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Lyme Disease: A Case Presentation in a Rural Western New York Primary Care Setting

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Introduction

Lyme disease is caused by the spirochete *Borrelia burgdorferi*, transmitted through the bite of an infected tick from the *Ixodes* genus. In the northeastern United States, *Ixodes scapularis* (blacklegged tick) serves as the primary vector. Lyme disease is the most commonly reported tick-borne illness in the U.S. [1]. First identified clinically in 1977 as "Lyme arthritis" during a study of a cluster of rheumatoid arthritis cases in Connecticut, it has been a nationally notifiable condition since 1991, with a substantial increase in incidence over time [2,3]. In 2022, 62,551 cases were reported in the U.S., with New York accounting for the highest number of cases at 16,798 [4].

Over the past two decades, the prevalence of Lyme disease in Western New York has risen significantly, mirroring trends observed throughout the northeastern U.S. An increasing number of cases of Lyme disease are originating from Central New York and the Adirondacks. While Syracuse tends to see more cases than Rochester and Buffalo, there appears to be a trend of increasing cases year over year in the Western New York region [5]. This increase is largely attributed to changes in tick populations and climate conditions. These trends underscore the importance of ongoing surveillance, public education, early diagnosis, and preventive measures to effectively manage Lyme disease in this region.

Here, we describe the case of a 54-year-old male who presented with neurological symptoms to a tertiary care hospital in Western New York.

Case Presentation

A 54-year-old male farmer with a past medical history of a motor vehicle accident and chronic neck pain presented to the hospital with intractable neck pain, a skin rash, and fever. His symptoms began three weeks prior to admission with a patch of erythema in the left groin, initially treated as cellulitis with Bactrim and Keflex in a rural primary care outpatient clinic. Although the erythema in the groin resolved, the patient developed additional red patches, body aches, fever, and neck stiffness, prompting him to visit the emergency room at a tertiary care hospital in Western New York in August. He described the rash as expanding red circles with central clearing. While the patient reported no known tick bites, he admitted to frequent outdoor activities. On examination, he had a low-grade fever of 99.1°F, neck stiffness, and multiple bull's-eye lesions on his torso and lower extremities. Laboratory tests showed mild liver enzyme elevation (AST 40, ALT 89), with normal white blood cell count, hemoglobin, and platelet counts. A CT scan of the head revealed no hydrocephalus or acute parenchymal abnormality. Lyme antibody screening was positive, and the Lyme Western blot was positive for IgM and negative for IgG. A lumbar puncture revealed 409 nucleated cells with 73% lymphocytes and 21% mononuclear cells, with CSF glucose at 47 and CSF protein at 188. The patient was diagnosed with disseminated Lyme disease with meningitis and was treated with IV ceftriaxone for 21 days, leading to a full recovery.

Microbiology and Vector

Lyme disease is an infection caused by spirochete bacteria, transmitted through the bite of infected ticks from the *Ixodes*

ricinus complex. In the United States, the disease is mainly caused by *Borrelia burgdorferi*, while in Europe and Asia, *B. afzelii*, *B. burgdorferi*, and *B. garinii* are the primary culprits.

The life cycle of *Ixodes scapularis* involves three stages: larva, nymph, and adult, spanning approximately two years [6]. Larvae hatch in early summer, seek a blood meal from small animals, and acquire *Borrelia burgdorferi* if the host is infected. After feeding, larvae molt into nymphs, which emerge the following spring. Nymphs play a critical role in transmission, as they feed on small mammals and spread the spirochete. Infected nymphs molt into adults in the fall, with adult females feeding primarily on white-tailed deer. After a blood meal, females lay eggs in the spring, perpetuating the cycle. Ticks use warmth and carbon dioxide to locate hosts, and the transmission of *B. burgdorferi* typically requires the tick to be attached for 36 to 48 hours [7].

