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FELINE health topics

for veterinarians

inside

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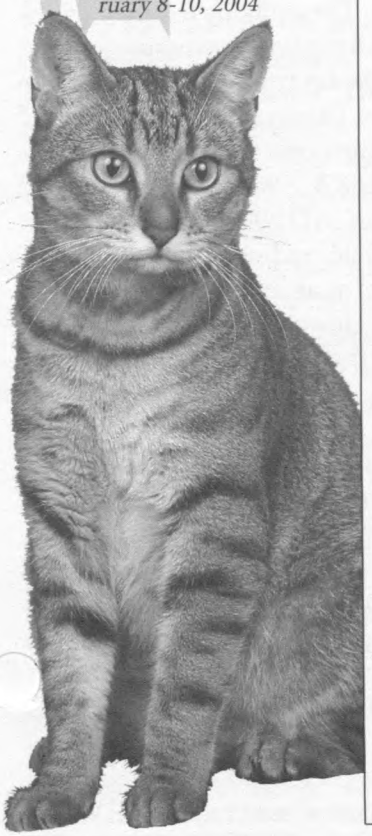

Feline Heartworm Disease: What's Different in 2004?

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Etiology and Pathophysiology

The domestic cat, though an atypical host, can be parasitized by *Dirofilaria immitis* with resultant heartworm disease (HWD). The clinical manifestations of the disease are different and more severe in this species, but the infection rate is only five to 20 percent of that of the dog.[1] Experimental infection of the cat is more difficult than in the dog; <25 percent of L3 reach adulthood. This resistance is also reflected in natural infections, in which feline heartworm burdens are usually less than 10, and typically only 2-4 worms.[2] Other indications of the cat's inherent resistance to this parasite are a shortened period of worm patency, high frequency of amicrofilaremia or low microfilaria counts, and shortened life span of adult heartworms (2-3 years).[2] Nevertheless, studies have shown a prevalence as high as 14 percent in shelter cats[1] and a study performed at North Carolina State University (NCSU) revealed HWD in nine percent of cats presented with cardiorespiratory signs.[3] Furthermore, antibody testing showed 26 percent of 100 of these cats to have been exposed to HW.[3] Similar to dogs, the male cat is at higher risk for heartworm infection (HWI) than is the female. Aberrant worm migration appears to be a greater problem in cats than in dogs.

The pulmonary arterial response to adult heartworms is more severe than that of the dog, although pulmonary hypertension has infrequently been reported. Dillon demonstrated pulmonary enlargement within one week of transplantation of adults, suggesting an intense host-parasite interaction.[4] A severe myointimal and eosinophilic response produces pulmonary vascular narrowing and tortuosity, thrombosis, and possibly, hypertension.[5] Because the feline pulmonary artery tree is smaller than that of the dog and has less collateral circulation, embolization, even with small numbers of worms, produces disastrous results with infarction and even death. Although uncommon, cor pulmonale and right heart failure can be associated with chronic feline HWD and is manifested by pleural effusion (hydro- or chylothorax) and/or ascites. The lung per se also is insulted by HWI, with eosinophilic infiltrates in the lung parenchyma (pneumonitis), pulmonary vasculature, and air spaces. The pulmonary vessels may leak plasma, producing pulmonary edema (ARDS?) and type II cells proliferate, both potentially altering O_2 diffusion. The end result is diminished pulmonary function, hypoxemia, dyspnea, and cough.

Clinical Signs

Cats with HWI may be asymptomatic and, when present, clinical manifestations may be either peracute/acute or chronic.[3,4,6-8] Acute or peracute presentation is usually due to worm embolization or aberrant migration and signs variably include salivation, tachycardia, shock, dyspnea, hemoptysis, vomiting and diarrhea, syncope, dementia, ataxia, circling, head tilt,

blindness, seizures, and death. Postmortem examination often reveals pulmonary infarction with congestion and edema. More commonly, the onset of signs is less acute (chronic form). Reported historical findings in chronic feline HWD include anorexia, weight loss, lethargy, exercise intolerance, signs of right heart failure (pleural effusion; rare), cough, dyspnea, and vomiting. We have found dyspnea and cough to be relatively consistent findings, and when present, should cause suspicion of HWD in endemic areas.[8]

