It is now well known that dogs can present with a variety of clinical syndromes including fever, anorexia and arthritis⁴, as well as a distinctive form of glomerulonephritis, commonly termed Lyme nephritis⁵. To the great frustration of practitioners, clinical Lyme disease impacts only a small fraction of “infected” canine patients⁶, and experimental models have failed to recreate exactly what they are seeing in their clinics⁷-¹⁰. In 2006, the ACVIM generated a Consensus statement to try to guide practitioners on how best to diagnose, treat and prevent Lyme disease¹¹. Eight years later, uncertainty still dominates this disease entity as experts continue to debate the merits of vaccination and treatment protocols.

Prevalence

As Borrelia burgdorferi is dependent upon a host for survival, the incidence of Lyme disease closely matches the prevalence of the key reservoir host species of the Ixodes hard ticks. Movement of the mammalian host species (white tailed deer, white footed mouse) into human and canine environments (and vice versa) has significantly expanded endemic areas⁴,¹². Dogs are now widely accepted as public health sentinels for areas of Lyme Borreliosis in people⁴,¹³.

Pathogenesis

Like most bacteria, Borrelia burgdorferi contains plasmids upon which they carry their functioning genes, many of which code for proteins on its outer surface⁴¹. While in the tick, the predominant Outer Surface Protein A (OspA), allows the bacteria to remain anchored within the midgut of the tick until an acceptable host is encountered. Once the tick begins feeding, a trigger (potentially the increased temperature provided by the host) signals a down regulation of the OspA protein and the initiation of OspC expression, allowing the spirochetes to migrate to the salivary glands of the tick and be inoculated into the host¹⁵-¹⁸. This OspC, in combination with a protein from the tick salivary gland (Salp 15), covers the borreliae, allowing them to avoid initial detection by the immune system. From the dermal site, the borreliae appear to replicate and migrate through the skin and connective tissues. The OspC protein allows the organism to become established within the host, and is later supplanted by VlsE, 3-4 weeks post-infection. VlsE plays a role in chronic persistence of the bacteria and its continued avoidance of the immune system. This protein changes amino acid structure quickly, facilitating B. burgdorferi’s evasion from the host’s immune system⁴,¹⁹. It is within one constant region of this protein not expressed in the borreliae while in the tick, that a universal sequence was identified as an indicator of natural infection (peptide C6)²⁰-²².

Lyme disease was first reported in 1975 as an intermittent but mild arthritis in a group of children living in Lyme, CT¹. Borrelia burgdorferi, a tick borne spirochete, was identified as the causative agent in 1981, although transmission by the black legged tick, Ixodes scapularis had been accepted since 1977. The first canine cases were reported by 1984-1985²,³.
Clinical Syndrome
While many dogs are serologically positive, only a few (5-10%) are symptomatic. Co-infections with other organisms like Anaplasma phagocytophilum have been shown to increase likelihood of clinical presentation. The acute signs of clinical disease are often overlooked, and represent fever, lymphadenopathy, malaise and lameness in days to weeks after infection. Pain, swelling and lameness can occur weeks to months after infection, as the borreliae disseminate into the connective tissues and immune complexes are formed. In experimental infections, severe lameness can take 2-6 months to present. Signs can present as a mono- or polyarthropathy. Intermittent limping is commonly seen and may recur 2-3 weeks later in the same, or a different limb. While the clinical arthritis may be transient, inflammatory changes within the synovial fluid can be persistent.

A morphologically distinct protein-losing nephropathy has been described. One report lists incidence as high as 1-2% of serologically affected dogs. In a retrospective study of 49 dogs, the disease was primarily lethal or resulted in euthanasia in all 49 dogs within 1-8 weeks of diagnosis. Labrador and Golden Retrievers appear to be overrepresented in presenting with this invariably fatal sequel to infection.

Diagnosis
There is no pathognomonic test for Lyme Borreliosis, however serologic ViS/E/C6 based screening tests are potentially valuable for determining local incidence and thus establishing in-clinic prevention protocols. While these tests do not prove causation of signs, they do indicate exposure and the need for improved tick control. As ViS/E is not present in any of the commercial vaccines, these tests do distinguish vaccination from infection. Some laboratories also provide quantitative titer levels in an attempt to distinguish active vs. chronic infection. Currently, no titer level has been directly associated with clinical disease. While management of nonclinical, serologically positive dogs is controversial, most specialists agree that all positive dogs should be evaluated for proteinuria.

Treatment
Clinical disease in dogs has been shown to respond to doxycycline at 10 mg/kg orally once or twice a day x 30-42 days. Dogs with proteinuria may need a longer course of treatment. While not studied, minocycline at 10mg/kg PO twice a day appears to be a reasonable substitute if doxycycline is not available. In veterinary medicine, penicillin derivatives (amoxicillin and cefovecin) have also been effective but do not manage the co-infections that may magnify clinical signs. While antibiotic therapy provides resolution of signs, it may not eradicate Borrelia from the body, as evidenced by spirochete detection and even titer increases in experimental studies months after treatment. This is one of the reasons treatment of non-clinical dogs remains controversial. Those against empirical treatment raise concerns regarding the potential development of resistance, unnecessary adverse events, and the natural immunity that seems to be present in some dogs. However, proponents emphasize that treatment lowers titers faster and will decrease circulating immune complexes, the key contributors to clinical signs and the most devastating complication of Lyme nephritis. Currently, no published data exists to determine the best management. In the meantime, many are adopting the recommendation of treating animals above a certain titer level that may be associated with “active” infection. Without a true consensus, client education and involvement is critical in establishing a management plan for each individual dog.

Prevention
In some cases, aggressive prevention programs involving screening, treatment, vaccination and aggressive tick control have seemingly decreased the in-clinic incidence of Lyme positive dogs. There are currently four companies that manufacture Lyme vaccines. All produce anti-Osp A borreliacidal antibodies that can kill the borrelia within the tick as it obtains a blood meal. One of these vaccines also contains a unique isolate that induces anti-Osp C borreliacidal antibodies that provide protection against not only clinical Lyme disease but also subclinical arthritis. There continues to be debate amongst Lyme experts as to whether vaccination of positive dogs is beneficial, however, data in humans indicates that natural infection does not protect against future disease.

The greatest consensus in Lyme prevention comes in establishing a protocol for tick prevention. If avoidance of tick laden areas is impossible, it is recommended to use a tick control product with efficacy rapid and persistent enough to prevent engorgement, or a permethrin containing product in dogs for the added benefit of repellency. As no preventative is 100% effective, thorough daily tick checks are an additional measure to minimize risk.

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