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An Overview of Feline Heartworm Disease

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An Overview of Feline Heartworm Disease

Heartworm disease was previously considered a serious clinical problem only in dogs. Recently, however, the incidence of feline heartworm disease appears to be increasing. Factors contributing to this include increased awareness by veterinarians, a wide host preference by the vector, and the realization that host specificity of *Dirofilaria immitis* was less than previously thought.¹

The diagnosis of feline heartworm disease can be challenging. Several diagnostic tools are available, however. The purpose of this report is to present an overview of feline heartworm disease with special emphasis on its diagnosis and treatment.

Development

Cats can provide an adequate environment for *D. immitis* to develop to maturity; however, the cat is not an ideal host for the parasite. Studies indicate that cats are more resistant to adult infection, and that they rarely permit heartworm reproduction. After being exposed to infective *D. immitis* larvae, the infection rate in cats ranges from 2–30%, whereas that in dogs is 36–65% under similar conditions.²,³ The average adult worm burden in the cat usually ranges from 1–9, which is less than in dogs.²,³ Also, the life span of *D. immitis* adults in cats appears to be less than two years, which is considerably less than the life span in dogs (five years).²,³ Feline leukemia virus infection does not appear to be a predisposing condition.

Microfilaremia is seen in fewer than 20% of cats infected with *D. immitis*.¹,⁷ The absence of microfilaremia can result from a unisex infection, infertile worms, or possibly immune mechanisms.¹,⁵ The prepatent period in cats that do develop microfilaria is much longer (average = 8 months) than in dogs.³ The period of microfilaremia is short and the levels are often low (<2 microfilaria/ml venous blood).³ The lack of microfilaremia in most cases makes diagnosis difficult by conventional microfilarial screens.

Pathology

The cardiopulmonary changes seen in cats with heartworm disease are quite similar to those seen in dogs. Histopathologically, villous endarteritis of the larger pulmonary arteries is seen. The intima can become so proliferative that the vessel lumen is occluded.⁴ Hyperplastic smooth muscle cells cause thickening of the arterial media. Grossly, enlarged caudal pulmonary arteries are a prominent finding.

Clinical Signs

The clinical signs associated with feline heartworm disease can be quite variable. Cats may have acute cardiopulmonary collapse, or they may be presented with a history of chronic problems. Many cats infected with *D. immitis* are asymptomatic and heartworms can be an incidental finding at necropsy.

The most common clinical signs seen are acute or chronic episodes of coughing or dyspnea. Emesis, lethargy, and anorexia are other common signs. Usually, an infected cat will exhibit either respiratory or gastrointestinal signs, rarely both.

The coughing is often intermittent, but it may develop into severe paroxysms. Occasionally,
hemoptysis occurs. Corticosteroids tend to alleviate the coughing by reducing the inflammatory reaction in the pulmonary parenchyma.\(^2\) It is believed that the dyspnea is due to acute pulmonary embolism.\(^2,3\)

Vomiting, when it occurs, is usually intermittent and often tends to be postprandial. Heartworm disease should be considered a differential diagnosis in cats presented with chronic emesis, especially in heartworm endemic areas. Neurologic signs have been seen in cats with aberrant worm migration.\(^2,3\)

The clinical signs of heartworm disease are quite similar to those caused by feline asthma, *Aleurostrongylus abstrusus* infection, *Paragonimus kellicotti* infection, or cardiomyopathy (although cough is rare with heart failure).

Sudden death may occur. These sudden deaths have been attributed to acute pulmonary arterial infarction with resultant circulatory and pulmonary failure.\(^2,3\) Therefore, cats that die suddenly, especially in heartworm endemic areas, should be examined for heartworm disease.

**Diagnosis**

The minimum data base should include a thorough history, physical examination, complete blood count, a microfilarial concentration test, multiple fecal exams, thoracic radiographs, tracheal wash, electrocardiogram, and serologic tests. Sometimes a non-selective angiogram is also helpful.

The physical exam is usually unremarkable. Harsh lung sounds are the most common abnormality. Occasionally, a cardiac gallop rhythm or systolic murmur may be heard upon thoracic auscultation.

Changes seen on the complete blood count may include a mild, nonregenerative anemia; eosinophilia, and basophilia. Only about one third of infected cats will exhibit a peripheral eosinophilia which occurs sporadically four to seven months post infection.\(^2,3\) Although supportive of a diagnosis, the presence of an eosinophilia or basophilia is not pathognomonic for heartworm disease.

