BORRELIOSIS AND TICK-BORNE ENCEPHALITIS

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BORRELIOSIS

Borrelia spirochaetes
Borreliosis (Lyme borreliosis) is caused by spirochaete bacteria belonging to the Borrelia burgdorferi sensu lato complex e.g. Borrelia burgdorferi sensu stricto, Borrelia garinii, and Borrelia afzelii. B. burgdorferi bacteria are transmitted to dogs and cats through the bite of ticks and have been found in Ixodes ticks all over Europe and in parts of the US (1). The main reservoir hosts for B. burgdorferi are small wild mammals such as shrews, voles and mice.

Clinical findings, diagnosis and treatment
Lyme borrelioses have been diagnosed in animals such as horses, cattle, dogs and cats as well as in humans (2). Several clinical features have been associated with borreliosis in animals. Most of them involve the musculoskeletal system (3). The unique skin lesion termed erythema migrans often seen in human borreliosis has not been reported in animals infected with B. burgdorferi. Some dogs with borreliosis present with lameness of sudden onset, fever and lethargy. These dogs often have a local joint pain and exhibit swelling in one or more joints. Another kind of canine patient presents with signs similar to humans with neuroborreliosis e.g. painful manipulation of the head and neck. These dogs also have a temperature, are reluctant to move and show general pain not localised to the joints. The canine borreliosis patients most difficult to diagnose are the ones with intermittent, non-specific lameness, slight lethargy and with slightly elevated temperature (4).

Dogs with carditis and nephritis caused by borrelia infections have also been reported (5,6).

The Lyme borreliosis diagnosis is a clinical conclusion based on a combination of the history of tick exposure, compatible clinical signs and laboratory data. The serology available is only conclusive if a seroconversion or a four-fold titre change is shown. Positive serology is merely an indication of previous exposure to B. burgdorferi bacteria. Early in the disease the borrelia serology may be negative. In specialised laboratories and in research institutes immunoblot analyses may be available (7).

The growth of B. burgdorferi is inhibited in vitro by several antibiotics such as doxycycline, tetracycline, amoxycillin and ceftriaxone. The recommended treatment of Lyme borreliosis in dogs and cats is doxycycline 10 mg/kg once daily. In young animals amoxycillin is used as a first choice but also doxycycline may be used in severe cases. The risk of discoloration of the teeth is small when doxycycline is used compared to treatment with tetracycline.

TICK-BORNE ENCEPHALITIS

Tick-borne encephalitis (TBE) is a viral disease of the central nervous system. The disease is endemic in some areas in Europe and Asia and is locally known as Russian Spring-Summer Encephalitis (RSSE), FrühSommer-Meningoencephalitis (FSME), ‘Ryssjukan’, or ‘Kumlingsjukan’.

TBE virus
TBE virus (TBEV) belonging to the Flavivirus group causes TBE. Viruses of this group form different antigenic complexes. The main
complexes are TBE (e.g. TBEV, louping ill virus, Powassan virus), dengue (dengue virus) and Japanese encephalitis (e.g. West Nile virus).

TBEV is, as the name suggests, tick-borne and the main vector and reservoir for TBEV in Europe is the widespread tick species *Ixodes ricinus*.

Many animals serve as hosts for TBEV and/or *I. ricinus* ticks. Rodents such as *Apodemus* and *Clethrionomys* probably act as reservoir hosts for the virus. Bigger wild animals are considered to be incompetent for virus transmission but are hosts for the ticks. The louping ill (LI) virus is also spread by *I. ricinus* ticks. LI virus causes meningoencephalitis in sheep mainly in the UK but the disease is also seen in Spain, Greece, Turkey and Norway. Humans are susceptible to infection with LI virus but this occurs rarely. On the other hand TBEV seems to be primarily pathogenic for humans.

**TBE in animals**

Seropositivity has been found in nature in many different wild and also domesticated animals. In endemic areas in Switzerland a higher seroprevalence has been reported in dogs than in humans. The small numbers of clinical canine cases in these areas prove that the risk for a tick-infested dog to develop clinical manifest TBE is rather small.

Several experimental infections with TBEV have been performed in wolves, dogs and foxes. Conclusive evidence of pathogenicity or susceptibility in animals has not been stated as the animals in some studies showed symptoms while animals in other studies only became seropositive without apparent clinical signs.

Documented cases of clinical TBE in dogs have been reported from Austria, Switzerland, Germany and Sweden. Clinical TBE has also been reported in a horse and in an elk. No report of TBE in cats has been found.

**Clinical findings, diagnosis and treatment**

The incubation period is in most cases between 7 and 14 days. Clinical canine TBE is a febrile illness with multifocal neurological manifestations such as gait ataxia, uncoordinated movements, abnormal reflexes, convulsions, tremor, paresis, paraplegia, and cranial nerve deficits such as facial paresis. The neurological signs are often progressive and TBE may lead to death.

A TBE diagnosis can only be verified by means of laboratory techniques. Diagnostic serology is available at reference laboratories. A TBE infection evokes a life-long immunity.

Since there is no chemotherapy or specific treatment available targeting the TBE virus itself symptomatic treatment (maintenance of the water and electrolyte balances and so on) is required.

**TBE in Swedish dogs**

The first case of TBE in a Swedish dog was reported in 1960. Clinical cases are rare and nowadays between one and three cases of canine TBE are diagnosed in Sweden every year. In comparison, between 45 and 116 cases of human TBE were reported in Sweden per year during the 1990s. Approximately 10 canine serum samples per year are submitted to the Swedish National Veterinary Institute with a request for analyses regarding TBEV infection.

In a sero-survey performed in 1992, sera from 225 Swedish dogs with reported tick bites but with non-conclusive clinical signs were tested for TBEV antibodies. Of these 225 dogs, 18 (8%) were seropositive. Neurological clinical signs such as gait ataxia, tonic paresis and sensitivity to sounds were reported in 16 of these dogs. Fifteen of the 18 dogs recovered, four dogs after a week and 11 after two to three months. Three of the dogs were euthanased.

This sero-survey shows that TBE may be more common than anticipated in geographical areas where *I. ricinus* ticks are prevalent.

**REFERENCES**

7. Hoviunis, KE; Stark, LA; Bleumink-Pluyum, NM; van de Pol, I; Verbeek-de Kruijf, N; Rijpkema, SG; Schouls, LM; Houwers, DJ. Presence and distribution of *Borrelia burgdorferi sensu lato* species in internal organs and skin of naturally infected symptomatic and asymptomatic dogs, as detected by polymerase chain reaction.