Neosporosis in Dogs

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Introduction

*N. caninum* is a protozoan parasite that is a major pathogen of cattle and dogs, but occasionally causes clinical infections in goats, sheep, deer and horses, plus there are individual reports in rhinoceros and hyena. Dogs and more recently coyotes have been confirmed as definitive hosts and dogs may also be intermediate hosts. All the other species are intermediate hosts. In canines neosporosis is an important cause of meningoencephalomyelitis, polymyositis and polyradiculoneuritis in dogs throughout the world. Ascending paralysis of puppies is the most common presentation although a wide variety of clinical signs can be observed in dogs of all ages. The causative agent *Neospora caninum* is a protozoal parasite closely related to *Toxoplasma gondii*, with worldwide distribution. Prevalence of infection (not disease) varies from 0.5% to over 30%. To date no natural infections have been reported in cats or humans.

Life-Cycle and Epidemiology

Dogs and coyotes (confirmed) and other wildlife carnivores (suspected) serve as definitive hosts with fecal oocyst shedding. This shedding of oocysts is usually of short duration and few oocysts are shed compared to *Toxoplasma gondii*. In some circumstances however, shedding may continue for up to 4 months. Dogs / coyotes are infected when they feed on tissues of an infected intermediate host, normally cattle (bovine foetal membranes and raw meat). Most dogs infected this way shed oocysts but do not usually develop a systemic infection, may not seroconvert and rarely show clinical signs. A few animals may develop systemic infection and they become sub-clinically infected.

Trans-placental infection in these sub-clinically infected animals is common with many pups in these litters being infected, but abortion is rare. Successive litters from the same sub-clinically infected bitch may be born infected, but possibly at a reduced rate. However, postnatal infections following recrudescence of sub-clinical infections following an immunosuppressive incident (steroids, MLV vaccination, illness etc) are being reported with increased frequency.

Tachyzoites and tissue cyst forming bradyzoites compose the intermediate host parasite stages. Neospora tachyzoites occur in large cultures of 20-40 organisms and this accounts for the severe necrosis and florid inflammatory infiltrates commonly observed in skeletal/cardiac muscle and other visceral organs. This contrasts the usually less florid inflammatory reaction to *Toxoplasma gondii* tachyzoites, which are usually present in lesser numbers. Tissue cysts are found principally in the CNS but have also been reported in the skeletal muscles of naturally infected dogs and cattle. (Figure 1)
Clinical Signs

Dogs of any age may develop neosporosis although clinical disease is most commonly observed in puppies. It is currently not known whether infection also causes abortion and stillbirths in dogs. There is no known breed or sex predilection. Disease is reported in single pet households, as well as kennels and is seen in both urban and rural dogs.

Pups < 6 months = ascending lower motor neuron disease with rigid paralysis of the hind-limbs (rigidity is due to severe polymyositis with scarring) to tetra-paresis, radiculitis and encephalomyelitis. Puppies may also develop multi-organ involvement with acute death due to myocarditis.

Dogs > 6 months = lower motor neuron disease with flaccid paralysis (due to myelomalacia of recrudescent infection), regional or generalized myositis. Central nervous system (CNS) manifestations of meningitis, encephalomyelitis and cerebellitis (ataxia, gait abnormalities), plus systemic signs may be observed.

Most clinical cases are reported in dogs < 1 year of age, characterized by ascending paresis with rigid hyperextension of pelvic limbs. (Figure 2 and 3) In adults signs of multifocal CNS involvement are most common although polymyositis, dermatitis and multi-organ infection have all been described. Fever and inappetance are rare with most dogs remaining alert and bright until the later stages of disease when forelimb weakness and difficulty in swallowing and breathing develop.

Diagnosis

Live animal

- Serology: the indirect fluorescent antibody test (IFAT) is the most commonly employed serological test measuring antibodies to *N. caninum*. Titres > 1:50 indicate exposure but not necessarily disease. Titres > 1:800 in a dog with clinical signs is strong supporting evidence of neosporosis.
- PCR on cerebrospinal fluid (CSF) is being used with increased frequency as a confirmatory diagnostic test.
- Immunohistochemistry (IHC) on muscle biopsies or skin biopsies (in cases with skin involvement), collected into 10% buffered formalin, is a confirmatory test.
- Fecal floatation procedures have very poor sensitivity due to short duration of fecal shedding and low numbers of oocysts shed.

Post Mortem

- Histopathology enables visualization of the characteristic pathology associated with *N. caninum*. (Figure 4)
- IHC on formalin-fixed tissues (muscle, spinal cord, brain) are used to confirm the diagnosis. (Figure 5)

Treatment

- Clindamycin 11-22mg/kg BID or
- Potentiated sulphonamides (15mg/kg BID) + Pyrimethamine (Daraprim 25mg® – 1mg/kg once daily)
Treatment should be continued until the animal has fully recovered or until no further clinical improvement is observed (2-9 weeks). Supportive treatment (non-steroidal anti-inflammatory drugs (NSAIDs), low dose corticosteroids, nursing care) are also beneficial. About 50% of the appropriately treated dogs will make a full or functional recovery. Some dogs may be left with an unusual gait or some muscle atrophy. Rigid hyperextension of the hind-limbs are the cases least likely to be reversed, while per-acute and very chronic cases are the least likely to respond. Relapses may occur but in general these infections respond well to a further short course of therapy. Mildly affected dogs may make a spontaneous recovery.

Control and Prevention

Trans-placental transmission is the most important and most common route of infection of puppies. The number of puppies infected per litter varies from none to all with an average of about 20% of the litter. Less than 50% of infected pups will develop clinical signs. Transmission can occur repeatedly over several infected litters. Preventative treatment of bitches during pregnancy or of seropositive apparently normal littermates of clinical cases, to block pre-natal infection of pups, has proven unsuccessful. Therefore, bitches that produce infected litters should be sterilized and removed from any breeding program. Bitches with high antibody titres (>1:800 on IFAT) are more likely to produce infected pups. Serological screening of bitches in a breeding facility can be used to identify high risk bitches which should then be sterilized and removed from the breeding program.

Post natal infection occurs, although it is still not known whether disease in adult dogs is due to a recent infection or a relapse of a congenital infection. Canines frequently acquire the infection through ingestion of infected material especially from bovine fetal membranes or raw meat. This results in oocyst shedding but these dogs do not normally develop a systemic infection. In breeding facilities all meat fed should be thoroughly cooked, any access to bovine placentas must be avoided and fecal management to reduce the risk of fecal contamination of feed and water sources should be practiced.