In an NCSU report of 50 natural cases of feline heartworm infection, presenting signs were most commonly related to the respiratory system (32 cats; 64 percent), with dyspnea (24 cats; 48 percent) being most often noted, followed by cough (19 cats; 38 percent), and wheezing.[8] Vomiting was reported in 17 (38 percent) cats and was noted frequently in eight (16 percent). Five (10 percent) heartworm-infected cats were reported to have exhibited vomiting without concurrent respiratory signs and vomiting was a presenting sign in seven (14 percent). Neurological signs (including collapse or syncope) which were described in five (10 percent) were reported in seven (14 percent) cats. Five (10 percent) of the cats were dead at the time of presentation. Murmurs were infrequently noted in cats that did not have concurrent heart disease, independent of heartworm infection. Heart failure was present in one cat but this cat had concurrent hypertrophic cardiomyopathy. Heartworm infection was considered to be an incidental finding in 14 (28 percent) of the cats in this study.

Physical examination is often unrewarding although a murmur, gallop, and/or diminished or adventitial lung sounds may be audible. In addition, cats may be thin and/or dyspneic. If heart failure is present, jugular venous distension, dyspnea, and rarely, ascites are detected.

Diagnosis

The diagnosis of HWI/HWD in cats poses a unique and problematic set of issues.[4] First, the clinical signs are often quite different from those of the dog. In addition, the overall incidence in cats is low, so suspicion is lessened; eosinophilia is transient or absent; electrocardiographic findings are minimal; and most cats are amicrofilaremic.

Immunodiagnostic methods are also imperfect in cats because of the low worm burdens (range=1-12, mean=3) and hence, antigenic load. In a recent study, ELISA antigen tests were positive on sera from 36-93 percent of 31 cats harboring one to seven female HW, with sensitivity increasing as female worm burden increased.[9] Cats with male worms only were not detected as positive. Therefore, false negative tests occur frequently, depending on the test used, maturity and gender of worms, and worm burden. All tests were, however, virtually 100 percent specific. It is important to realize that infection with signs may be present prior to the presence of detectable antigen (from gravid adult females). McCall reports that, in natural infections, the antigen test detects less than 50 percent of cases.[10] Snyder and colleagues present differing data from natural infections in which blood was obtained as long as two hours post-euthanasia, with the antigen test reported to be more sensitive than previous reports (74 percent).[11] Recently an antigen test "for cats" (IDEXX's SNAP® Feline Heartworm Antigen Test) has been marketed. This is an adaptation of the canine test with a reported increase in sensitivity of 15 percent over conventional antigen tests.

Though less specific, HW antibody tests may be of use in the detection of feline HWI, even when antigen tests are negative. The antibody test may also be useful as a marker for exposure to HWI, even if the cat never develops a mature infection. There are now two "in clinic" feline heartworm antibody tests available (HESKA™ SOLO STEP™ FH and Synbiotics' WITNESS® FH).

Thoracic radiographs have been suggested as an excellent screening test in cats. However, Schafer and Barry showed that the most sensitive radiographic criterion (left caudal pulmonary artery greater than 1.6 times the ninth rib at the ninth intercostal space) was only detected in 53 percent of cases.[12] Furthermore, even though most cats with clinical signs have some radiographic abnormality, the findings are not specific to HWD. In addition, a study by Selcer, et al., demonstrated that radiographic findings were often transient and that radiographic abnormalities were found in cats which ultimately resisted maturation of HW and

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were negative on post-mortem (i.e., "false positive").[13] Radiographic findings include enlarged caudal pulmonary arteries, often with ill-defined margins; pulmonary parenchymal changes include focal or diffuse infiltrates (interstitial, broncho-interstitial, or even alveolar), perivascular density, and occasionally, atelectasis. Pulmonary hyperinflation may also be evident. Pulmonary angiography has also been utilized to demonstrate radiolucent linear intravascular "foreign bodies," as well as enlarged, tortuous, and blunted pulmonary arteries.

