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FELINE TOXOPLASMOSIS

Toxoplasma gondii infection is common in cats and people throughout the world. The seroprevalence in cats and people varies with the region and country, but is commonly 30-40%. Once infected with *T. gondii*, the organism probably remains in tissues for life. Fatal and serious clinical syndromes can occur in cats infected by *T. gondii*.

CLINICAL PRESENTATION

Intestinal toxoplasmosis. Cats are commonly infected by ingestion of *T. gondii* bradyzoites in tissues of prey species. The intestinal cycle is completed in about 10 days or less, and then oocyst shedding almost never reoccurs. Some experimentally inoculated cats develop self-limiting, small bowel diarrhea; this is presumed to be from enteroepithelial replication of the organism. However, detection of *T. gondii* oocysts in feces is rarely reported in naturally exposed cats with or without clinical disease. Thus, the intestinal phase of feline toxoplasmosis appear relatively unimportant clinically. By the time most cats seroconvert and become antibody positive, the oocyst shedding period has ceased. A seropositive cat is therefore a minimal zoonotic risk.

Other forms of toxoplasmosis. Toxoplasmosis induced transplacentally or by suckling in kittens is often clinically severe as a result of overwhelming tachyzoite replication. Anorexia, vocalization, signs of depression, lethargy, hypothermia, dyspnea, and sudden death are common clinical presentations in kittens born alive. Clinical signs develop within 2 days and as late as 25 days after birth. Necrosis with infiltrates of macrophages in

lung, liver, cardiac, muscle, central nervous system, ocular, adrenal and renal tissues are characteristic findings. Clinical findings for 100 cats with histologically confirmed toxoplasmosis, diagnosed between 1952 and 1990, were as follows: 36 cats had generalized toxoplasmosis; principal lesions for the rest were pulmonary (26), abdominal (16), hepatic (12), cardiac (12), neonatal (9), neurologic (7), and pancreatic (1). Fever (73%); dyspnea, polypnea, anorexia, lethargy, icterus (24%); abdominal discomfort; and signs of central nervous system disorder and ocular inflammation were common.

Clinical toxoplasmosis of a more chronic course has been suspected in a number of cats. The primary clinical findings include anterior or posterior uveitis, fever, muscle hyperesthesia, weight loss, anorexia, seizures, and ataxia. Fever and muscle hyperesthesia usually resolve quickly following treatment and rarely reoccur. Uveitis can be unilateral or bilateral; severe anterior segment inflammation can occur with hypopyon, flare, and keratic precipitates which make visualization of the posterior segment difficult. Chorioretinitis can be unifocal or multifocal, and punctate or diffuse. Lens luxation and secondary glaucoma are common sequelae. Cats with uveitis are commonly seropositive for *T. gondii* and other potential ocular pathogens like FeLV, FIV, and FIP, which may complicate serologic and clinical diagnoses. Ocular and central nervous system disease may occur alone or concurrently and can be detected in cats without polysystemic signs of disease.

FELINE TOXOPLASMOSIS

(CONT'D.)

DIAGNOSIS

Hematologic, biochemical and urinalysis findings. There are no pathognomonic laboratory findings associated with clinical toxoplasmosis. However, if the clinical history is consistent, the following laboratory abnormalities raise the suspicion of toxoplasmosis: nonregenerative anemia, neutrophilia or neutropenia, lymphocytosis, monocytosis, and eosinophilia; increased creatinine kinase, alanine aminotransferase, alkaline phosphatase and lipase activities; and hyperproteinemia, hyperbilirubinemia, proteinuria and bilirubinuria.

Radiographic findings. Interstitial and alveolar radiographic patterns are detected with pulmonic toxoplasmosis but pleural effusion is rarely seen. Abdominal radiographic findings are non-specific but can include homogenous increased density due to peritoneal effusion, hepatomegaly, lymphadenopathy, and intestinal masses, or loss of contrast in the cranial right quadrant of the abdomen due to pancreatitis. In cats with central nervous system involvement, lesions are potentially detectable by myelography, CT scan, or MRI.