Clinical Manifestations

The clinical manifestations of Lyme disease are categorized into three phases: early localized, early disseminated, and late disease.

a) Early localized disease: Characterized by the appearance of erythema migrans (EM), a distinctive skin lesion, which may or may not be accompanied by constitutional symptoms. EM typically appears at the site of the tick bite within 7-14 days [8].

b) Early disseminated disease: Marked by multiple EM lesions (usually occurring days to weeks after infection) and/or neurologic [9] and/or cardiac findings [10] that typically appear weeks to several months after the onset of infection. Some patients may not have a history of antecedent early localized Lyme disease. Lymphocytic or monocytic meningitis, often occurring on its own or in conjunction with cranial nerve or spinal nerve root involvement, is the most prevalent form of nervous system involvement in Lyme disease. This type of meningitis typically develops several weeks to a few months following a tick bite and may even serve as the initial indication of Lyme disease.

c) Late Lyme disease: Often associated with intermittent or persistent arthritis involving one or a few large joints, particularly the knee, and/or certain rare neurologic issues, such as subtle encephalopathy or polyneuropathy. Late Lyme disease may develop months to a few years after the initial infection, with arthritis sometimes being the presenting manifestation of the disease [11].

Diagnosis

The diagnosis of Lyme disease should be considered in patients with a history of tick exposure who present with consistent symptoms. The diagnostic approach depends on the stage of infection. For patients with erythema migrans (EM), diagnosis can often be made based solely on clinical presentation, especially if the patient has characteristic EM lesions and has either resided in or recently traveled to an endemic area. In such cases, serologic testing is not required when the clinical presentation is clear.

For patients with early disseminated or late Lyme disease, serologic testing is recommended to support the diagnosis. However, serologic testing should complement, not replace, clinical diagnosis, as it cannot independently confirm or exclude Lyme disease. Clinical judgment, along with the patient's history and symptomatology, is crucial for an accurate diagnosis.

When specific diagnostic tests are warranted, a two-tiered conditional strategy should be employed. This strategy typically involves an initial enzyme-linked immunosorbent assay (ELISA) or immunofluorescence assay, followed by either a Western blot (traditional two-tiered testing) or a second ELISA targeting different antigens (modified two-tiered testing) [12]. For cases involving synovial or cerebrospinal fluid, PCR testing may be requested to confirm Lyme disease in patients showing signs of aseptic meningitis, radiculoneuritis, or Lyme arthritis. Polymerase chain reaction (PCR) testing for *B. burgdorferi* is not usually recommended for patients suspected of having Lyme disease in the central nervous system (CNS). This is because the test has low accuracy, likely due to the very small number of bacteria present in spinal fluid. Additionally, some labs struggle to prevent false-positive results, making the test less reliable overall. Urinary antigen testing and PCR on inappropriate specimens, such as blood and urine, are not very accurate for diagnosis and have limited utility.

Treatment

The treatment of Lyme disease depends on the stage of the disease:

a) Early localized disease: A 10-day course of doxycycline is indicated [13].

b) Early disseminated disease with neurologic findings: A 21-day course of oral doxycycline is sufficient [14]. If there is concern for severe neurologic disease, ceftriaxone 2g daily for 2-4 weeks can be used.

c) Lyme carditis and severe neurologic conditions (e.g., Lyme encephalitis): Treatment with intravenous ceftriaxone for 14-28 days is recommended [15].

d) Late Lyme disease with arthritis: At least 28 days of oral doxycycline are indicated [16].

Conclusion

The incidence of Lyme disease is on the rise in North America, with the disease presenting with a wide variety of symptoms. Lyme disease can affect the nervous system and can manifest in both early and late stages. Clinicians should maintain a high level of suspicion and initiate empiric treatment when a patient's clinical presentation is consistent with neurological involvement of Lyme disease. Early manifestations of nervous system Lyme

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disease typically occur within 6 months of infection and may include cranial and peripheral neuropathy, radiculitis, and aseptic meningitis. Late manifestations can present as chronic encephalomyelitis.

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