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Equine Lyme Disease

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ABSTRACT

Borrelia burgdorferi infection in horses is common in some areas of North America, but the incidence of clinical disease has not been determined, which makes Lyme disease controversial in the horse. Progress is being made case by case in defining the several clinical presentations of Lyme disease in adult horses. Current serologic tests are very sensitive and specific for detecting either infection (previous or concurrent) or exposure but may not distinguish between them. Treatment protocols are available for Lyme disease in horses; however, the ability of those protocols to eliminate the organism remains questionable. Currently, minocycline is being attempted in some presumed Lyme cases as an improved alternative to doxycycline and oxytetracycline. Vaccination, like many aspects of Lyme disease, remains controversial, although the canine-approved vaccines have the potential to prevent infection based upon vaccine studies in ponies and other animals.

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1. Etiology and Epidemiology

Lyme disease is caused by at least three strains of the *Borrelia burgdorferi* sensu lato spirochete complex [1,2], which includes several species worldwide and multiple variants of each species. The North American strain is *B. burgdorferi* sensu stricto [3], which may have several strain variations [4]. More recently, *Borrelia miyamotoi* has been identified in a small number of ticks in the United States, but its importance in Lyme disease is unknown [5]. *B. burgdorferi* bacteria are not free-living organisms and are maintained in a 2-year enzootic life cycle that involves Ixodid ticks (*Ixodes scapularis* in the eastern United States and *I. pacificus* on the west coast of North America) and mammals [6]. The mammals most commonly involved in maintaining the life cycle of this spirochete are white-

footed mice, which provide a continual source of the spirochete, and deer, which maintain the tick vector.

The mid-Atlantic and northeastern states have a high seroprevalence for *Borrelia* in the equine population, as do areas of Minnesota and Wisconsin, extending into southern Canada. Infected horses are also reported in some regions of California. In one New England survey, 45% of horses had *Borrelia* antibodies [7]. A Wisconsin study found that 118 of 190 horses were serologically positive [8].

Infection in horses and other mammals is caused by attachment and prolonged (>24 hours) feeding of infected adult *Ixodes* spp. ticks. Female ticks are likely the competent vector, as adult males rarely feed. Nymphs are responsible for a high percentage of infections in humans because they are small and often escape visual inspection. It is not known whether these stages transmit the spirochete to horses. *I. scapularis* adults are most active in the fall and throughout winter when temperatures are above freezing. Once feeding begins, the organism begins its complicated up-and-down regulation of genes to enhance survival in the host.

The *Borrelia* organism lives in the tick's gastrointestinal tract and is transferred to animals during blood meals. Generally, 24–48 hours of attachment are required to transfer the organism successfully from the tick to the mammalian host [9]. This time may be needed for the

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organism to downregulate an outer membrane protein (OspA), which could be important in maintaining survival in the mammalian host [10]. Conversely, other surface proteins (e.g., OspC, OspE, OspF) that are normally in low concentration in the tick gastrointestinal tract are upregulated. This is particularly true for OspC (found in the tick salivary gland), which enhances complement resistance and other methods of immune evasion in the mammalian host [11]. These changes in expression of surface proteins may be triggered by the blood meal. Other proteins, such as the C6 peptide found in a dominant invariable region of the variable major protein-like gene *VisE*, permit antigenic variation, ensuring survival in the host [12]. *B. burgdorferi* may also survive in the host by residing in collagen and connective tissue, likely via low-molecular-weight decorin-binding proteins, thus having no requirement for iron [13–15]. After experimental infection of ponies, the organism appears to reside mostly in skin near the tick bite, as well as in connective tissue and muscle and around nerves and blood vessels near synovial membranes [16]. Persistent infection, especially following antibiotic treatment, has been very controversial in humans, and most studies do not support a syndrome of chronic infection/chronic Lyme disease in properly treated patients [17,18]. Conversely, a recent article provided evidence that both cysts (round bodies) and biofilm-like colonies exist and may cause chronic disease in humans [19].