To help eliminate lung worms as a diagnosis, multiple fecals should be done to look for the eggs or larva of *A. abstrusus* or for the operculated eggs of *P. kellicotti*. A Baerman floatation test may help concentrate the larvae of *A. abstrusus*.

Although microfilaremia is uncommon, a microfilarial concentration test such as the Knott’s should be performed. Repetitive testing with a larger volume (5 mls) may be beneficial.\(^2\) A negative blood test does not rule out heartworm disease, but a positive test is diagnostic.

Heartworm infected cats rarely have significant electrocardiographic changes. Occasionally, electrocardiographic signs of right heart enlargement may be present.

A tracheal wash may contain numerous eosinophils; this is compatible with heartworm disease, the parasitic lung diseases, and feline asthma. Finding eggs or larvae in the tracheal wash is diagnostic for the parasitic lung diseases. The greatest number of eosinophils in the wash are usually found at four to seven months post heartworm infection.\(^2\) Some tracheal washes contain cells that are more typical of a chronic inflammatory response.

Thoracic radiographs are essential in the diagnostic workup of a coughing or dyspneic cat. The radiographic changes seen in the feline with heartworm disease are quite similar to those seen in the canine. The changes seen include enlarged tortuous pulmonary arteries, especially the caudal lobar arteries; parenchymal interstitial densities, and sometimes right ventricular enlargement. Unlike in dogs, enlargement of the main pulmonary artery is rarely seen since the main pulmonary artery segment lies more on the midline in cats and is obscured by mediastinal structures.

Nonselective angiography is a simple and usually safe procedure for supporting a diagnosis of heartworm disease. Contrast material (4 to 6 mls of a water soluble iodide) is injected into the cephalic or jugular vein, and radiographic exposures are made five to six seconds after the injection.\(^2\) On the ventrodorsal view, the classic enlarged caudal lobar arteries are easily seen. Linear filling defects in the vessels represent adult worms. The radiographic changes seen may not reflect the severity of clinical signs nor be predictive of a post-adulticide reaction.

In canine occult dirofilariasis, detection of antibodies against the microfilarial cuticular antigen by an IFA test has been used extensively.\(^6\) The test has been used in cats, and if positive, is diagnostic. A negative test does not rule out heartworm disease since microfilariaemia can also be due to a unisex infection, a prepatent infection, or due to an infection with sterile worms.\(^6\)

An ELISA test for detection of adult antigen in circulation (Filarocheck, by Mallinckrodt) is often positive in cats infected with *D. immitis*. The antigen detected arises from the reproductive tract of adult female worms. Therefore, tests on cats with sexually immature worms are negative.\(^2\)
positive test would be considered diagnostic, however a negative test does not rule out heartworm disease.

**Therapy**

Treatment of *D. immitis* adults can be done with intravenous thiacetarsamide sodium (2.2 mg/kg B.I.D. for two days).

The drug is well tolerated by most cats; however, the risk of post-adulticidal complications tends to be more severe than in dogs. Pulmonary thromboembolism causes the post therapy complications. Acute lung disease and sudden death can occur within the first two weeks post adulticide. Pulmonary emboli usually involve the caudal lung lobes and result in dyspnea, hemoptysis, and collapse. Oxygen therapy during this time may be beneficial. Corticosteroids (2.2-4.4 mg/kg of prednisolone T.I.D.) and supportive therapy may give the patient some relief.

The use of aspirin in feline heartworm disease appears to be indicated to help prevent platelet aggregation in post adulticidal reactions and to help reduce the severe endarteritis. Platelet aggregation is inhibited when aspirin is used at a dose of 25 mg/kg twice a week.

Since heartworm disease in cats is typically occult in nature, microfilaricide treatment is usually not indicated. If necessary, Dithiazanine iodide and levamisole have both been used with satisfactory results.

Since cats are a relatively resistant host, the use of diethylcarbamazine citrate as a preventative is not currently recommended.

Heartworm disease should be considered as a differential in cats presented for coughing, dyspnea, or intermittent vomiting. This is particularly important in heartworm endemic areas. Although reports indicate that clinical and radiographic signs can resolve with thiacetarsamide treatment, it is important to emphasize the possibility of severe post-adulticidal reactions.

**REFERENCES**