Echocardiography, in our experience, is more sensitive in cats than in dogs.[3,14] Typically, a "double-lined echodensity" is evident in the main pulmonary artery, one of its' branches, the right ventricle, or occasionally at the right atrioventricular junction. We found HW echocardiographically in a78 percent of nine cases[3] as did Selcer an 16 experimental infections.[13]

Treatment and Prevention

The question arises as to whether HW prophylaxis is warranted for cats because they are not the natural host and because the incidence is low. Necropsy studies of feline HWI in the Southeast have yielded a prevalence of 2.5 to 14 percent with a median of seven percent.[1] When considering the question of institution of prophylaxis, it is worth considering that this prevalence approximates or even exceeds that of FeLV and FIV infections.[15] A 1998 nationwide antibody survey of over 2000 largely asymptomatic cats revealed an exposure prevalence of nearly 12 percent.[16] It is also noteworthy that, based on owners' information, nearly one-third of cats diagnosed with HWD at NCSU were housed solely indoors.[8] Lastly, the consequences of feline HWD are potentially dire, with su no clear therapeutic solutions. Therefore, I advocate preventative therapy in cats in endemic areas. There are now three drugs with FDA approval, which are marketed for use in cats. Ivermectin is provided in a chewable formulation, milbemycin as a flavored tablet and selamectin, a broad-spectrum parasiticide, comes in a topical formulation. The spectrum, as well as the formulation, of these products varies; hence the clients' individual needs are easily met in most cases.

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Since the vast majority of cats are amicrofilaremic, microfilaricidal therapy is unnecessary in this species. The use of arsenical-adulticides is problematic. Thiacetarsemide, if available, poses risks even in normal cats. Turner and colleagues reported death due to pulmonary edema and respiratory failure in three of 14 normal cats given thiacetarsemide (2.2 mg/kg twice over 24 hours).[17] Dillon could not confirm this acute pulmonary reaction in 12 normal cats receiving thiacetarsemide, but one cat did die after the final injection.[18] More importantly, a significant, though unquantified, percentage of cats with HWI develop pulmonary thromboembolism (PTE) after adulticidal therapy.[4,6,7] This occurs several days to a week after therapy and is often fatal. In 50 cats with HWI, seen at NCSU, 11 received thiacetarsemide. There was no significant difference in survival between those receiving thiacetarsemide and those receiving symptomatic therapy.[8]

Data on melarsomine in experimental (transplanted) HWI in cats are limited and contradictory. Although there is an abstract report in which one injection (2.5 mg/kg; one-half the recommended canine dosage) of melarsomine was used in experimentally-infected cats without treatment-related mortality, the worm burdens after treatment were not significantly different than those found in untreated control cats.[19] Diarrhea and heart murmurs were frequently noted in treated cats. A second abstract report, using either the standard canine protocol (2.5 mg/kg twice over 24 hours) or the "split-dosage" (one injection, followed by two injections, 24 hours apart, in one month), gave more favorable results.[20] The standard treatment and split-dosage regimens resulted in 79 percent and 86 percent reduction in worm burdens respectively, and there were no adverse reactions. Although promising, these unpublished data need to be interpreted with caution as the transplanted worms were young (<8-months-old and more susceptible) and the control cats experienced a 53 percent worm mortality (average worm burden was reduced by 53 percent by the act of transplantation). Additionally, the clinical experience in naturally-infected cats has been generally unfavorable, with an unacceptable mortality. Because of the inherent risk, lack of clear benefit, and

the short life expectancy of heartworms in this species, this author does not advocate adulticidal therapy in cats. Surgical removal of heartworms has been successful and is attractive because it minimizes the risk of thromboemboli. The mortality seen in the only published case series was, unfortunately, unacceptable (two of five cats).[21] This procedure may hold promise for the future, however.