2. Clinical Findings

A wide variety of clinical signs have been attributed to *Borrelia* infection in horses, but cause and effect have been difficult to document in most cases. Because of both the high prevalence of antibodies against *Borrelia* in horses in some regions of the United States and the difficulty in proving clinical disease in most cases, Lyme disease is undoubtedly the most controversial equine disease in those areas. The clinical signs most often attributed to equine Lyme disease include stiffness and lameness in more than one limb, muscle tenderness, hyperesthesia, lethargy, and behavioral changes [20–25]. Unlike human Lyme disease, joint effusion has been minimal in most Lyme-suspect horses.

Muscle wasting and pain over the thoracolumbar area have been present in a few horses with high serum titers, and some of these horses have had neurologic signs. In one report, Lyme neuroborreliosis was diagnosed in 2 horses and both had chronic, necrosuppurative-to-nonsuppurative, perivascular-to-diffuse meningoradiculoneuritis on necropsy examination [26]. Hyperesthesia, lumbar pain, and muscle wasting were the initial clinical findings followed by ataxia of all four limbs, facial nerve paralysis, and finally head tremors with depression in 1 horse. On necropsy, spirochetes were identified by Steiner silver impregnation in both cases, predominantly in the affected dura mater of brain and spinal cord.

B. burgdorferi *sensu stricto* was identified by polymerase chain reaction and the highest spirochetal burdens were in tissues with inflammation, including the spinal cord, muscle, and joint capsules. In another report, a horse with severe neck stiffness that progressed to ataxia had lymphohistiocytic meningitis and *B. burgdorferi* DNA in the

cerebrospinal fluid (CSF) [27]. That horse originally responded to doxycycline treatment but relapsed after treatment was discontinued. Another case was a Thoroughbred hunter that presented for lameness and ataxia and had lymphocytic pleocytosis and was PCR positive for *Borrelia* on CSF analysis. The horse initially responded well to doxycycline but had some deterioration when treatment was discontinued [28]. The author examined 2 other horses suspected of having Lyme disease, with ataxia and severe lymphocytic infiltration and thickening of the meninges. Based upon these few cases, it may be that ataxia and lumbar muscle wasting caused by lymphohistiocytic meningitis and radiculoneuritis with occasional fasciculations and neck stiffness are common characteristics of neuroborreliosis in the horse. CSF would likely show lymphocytic pleocytosis and, although uncommon in humans with neuroborreliosis, some horses have CSF that is PCR positive for *B. burgdorferi*.

Bilateral uveitis was recently reported in 2 horses associated with *Borrelia* infection of the eye [29]. *Borrelia* were found on cytologic examination and confirmed by PCR in the vitreous in both horses. No organisms were observed in the aqueous although one aqueous sample was PCR positive. *Borrelia* was also observed with silver stain in the inflamed uveal tissue. In addition to uveitis, a chronic multifocal lymphohistiocytic ganglioradiculitis and neuritis with presumptive neuronal degeneration in the spinal nerves were found in 1 horse with uveitis.

Another report describes a horse with Lyme pseudolymphoma (multiple lymphohistiocytic cutaneous nodules). The horse responded completely to doxycycline treatment, as might be expected for a cutaneous form of borreliosis [30].

High fever and limb edema are sometimes reported in association with *Borrelia* seroconversion, although it is unlikely they are caused by the *Borrelia* infection. These clinical findings are most often the result of *Anaplasma phagocytophila* infection, because many ticks are concomitantly infected with both *Borrelia* and *A. phagocytophila* [31].

Experimental infection of ponies with *B. burgdorferi* caused consistent lesions in the skin and inconsistent lesions in muscle, fascia, nerves, and perisynovial tissues. However no clinical signs were observed in these ponies in spite of these lesions. Except for the lymphocytic/histiocytic dermatitis, the pathology was mild in most of the experimentally infected ponies [16]. Until clinical signs can be experimentally reproduced, the association between *B. burgdorferi* infection and clinical disease in many horses will remain speculative. The diagnosis of Lyme disease seems most common in sport horses. This might be because subtle clinical signs of stiffness and hyperesthesia are most easily recognized in horses of this discipline, rather than being related to a genetic predisposition to disease.

3. Diagnosis

The diagnosis of exposure to *Borrelia* can usually be determined by serology. To determine whether the horse is currently infected is more difficult, and to determine furthermore whether clinical disease is associated with *Borrelia* is extremely difficult! Enzyme-linked

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