Cytology and cerebrospinal fluid analyses. In a series of cats with suspected CNS toxoplasmosis, CSF protein levels ranged from normal - 149 mg/dL and nucleated cell counts ranged from < 5 - 28 cells/cmm. Small mononuclear cells were the predominant leukocytes. Cats with these findings should be screened for *T. gondii* infection.

Serology. The combination of serum *T. gondii* IgM and IgG antibody testing can be used to aid in the diagnosis of feline toxoplasmosis. Experimentally infected healthy cats have detectable serum *T. gondii*-specific IgM titers within 2-4 weeks following inoculation with *T. gondii*, but IgM

usually becomes undetectable within 16 weeks post-infection. Since this antibody class is present for a short duration in serum, only 1.2% of healthy cats are found to be *T. gondii* IgM positive. In contrast, detectable *T. gondii* IgM titers were present in serum of 93.3% of cats in a study of clinical toxoplasmosis, whereas *T. gondii* IgG titers were detected only in 60% of them. Thus, *T. gondii* IgM antibody class appears to correlate to clinical toxoplasmosis better than IgG.

Persistent *T. gondii* IgM titers (> 16 weeks) have been documented in healthy cats coinfecting with FIV. In some chronically infected cats, IgM antibody can be detected again after repeat inoculation with *T. gondii*, coinfection with the Petaluma isolate of FIV and administration of glucocorticoids. Therefore, IgM titers cannot accurately predict when a cat sheds oocysts and should not be used alone to definitively document clinical toxoplasmosis.

Serum *T. gondii*-specific IgG can be detected by ELISA in most healthy experimentally inoculated cats within 3-4 weeks post-infection. These IgG antibody titers can be detected for at least 6 years after infection and probably persist for life. Single, high *T. gondii* IgG titers may reflect recent or active infection, as healthy cats can have titers > 10,000 6 years after experimentally induced toxoplasmosis. Demonstration of an increasing IgG titer may reflect recent or active disease, although in experimentally infected cats, time span from the first detectable IgG titer to maximal IgG titer was about 2-3 weeks. Many cats with clinical toxoplasmosis have chronic, mild clinical signs and so may not be evaluated serologically until after IgG antibody titers have reached maximal values. In humans and cats with reactivation of chronic toxoplasmosis, *T. gondii* IgG titers

only rarely increase. Thus, failure to document increasing IgG titers does not exclude the diagnosis of clinical toxoplasmosis.

Definitive diagnosis of clinical feline toxoplasmosis requires documentation of the organism in tissues or effusions in association with inflammation. This is usually achieved at necropsy in cats with overwhelming tachyzoite replication.

Since *T. gondii*-specific antibodies can be detected in the serum of normal cats as well as those with clinical signs of disease, it is impossible to make an antemortem diagnosis of clinical toxoplasmosis based on these tests alone. The following combination can be used to make a presumptive antemortem diagnosis of clinical feline toxoplasmosis:

- demonstration of specific antibodies in serum which document exposure to *T. gondii*;
- demonstration of *T. gondii* IgM titer > 1:64 or a 4-fold or greater increase in *T. gondii* IgG titer which suggests recent or active infection;
- clinical signs of disease referable to toxoplasmosis;
- exclusion of other common causes of this clinical syndrome;
- positive response to appropriate treatment.

TREATMENT

Cats with suspected clinical toxoplasmosis should be administered supportive care as needed. Clindamycin hydrochloride administered at 12 mg/kg PO, q 12 hr for 4 weeks or trimethoprim-sulfonamide administered at 15 mg/kg, PO, q 12 hr for 4 weeks have been used most frequently. Axithromycin and doxycycline are potentially effective alternate treatments.

[Contributed by Dr. Michael R. Lappin, Colorado State University College of Veterinary Medicine, Ft. Collins, CO.]