Cats with HWI should be placed on a monthly preventative and short-term corticosteroid therapy (e.g., prednisone at the lowest dosage needed to control signs, ranging from as high as 2 mg/kg tid for short-term management of critical signs to as little as 1 mg/kg q48h for chronic maintenance) used to manage respiratory signs. If signs recur, alternate day steroid therapy (at the lowest dosage that controls signs) can be continued indefinitely. For embolic emergencies, oxygen, corticosteroids (dexamethasone at 1 mg/kg IV or IM or prednisolone sodium succinate at 50-100 mg IV/cat) and bronchodilators (aminophylline at 6.6 mg/kg IM q12h, theophylline sustained release at 10 mg/kg PO or terbutaline at 0.01 mg/kg SC) may be employed. Bronchodilators have logic, based on the ability of agents such as the xanthines (aminophylline and theophylline), to improve function of fatigued respiratory muscles. In addition, the finding of hyperinflation of lung fields may indicate bronchoconstriction, a condition for which bronchodilation would be indicated. Nevertheless, this author does not routinely utilize bronchodilators in feline HWD.

The use of aspirin has been questioned as vascular changes associated with HWI consume platelets, increasing their turnover rate and effectually diminishing the antithrombotic effects of the drug. Conventional

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Feline Thyrotoxic Heart Disease

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Hyperthyroidism in cats was first described by Holzworth in 1981. Most often caused by a thyroid adenoma, hyperthyroidism is currently the most important endocrinopathy in the cat, affecting multiple organ systems in as many as one in 300 cats. Despite increased awareness, Fox reports that the incidence of heart failure occurs with the same frequency in cats with hyperthyroidism as it did 10 years ago.

Thyrotoxic heart disease remains one of the most important cardiac diseases in elderly cats.

Pathophysiology

Thyroid hormone produces both direct and indirect effects on the heart. Direct actions include increased mitochondrial protein formation and sodium-potassium ATPase activity, as well as structural alteration of myosin, whereby increasing contractility. Enhanced efficiency of the sarcoplasmic reticulum's handling of calcium enhances both systolic and diastolic function. Thyroid hormone also produces an increased rate of diastolic depolarization and reduction of action potential duration in sinoatrial nodal cells, thereby increasing heart rate. Indirectly, thyroid hormone enhances cardiac performance by increasing myocardial left atrial and ventricular beta 1 adrenergic receptors. The increased basal metabolic rate and increased tissue needs for nutrients produced by thyroid hormone results in reduction of systemic vascular resistance and increase in cardiovascular volume and red blood cell mass. Eccentric cardiac hypertrophy likely results from direct effects of thyroid hormone on protein synthesis as well as from the increased demands placed on the heart by the hyperthyroid state.

The clinically recognizable manifestations of thyroid hormone on the cardiovascular system are increased cardiac mass and ventricular size, heart rate, contractility, and cardiac output.

The long-term effects of the hyperthyroid state include left and right ventricular eccentric hypertrophy with resultant AV valvular insufficiency, sinus tachycardia, coronary intimal proliferation, myocardial fibrosis, arrhythmias, and increased cardiovascular volume and red cell mass. Cardiac output is increased and hypertension often results. Despite left ventricular hypertrophy (which tends to normalize afterload), the overall effect of thyrotoxic heart disease is increased afterload (which produces more hypertrophy), with diastolic dysfunction. The result is a heart with increased oxygen needs (tachycardia and increased afterload), potential falling cardiac output (due to tachycardia, when excessive; increased afterload; diastolic dysfunction; and mitral regurgitation), and diminished ability to nourish the myocardium (coronary flow occurs mainly in diastole, the portion of the cardiac cycle most impacted by tachycardia; myocardial blood flow may be diminished by coronary arterial lesions; and a potentially falling cardiac output, possibly with diminished coronary perfusion pressure). Ultimately, this high output state with high MVO_2, may produce myocardial cell death, fibrosis, and cardiomyopathy of overload. This is manifested by systolic myocardial dysfunction and possibly heart failure.

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Cardiovascular signs

The median age of cats with hyperthyroidism is 12.8 years (range 6-20). Cardiovascular manifestations include bounding, rapid pulses and strong apex beat, heart murmur (mitral regurgitation, secondary to eccentric hypertrophy in approximately 50 percent of cases), cardiac gallop indicative of myocardial stiffness, tachycardia, arrhythmias, hypertension, diastolic dysfunction, systolic dysfunction, and congestive heart failure manifested by pleural effusion and/or pulmonary edema.

