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Infectious Diseases Dentistry

Neosporosis in dogs

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ETIOLOGY

Neospora caninum is a coccidian parasite that had been confused with T. gondii until 1988. The domestic dog (Canis familiaris) and the coyote (Canis latrans) are its definitive hosts and cattle, sheep, goats, horses, deer, and several other animals are its intermediate host. As yet, there is no evidence for human infections. Herbivores likely become infected from ingesting oocysts shed by the definitive host and by subclinical congenital infection from transplacental transmission. Tachyzoites and tissue cysts are tissue stages found in all hosts whereas oocysts are excreted in canine feces. Tachyzoites are 5 to 7 μm in diameter are found mainly in neural cells. Oocysts are shed unsporulated in canine feces and they sporulate outside the body.

CLINICAL FINDINGS

Naturally occurring infections have been reported worldwide in dogs and it is likely that many dogs diagnosed with toxoplasmosis before 1988 actually had neosporosis. Clinical signs predominate in neural and muscular tissues but may also include skin, lungs, liver, heart or other tissues. Both pups and adult dogs are clinically affected, and the infections can be transmitted congenitally. The most severe and frequent infections have been in young (<6 months) dogs that presented with ascending paralysis of the limbs. In the youngest pups, signs are often noticed beginning at 3 to 9 weeks of age. Features that distinguish neosporosis from other forms of paralysis are gradual muscle atrophy and stiffness, usually as an ascending paralysis; the pelvic limbs are more severely affected than the thoracic limbs. Paralysis progresses to rigid contracture of the muscles of the affected limb. This arthrogryposis is a result of the scar formation in the muscles from lower motor neuron damage and myositis. In some pups, joint deformation and genu recurvatum may develop. Cervical weakness, dysphagia, megaesophagus, and ultimately death can occur. In some dogs, the progression may become static. Dogs do not develop severe intracranial manifestations and maintain alert attitudes. They can survive for months with hand feeding and care but remain paralyzed with associated complications. Older dogs, which are less commonly affected, often have signs of multifocal CNS involvement or polymyositis; less common manifestations result from myocarditis, dermatitis, pneumonia, or multifocal dissemination. Death can occur in dogs of any age.

DIAGNOSIS
Hematologic and biochemical findings have been variable, depending on the organ system of involvement. With muscle disease, creatine kinase and AST activities have been increased. Serum ALT and alkaline phosphatase activities are increased in dogs that develop hepatic inflammation. CSF abnormalities have included mild increases in protein (>20 but <150 mg/dl) and nucleated cell (>10 but <100 cells/dl) concentrations. Differential leukocyte counts included lymphocytes, monocytes and macrophages, neutrophils, and eosinophils in decreasing numbers. CSF results can be within reference limits in some dogs. Fibrillation potentials, positive sharp waves, and occasional repetitive discharges. Nerve conduction velocities may be reduced in the most severely affected limbs, especially proximally, but they are often within reference range. Low evoked action potentials may be found with myositis.

Demonstrating serum antibodies to *N. caninum* can help confirm the diagnosis of neosporosis. Serum is reacted with cell-cultured *N. caninum*. Serum indirect FA titers can vary between laboratories. CSF can be tested, but titers are of lesser magnitude. Indirect FA IgG titers in most species increase 1 to 2 weeks after infection. There is no correlation between magnitude of titer and clinical status; severe neosporosis has been diagnosed in congenitally-infected pups that have low titers. There are several ELISA tests to detect antibodies to *N. caninum*.

Antibodies to *T. gondii* do not cross-react with *N. caninum*, at dilutions of 1:50 or less, so that serum or CSF antibody titers to *T. gondii in Neospora*-infected dogs are negative. Slight cross-reactivity with sera from dogs infected with *Babesia gibsoni* but not *B. canis* has been observed. Some cross-reactivity has been observed with ELISA testing when crude extracts are used as antigens.

*N. caninum* may be found in CSF or tissue aspirates and biopsies of some dogs and may be detected with any material used to stain blood films. Biopsy of affected muscle may yield a definitive diagnosis when organisms are detected. Immunohistochemical staining and PCR can help to distinguish *Neospora* from other related parasites.

Gross lesions include multifocal streaks of necrosis, fibrosis, and mineralization of striated muscles, especially the diaphragm. Hepatomegaly, pneumonia, and discoloration of brain or spinal cord tissues may be apparent on cut section.

Nonsuppurative encephalomyelitis, polyradiculoneuritis, ganglionitis, myositis (of all striated muscles), and myofibrosis are the predominant histologic findings. The encephalomyelitis is characterized by inflammation, axonal degeneration, and formation of glial nodules in gray and white matter. Finding tachyzoites in lesions is indicative of etiology. *N. caninum* appears to induce more inflammation than *T. gondii* and has been found to cause severe phlebitis and dermatitis. Nonsuppurative myocarditis, pneumonia, and hepatitis are commonly present as subclinical lesions. Lesions caused by *N. caninum* are similar to those by *T. gondii* or to granulomatous meningoencephalitis. Confirmation, therefore, requires serologic or immunohistochemical methods.

**THERAPY**

Information on effective therapy for this disease is limited. Clindamycin, sulfadiazine, and pyrimethamine alone or in combination have been administered to treat canine neosporosis. However, clinical improvement is not likely in the presence of muscle contracture or rapidly advancing paralysis. To reduce the chance of illness, all dogs in an affected litter should be treated as soon as the diagnosis is made in one littermate. Older (>16 weeks) puppies and adult dogs respond better to treatment. There is no known therapy to prevent a bitch from transmitting infection to her pups.

**PREVENTION**

In dogs, *N. caninum* can be transmitted repeatedly through successive litters and litters of their progeny. This should be considered when planning the breeding of *Neospora*-infected bitches. Dogs should not be fed uncooked meat, especially beef. There is no vaccine to combat neosporosis. No drugs are known to prevent transplacental transmission.