Dirofilaria immitis and D. repens in dog and cat and human infections

Editors
Claudio Genchi, Laura Rinaldi, Giuseppe Cringoli

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Heartworm (*Dirofilaria immitis*) disease in cats

Luigi Venco
Cat is considered a susceptible but not ideal host for *Dirofilaria immitis*. Increased host resistance is reflected by the relatively low adult worm burden in natural infections (cats generally harbour 1 to 8 worms with 2 to 4 worms being the usual burden, Genchi et al., 1992), the low number of heartworms that develop after experimental inoculation with infective larvae, the prolonged pre-patent period (7-8 months), the low level and short duration of microfilaremia, and the short life span of adult worms (2-3 years) (Dillon, 1984; Calvert, 1989; McCall et al., 1992; Atkins et al., 1995).

In cats, changes in pulmonary arteries and lungs after infection seem to be similar to those found in dogs, but right cardiac chambers well bear pulmonary hypertension and right cardiac heart failure is an unusual finding (Dillon et al., 1995; Atkins et al., 1995).

In cats, the clinical presentation is quite different than in the canine counterpart. Most cats seem to well bear the infection for long time. These cats may have a spontaneous self-cure due to the natural death of parasites without any kind of clinical signs or may suddenly show dramatic acute symptoms. Respiratory signs as coughing, dyspnoea, haemoptysis are usually seen but also vomiting frequently occurs. Sudden death in apparently healthy cats is furthermore not rarely observed (McCall et al., 1994; Holmes, 1995; Atkins et al., 1995) (Fig. 1). Chronic symptoms including coughing, vomiting, diarrhoea, weight loss can be less frequently observed.

On the contrary than in dogs, symptomatology related to right ventricular heart failure is not considered consistent with heartworm infection in cats.

**Fig. 1.** Two adult female heartworms in a cat that experienced sudden death.

The onset of symptoms in most cases seems to be related to the natural death of parasites or to the first arriving of L5 heartworms in the pulmonary arteries.

**Diagnosis**

**Blood test for microfilariae**

As microfilaremia in cats is unlikely, sensitivity of test for detection of circulating microfilariae is very low despite specificity is considered 100% as in dogs (Atkins et al., 1995).

**Blood test for adult antigens**

Test detecting adult female heartworm antigens can provide a definitive proof of infections in cats because of the very high specificity. Nevertheless because the worm burden is usually very light in cats, infections caused only by male heartworms are not infrequent and symptomatology may be frequently due to immature worms, these test yield false negative result in a large average. A negative test can not therefore considered sufficient to rule out the infection (Atkins et al., 1995).
**Blood test for antibodies to adult heartworm**

Due to the low sensitivity of tests for circulating microfilariae and adult antigens in cats, test for detection of antibodies to adult heartworm can be useful used (Atkins et al., 1995; Prieto et al., 1997; Genchi et al., 1998). This kind of test has high sensitivity but not complete specificity because of cross reactivity with other parasites or antibodies to abortive infections. Consequently antibody tests should be interpreted carefully, taking other relevant clinical information into consideration.

**Thoracic radiographs**

Thoracic radiography is an important tool for the diagnosis of feline heartworm disease. Despite thoracic abnormalities in few cases are absent or transient (Selcer et al., 1996), typical findings as enlarged peripheral branches of the pulmonary arteries accompanied by varying degrees of pulmonary parenchymal disease are strongly consistent with heartworm infection (Fig. 2). Enlargement of the main pulmonary artery cannot be observed because this tract of artery is obscured by cardiac silhouette. Right-sided cardiomegaly is not considered a typical finding in cat.

**Non selective angiocardiography**

Non selective angiocardiography is useful in visualizing the gross morphology of the pulmonary arteries. Seldom the heartworms can be seen as negative filling defects within opacified arteries (Atkins et al., 1995).

**Electrocardiography**

Heartworm infection does not involve right cardiac chambers. Consequently electrocardiography cannot provide useful information in infected cats.

**Echocardiography**

Cardiac ultrasound allows the direct visualization of the parasites in right atrium and ventricle, main pulmonary artery and proximal tract of both its peripheral branches (Fig. 3). Specificity is virtually 100% and sensi-
Heartworm disease in cats seems to be very high (Venco et al., 1998, 1999b) because the portion of caudal pulmonary arteries that can not be thoroughly interrogate because of the acoustic impedance of the air inflated lungs is very short when compared with the length of the adult parasite. Based on these considerations, cardiac ultrasonography should be always performed when heartworm infection is suspected.

**Transtracheal lavage**

The presence of eosinophiles in a tracheal wash, with or without eosinophilia, may be noted 4 to 7 months after infection but this findings is not specific and infection with other pulmonary parasites (*Paragonimus kellicotti*, *Aelurostrongylus abstrusus*) and allergic pneumonitis should be ruled out (Atkins et al., 1995).

**Therapy**

Diminishing doses of prednisone are advised in cats in order to relief respiratory distress. The dosage is 2 mg/kg daily initially, then declining to 0.5 mg/kg every other day for two weeks, and then discontinuing treatment after an additional two weeks (Atkins et al., 1995).

If crisis is due to embolization of dead worms high doses of prednisone (1-2 mg/kg 3 times a day) are recommended (Dillon, 1986).

In previous studies, when thiacetarsamide was the only arsenical available compound, some treatment regimen was attempt against adult worms. The same dosage and regimen used in treating dogs, 2.2 mg/kg twice daily for two days, was used for cats. The results were debatable. Turner et al. (1989) reported some toxicity in heartworm naive cats, but later reports showed that thiacetarsamide delivered to normal cats produced no respiratory distress or altered the body temperature (Dillon et al., 1992). However a large average heartworm infected cats develop acute respiratory distress or sudden death in the post-treatment period. These effects seem to be due to embolization associated with worm death (Dillon et al., 1992).

The organical arsenical melarsomine dihydor chloride is the only available compound now on the market, but there is insufficient experience about its use in cats until now. Furthermore, few data suggests that melarsomine is toxic to cats at dosages >3.5 mg/kg. Ivermectin at 24 µg/kg monthly given for 2 years has been reported to reduce worm burdens as compared to untreated cats. Since cats usually harbour low worm burdens and the main problem is the reaction that arise when the worms die (and not the worm mass by itself) this reaction could probably occur even when the ivermectin-treated worms die, but its intensity is unknown.

Anyway, there are no studies suggesting that medical adulticidal therapy increases the survival rate of heartworm naturally infected cats (Knight et al., 2002). Due to these reasons, macrofilaricidal treatment is not advised in cats unless in selected cases.

In case of caval syndrome or when a heavy worm burden is visualized by echocardiography in right cardiac chambers, surgery may be attempted. Worm can be extracted via jugular vein using thin alligator forceps, horse hair brush or basket catheters (Glaus et al., 1995; Borgarelli et al., 1997; Venco et
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al., 1999b). Because of the small size of the feline heart pulmonary arteries cannot be accessed. Special care must be taken during the heartworm removal because traumatic dissection of a worm may result in circulatory collapse and death (Venco et al., 1999b).

References


