Texas Occurrence of Lyme Disease and Its Neurological Manifestations

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Abstract

Today, Lyme disease is the most commonly reported tick-borne disease in the United States and Europe. The culprits behind Lyme disease are the *Borrelia* species of bacteria. In the USA, *Borrelia burgdorferi* causes the majority of cases, while in Europe and Asia *Borrelia afzelii* and *Borrelia garinii* carry the greatest burden of disease. The clinical manifestations of Lyme disease have been identified as early localized, early disseminated, and late chronic. The neurological effects of Lyme disease include both peripheral and central nervous systems involvement, including focal nerve abnormalities, cranial neuropathies, painful radiculoneuritis, meningitis, and/or toxic metabolic encephalopathy, known as Lyme encephalopathy. Given the geographic predominance of Lyme disease in the Northeast and Midwest of the USA, no major studies have been conducted regarding Southern states. Between 2005 and 2014, the Center for Disease Control has reported 582 confirmed cases of Lyme disease in Texas. Because of the potential for increased incidence and prevalence in Texas, it has become essential for research and clinical efforts to be diverted to the region. The Texas A&M College of Veterinary Medicine and Biomedical Sciences Lyme Lab has been investigating the ecology of Lyme disease in Texas and developing a pan-specific serological test for Lyme diagnosis. This report aimed to exposure materials and raise awareness of Lyme disease to healthcare providers.

Introduction

When discussing neuro-infectious diseases, Lyme disease can be considered one of the more novel and mysterious entities currently in existence. Indeed, its formal discovery in the United States was as recent as 1977 and was originally identified as "Lyme arthritis" during studies of a cluster of children in Connecticut who were thought to have juvenile rheumatoid arthritis [1]. Today, Lyme disease is currently the most commonly reported tick-borne disease in the USA and Europe. The culprit behind Lyme disease are the *Borrelia* species. In the USA, *Borrelia burgdorferi* causes the majority of cases, while in Europe and Asia *Borrelia afzelii* and *Borrelia garinii* cause the most burden of disease. They cause a spirochetal infection transmitted by the bite of infected Ixodes ticks. The primary reservoir hosts for *B. burgdorferi* in northeastern USA are rodents, including white-footed mice, voles, and chipmunks [2].

The clinical manifestations of Lyme disease have been well documented over the last several decades and three distinct phases of the disease have been identified. In the early localized phase, which occurs several days to one month after infection, the characteristic erythema migrans rash manifests in approximately 80% of patients, with constitutional symptoms such as fatigue, malaise, lethargy, mild headache, mild neck stiffness, myalgias, arthralgias, and regional lymphadenopathy also appearing variably. These nonspecific clinical presentations make Lyme disease challenging to diagnose and treat at this stage, especially in non-endemic areas of the USA. If not treated properly, the disease progresses to the early disseminated phase and can present with carditis, neurological symptoms, migratory arthralgias, multiple erythema migrans lesions, localized scleroderma (morphoea) [3], lymphadenopathy, ocular, liver and kidney diseases and can last weeks to months. The late chronic disease presents months to years after initial infection and can include intermittent monoarticular or polyarticular arthritis, peripheral neuropathy or encephalomyelitis, and various cutaneous lesions [3,4].

The neurological effects of Lyme disease b itį3benl H L m b L D}N iple

to cognitive dulling [6]. Another study has examined cross reactivity between the outer surface protein A (OspA) protein expressed by B. burgdorferi and human neural proteins and found that cross-reactivity indeed exists between OspA and proteins expressed in brain, spinal cord, and dorsal root ganglia cells. This could indicate a reactive autoimmune component to the encephalopathy [8]. Fallon et al. explored cerebral blood flow indices in patients with chronic Lyme encephalopathy and found that patients with Lyme encephalopathy presented with slower-clearing flows

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