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Case Report

An Unusual Presentation of Encephalitis in a Patient with Lyme Neuroborreliosis

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ABSTRACT

Lyme disease is the most common vector-borne disease in the United States. With a rate of 100.14 new cases per 100,000 residents in 2021, West Virginia has one of the highest Lyme disease incidence rates in the nation. Lyme encephalitis presents a significant challenge in clinical diagnosis and management. This case report and limited literature review provides clinicians and researchers with valuable insight into clinical approaches, diagnostic tools, and available treatment strategies. Informed consent was obtained from the patient. This report and discussion highlights the importance of combining clinical, serological, and other diagnostic tools to improve the accuracy of Lyme encephalitis diagnosis.

Keywords
Lyme disease; Lyme neuroborreliosis; Encephalitis; Bell's palsy; Neurological manifestations; Infectious diseases; Borrelia burgdorferi; Lumbar puncture.

INTRODUCTION

A recently released estimate based on insurance suggests that approximately 476,000 Americans are diagnosed with Lyme disease every year. In 2017, West Virginia became a high-lyme disease-incidence state. Lyme's disease has been known to affect multiple organ systems. Typically, Lyme's disease affects the skin and joints. However, the third most involved organ system is the nervous system. Early Lyme neuroborreliosis includes meningitis, cranial neuritis, radiculoneuritis, and, more rarely, encephalomyelitis. It typically has an onset over hours or days and occurs in the first few months of infection. Later in the infection, it may similarly involve the central nervous system or peripheral nervous system but have a more indolent evolution. Pathophysiologically, there is probably little difference between the early and late stages.[1]

Rarely, patients in the United States have also reported inflammatory encephalitis associated with Lyme's disease.[2] Patients can present with either acute or subacute signs and symptoms.[3] Symptoms can occur anywhere between 1-month and 14 years after the onset of the disease. Some signs and symptoms include fatigue, memory loss, generalized malaise, mood changes, sleep disturbances, headaches, hearing loss, and weakness. In patients with neurological signs and symptoms of Lyme's encephalitis, it is essential to perform a lumbar puncture. These patients tend to present with an inflammatory cerebrospinal fluid (CSF) profile.[4] In this case report, we discuss a patient who presented with Bell's palsy along with significant weakness.

CASE PRESENTATION

The patient is a 42-year-old male, homeless, and a chronic smoker with no known significant past medical history. He presented to the emergency department with a 2-day history of right-sided facial numbness and tingling, paralysis making it difficult for him to eat or drink, and endorsing neck rigidity with the inability to flex, extend, or rotate his cervical spine. He also mentioned that his eyes were deviating to the left. These symptoms were associated with chills, weakness, and headaches. He denied any gait disturbances, speech difficulties, fever, nausea, vomiting, shortness of breath, cough, or vision changes. There was no recent insect or tick bite history or notable open wounds. In the emergency department, strokes were ruled out first. No additional intracranial imaging was done. Lyme's antibody testing was done considering his symptoms and examination were positive for Bell's palsy, and a

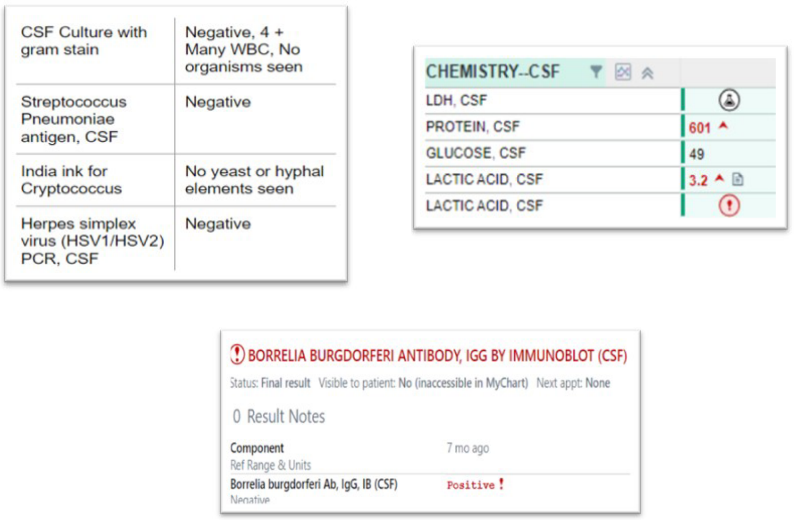
urine drug screen was performed, which was positive for fentanyl, methamphetamines, and marijuana. The patient was counseled to stop drug abuse as methamphetamine use could contribute to his symptoms. He was discharged to a skilled care facility considering his social history and asked to follow up regarding Lyme's antibody test results with the primary care provider. Unable to reach him the same day of the results, the patient was contacted the next day and advised to report to the emergency department immediately for a positive Lyme's antibody test.

On admission, the patient was afebrile, confused, and had a heart rate of 99 beats per minute, with worsening of the symptoms he presented with 4 days ago. The review of systems was now positive for sinus pain, photophobia, nausea, speech changes, and back pain. His physical examination was significant for the seventh cranial nerve deficit with facial palsy and perioral paralysis, bilateral ptosis, and right paralytic strabismus. Motor weakness, rigidity, and tenderness of the cervical spine were also noted.

