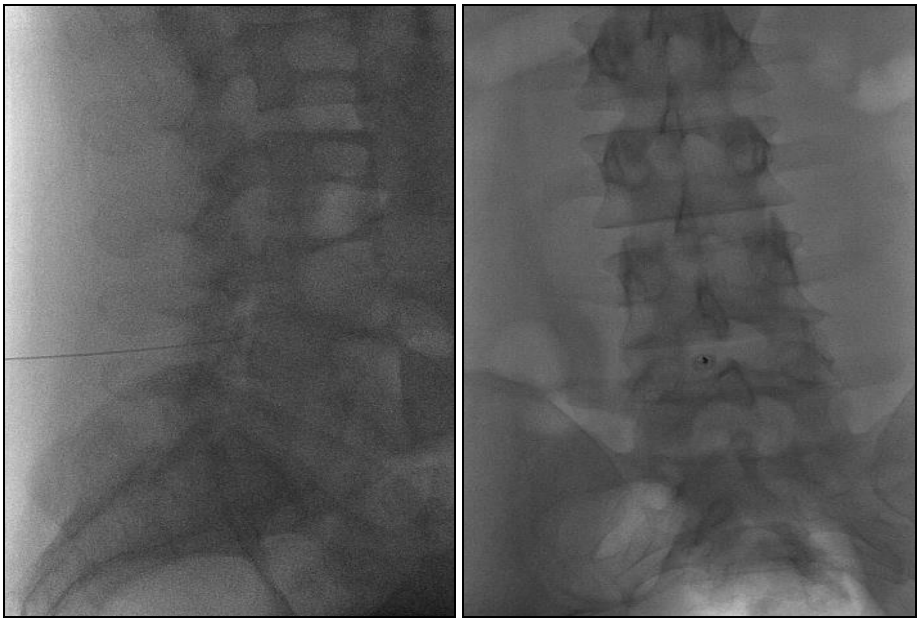

Fig 2: Lumbar puncture between L3-L4 under fluoroscopic imaging

Discussion

This case focused on a rare presentation of acute transverse myelitis caused by Lyme disease. While neurological manifestations can be observed in 3–15% of all cases of acute Lyme disease, it is typically characterized by meningitis, radiculoneuritis, and/or cranial neuritis. Any combination of these neurological symptoms is referred to as neuroborreliosis. This patient lacked the typical manifestations such as headache, nuchal rigidity, and facial nerve palsy, however presented with neurologic deficits in motor and sensory function.

In a German retrospective study following 68 patients with transverse myelitis, only 5 patients were found to have Lyme disease. The 5 patients demonstrated symptoms of paraspasticity, leg weakness, or arm weakness. By comparison to our patient, these patients presented later with a duration of symptoms between 23 and 733 days, thereby representing a manifestation of chronic neuroborreliosis [4]. A Swedish retrospective study reported isolated myelitis in 5 out of 141 patients with neuroborreliosis without further description of clinical severity [5]. Several case reports describe the manifestation of acute neuroborreliosis by myelitis, but the majority is on children [6-8].

Acute Transverse Myelitis often has varying presentations, but typically has devastating effects on the body due to neurologic involvement. While infectious causes represent less than 12% of cases of ATM, it is essential to identify the etiology in order to provide appropriate management. The diagnostic work-up of ATM requires an emergent MRI of the spinal cord to exclude acute cord compression, followed by contrast enhanced MRI of the spinal cord and brain, in addition to serum and CSF analysis. In our specific case, the surgical decompression was performed because of what was identified on imaging, however, it was suspected there was an additional secondary cause that would explain the other clinical manifestations seen in our patient. Lyme disease may not always be identifiable with CSF collection from lumbar puncture, and as a result the etiology of ATM may go unknown ultimately resulting in inadequate or lack of treatment. During the work up for this patient, Guillain-Barre syndrome was also considered given its similar presentation. However, the treatment consists of plasmapheresis and immunoglobulin. This is an important distinction to be made as the treatment modalities are vastly different. Once the etiology of Lyme disease was confirmed in this case, adequate antibiotic treatment was administered. In this regard, further research is encouraged to understand underlying risk factors and complications resulting in Lyme disease induced acute transverse myelitis. This may lead to greater efficacy in earlier diagnosis and treatment in patients.

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