# LYME DISEASE AND ANAPLASMOSIS

Lyme disease (or Borreliosis) and Anaplasmosis are recognised increasingly as causes of clinical disease in horses. Both organisms are transmitted by *Ixodes* ticks (that are common in many parts of the UK) and are obligate intracellular organisms that may infect multiple species. Some of the clinical signs are summarised below.

***LYME DISEASE***

Lyme disease is caused by the spirochete *Borrelia burgdorferi*. *B. burgdorferi* infects leucocytes and synovial lining cells and triggers an inflammatory (and potentially autoimmune) response. The persistence of the bacteria within both synovial structures and tendons in humans leads to the prolonged treatment required.

*B. burgdorferi* has been reported to cause neuroborreliosis leading to the clinical signs of ataxia, hyperaesthesia and mentation changes and this can be diagnosed based on CSF samples.

High rates of *Borrelia* seropositivity have been recorded in horses from many regions of the UK and it is likely that seropositivity for Anaplasma is similar. A study performed in 1994 indicated low levels (<7%) of seropositivity in Newmarket, Ireland, Yorkshire and Scotland but quite high levels (30-35%) in South Coast areas and East Anglia.

***ANAPLASMOSIS***

Anaplasmosis is caused by the Rickettsial organism *Anaplasma phagocytophilum* (formerly *Ehrlichia equi*). *Anaplasma phagocytophilum* infects neutrophils and eosinophils resulting in neutropaenia and anaemia. Clusters of intracellular organisms may be visible as blue-grey spoke-wheel inclusions during the initial phase of infection. The presence of visible intracellular organisms tends to correlate with the presence of pyrexia, which usually lasts for around 10 days after infection. The majority of acute infections will lead to a marked pyrexia.

| Lyme disease | Anaplasmosis |
|---|---|
| Mild pyrexia | Pyrexia |
| Lethargy | |
| Anorexia/Weight Loss | Anorexia/Weight loss |
| Stiffness/Lameness | |
| Muscle Soreness | |
| Synovial Effusions | |
| Laminitis | |
| Uveitis | |
| Somnolence/altered mentation | Somnolence/altered mentation |
| Hyperaesthesia | |
| Ataxia | Ataxia (collapse/recumbency) |
| | Ventral oedema |
| | Anaemia |
| | Petechiae/Ecchymotic haemorrhages |

***DIAGNOSIS***

Definitive confirmation of Lyme disease or Anaplasmosis is problematic and currently diagnosis is based upon finding a positive antibody titre for the suspected organism in a horse with suspicious clinical signs in an area where the disease, or at least *Ixodes* ticks, are known to be endemic. This has several limitations however. Firstly, with standard test methods it may take up to 3 months following infection for horses to seroconvert – meaning that many early cases will be 'negative' on serology. Secondly, horses may become infected and seroconvert without showing any clinical signs – hence many healthy horses or horses with other conditions could be misdiagnosed with Lyme disease on the basis of serology. Thirdly, horses that are successfully treated may still remain seropositive for a very long time thereafter – complicating interpretation of successful resolution.

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The ELISA method used at the LEH for detection of *Borrelia* targets antibodies against the *Borrelia* surface protein V1sE. In experimental infections, animals became seropositive to V1sE within 3-5 weeks of infection, well before clinical signs arose. Additionally, infected horses that were successfully treated showed waning antibody titres more rapidly than with other test methods (although this may still be a matter of months). Furthermore, the method was able to detect some seropositive cases that had been missed using standard Western Blot techniques. The diagnostic accuracy of the ELISA method used at the LEH for the identification of seroconversion to *Borrelia* has been evaluated in 3 studies with a sensitivity of 65% - 100% and a specificity of 95% - 100%.

We also now have a PCR assay targeting the OspA antigen of Borrelia confirming current infection.

The ELISA test used at the LEH to diagnose *Anaplasma* targets antibodies against peptides derived from the immunodominant p44 protein of *Anaplasma phagocytophilum*.

***TREATMENT***

Intensive treatment can include 7-10days of oxytetracycline (5 mg/kg IV SID/BID) followed by either oral doxycycline (10 mg/kg PO BID) or oral minocycline (4mg/kg PO BID) for 1-2 months for Lyme disease. Anaplasmosis can be treated with a shorter course of either oxytetracycline, doxycycline or minocycline. While treating with these antibiotic renal values should be closely monitored. Ceftiofur (2-4 mg/kg IM BID) has also been recommended.

Total eradication of organisms from clinical cases can be problematic and clinical signs can recur following apparently successful treatment. Although vaccines are available for Borreliosis in other countries, there are no licensed products available in the UK. Tick control is also an important component of management in endemic areas.

FURTHER READING:

Carter, S. D. *et al*. (1994). Borrelia burgdorferi infection in UK horses. Equine Veterinary Journal 26 (3), 187–190.

Chang, Y-F. *et al*. (2005). Antibiotic treatment of experimentally Borrelia burgdorferi infected ponies. Veterinary Microbiology, 107 (3-4), 285–294.

Johnson, A. L. *et al*. (2008). Validation of an In-Clinic Enzyme-Linked Immunosorbent Assay Kit for Diagnosis of Borrelia Burgdorferi Infection in Horses. Journal of Veterinary Diagnostic Investigation 20 (3), 321– 324.