His laboratory data revealed mild hyponatremia of 136 mmol/L and hyperkalemia of 5.7 mmol/L. The patient was hypoglycemic at 68 mg/dL. The chest X-ray was negative. A computed tomography (CT) brain without intravenous (IV) contrast did not show acute intracranial hemorrhage, mass, or infarct. Blood cultures were collected. A social worker was consulted to address the homelessness.

Hypoglycemia was treated with dextrose, and maintenance fluids (lactated ringer's (LR)) were started for the patient. Hyperkalemia was treated. Lumbar puncture was ordered with consideration of Lyme's meningitis; ceftriaxone, vancomycin, and acyclovir were initiated, and consultation was sent to Infectious Diseases. Intravenous Methylprednisolone was started in the setting of Bell's palsy. Counseling was done for polysubstance abuse. As per infectious diseases (ID), CSF antibody and herpes simplex virus (HSV) CSF polymerase chain reaction (PCR) were added to the studies to rule out *Streptococcus pneumonia* or the viral etiology of meningitis, with an increasing Ceftriaxone dose of 2 g twice a day. In 2 days, acyclovir was discontinued as the CSF HSV PCR was negative. Considering Lyme neuroborreliosis, IV Ceftriaxone was continued for 28 days (about 4 weeks), irrespective of the CSF culture results. Over the course of 4 days, the patient's facial palsy started improving. CSF cultures were negative for *Streptococcus pneumoniae*, Cryptococcus, and positive for *Borrelia Burgdorferi* immunoglobulin G (IgG). An magnetic resonance imaging (MRI) of the brain was not performed as CSF was diagnostic. The next plan was to send a patient to long-term acute care to complete his 28-day course of IV ceftriaxone (Figure 1).

Figure 1. *CSF Findings*



DISCUSSION

Lyme neuroborreliosis is a tick-borne neurological infection caused by the spirochete *Borrelia burgdorferi*. Typical manifestations of the central nervous system include radiculoneuritis, cranial neuritis, and lymphocytic meningitis. There exists the so-called Bannworth triad: a combination of painful meningo-radiculitis, peripheral motor paresis, and spinal fluid inflammation. Diagnosis of definite Lyme neuroborreliosis by using the criteria from the European Federation of Neurological Societies requires neurological symptoms compatible with lyme neuroborreliosis (LNB), CSF pleocytosis (white blood count (WBC) counts >5/cm^3), and detection of intrathecal *Borrelia burgdorferi*-specific IgG and/or immunoglobulin M (IgM) antibodies with a high sensitivity of 97-99%. In addition to the criteria used above, additional imaging, such as magnetic resonance imaging (MRI) without contrast, shows multiple bilateral foci of T2 hyperintensity in periventricular or subcortical white matter that may mimic multiple sclerosis.[5] Direct detection of *Borrelia burgdorferi* in blood by PCR or culture is rarely helpful, with reported sensitivities between 1% and 28% in patients with otherwise verifiable infections.[6]

Neurological manifestations may vary from confusion or disorientation to personality changes. Other common central nervous system (CNS) symptoms noted were aphasia, ataxia, and hallucinations, along with typical LNB symptoms such as radicular pain or peripheral facial nerve palsy. Although facial nerve involvement is the most common presentation, involvement of the nerves to the extraocular muscles, the trigeminal (5th), and occasionally the acousticovestbibular nerve occurs as well.[7] Treatment regimens for Lyme disease range from 14 days (about 2 weeks) in early localized disease to 14-21 days (about 3 weeks) in early disseminated disease to 14-28 days (about 4 weeks) in late disseminated disease. Neurological complications of Lyme disease may be treated with doxycycline, as it can be taken by mouth and has a lower cost; however, IV Ceftriaxone is preferred in late-disseminated disease. There are no statistically significant differences found in either response rate or adverse effects. In two studies, 14-day courses of oral doxycycline (200 mg/day), IV penicillin, and IV ceftriaxone were equally efficacious.[8,9] No studies have directly compared the efficacy of a 14-versus-21-day course in patients with involvement of neurological symptoms. Of note, IgM and IgG antibody levels may be elevated for years even after successful treatment with antibiotics.[10]

CONCLUSION

Early disseminated Lyme neuroborreliosis is an uncommon diagnosis that requires a high degree of clinical suspicion and awareness, as patients don't always recall a tick bite. 20% of patients have CNS involvement, with 5% having bell's palsy.[11] Highly sensitive and specific tests of the CSF for Lyme's neuroborreliosis are still lacking. However, CSF analysis has a primordial role in excluding other diagnoses. Measurement of the chemokine (C-X-C motif) ligand-13 (CXCL-13) biomarker in CSF for LNB is currently under development and, when widely accessible, may reveal higher specificity to obtain lumbar puncture (LP) in diagnosing and treating LNB.[12] Studies have shown that treating LNB with oral doxycycline for all early-disseminated neurological manifestations is also an effective option, as IV ceftriaxone makes the treatment easy and the decision unlikely to be altered by the results of a fairly invasive test.[13]

CONSENT

The authors have received written informed consent from the patient.

CONFLICT OF INTEREST

No potential conflict of interest related to this article was reported.

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