Electrocardiography

Electrocardiographic findings are those representing cardiac hypertrophy and arrhythmias related directly to the effects of thyroid hormone and to the secondary cardiac changes mentioned above. Peterson and colleagues found 66 percent of 131 cats were tachycardic, with 29 percent exhibiting a left ventricular enlargement pattern (increased R wave amplitude). Related changes included prolonged QRS duration (18 percent) and short QT interval (10 percent). Of these cats, 12 percent exhibited cardiac conduction disturbances, with the most common being left anterior fascicular block (six percent). Arrhythmias were evident in nine percent of hyperthyroid cats, with atrial premature complexes being most common (seven percent). Although rare, we have also noted atrial fibrillation. Normal electrocardiograms are recorded from 20 percent of cats with hyperthyroidism.

Radiography

Thoracic radiographs reveal cardiomegaly, often with a convex caudal

Medical management must address the underlying disorder as well as tachycardia, myocardial dysfunction, arrhythmias when clinically important, hypertension, and congestion.

left ventricular border on the lateral view. A valentine shape, similar to that seen in hypertrophic cardiomyopathy, may be seen on the dorsoventral projection. If heart failure is present, pleural effusion, and less often, pulmonary edema may be evident. In 82 hyperthyroid cats, thoracic radiographs revealed cardiomegaly in 49 percent, pleural effusion and pulmonary edema, each in five percent, and both pulmonary edema and pleural effusion in 10 percent.

Echocardiography

The echocardiographic findings in hyperthyroidism are those of eccentric hypertrophy, sometimes demonstrating severe concentric hypertrophy when concomitant hypertension exists. The atria are often enlarged, the severity of which is dependent on ventricular systolic and diastolic function and the degree and duration of AV valvular incompetence. Left ventricular performance is usually enhanced, although it may be decreased if heart failure is present. The classical echocardiographic picture is that of left ventricular dilatation with wall thicknesses normal to increased.

In descriptions of the echocardiographic findings in hyperthyroidism, over 70 percent of cats exhibit left ventricular hypertrophy (septum and/or posterior wall) and left atrial enlargement. Diastolic left ventricular luminal enlargement is noted in nearly 50 percent of cases. The normal ratio of free wall thickness to luminal dimension is maintained. In approximately 15-20 percent of cases, left ventricular performance is enhanced; however, in cats with heart failure, left ventricular performance is usually diminished. Doppler examination typically reveals mitral regurgitation in cats with murmurs. As is the case with the radiograph and electrocardiogram, even when the echocardiogram is abnormal, a definitive diagnosis cannot be made.

Other diagnostic tests

The clinical signs and signalment may be supportive of the diagnosis. In addition, polycythemia and/or elevations in liver enzyme tests are typical of hyperthyroidism. A definitive diagnosis of hyperthyroidism can be made with an elevated resting T4 concentration in the appropriate clinical setting. In some cases, however, serial T4 determinations, T3 suppression tests, or radionuclide studies may be necessary to confirm the diagnosis. If pleural effusion is present, fluid analysis will reveal a modified transudate which, on occasion, may be chylous.

Determination of systemic blood pressure may also be useful. However, whether elevated or not, a diagnosis of hyperthyroidism can neither be made nor excluded.

Management

Cardiac manifestations of thyrotoxic heart disease variably include murmurs, gallops, sinus tachycardia, other arrhythmias and conduction disturbances, systemic hypertension, rarely systemic arterial embolism, diastolic and systolic dysfunction, and congestive heart failure manifested by pleural effusion and/or pulmonary edema. Medical management must address the underlying disorder as well as tachycardia, myocardial dysfunction, arrhythmias when clinically important, hypertension, and congestion.

Sinus tachycardia is typically managed with the beta blocker, atenolol, at 6.25-12.5 mg sid-bid PO q24h. Alternatively, diltiazem (7.5 mg tid) or sustained release diltiazem (Cardizem CD® at 45 mg once daily, Dilacor® at 30 mg bid PO), may be utilized. In general, the beta blockers control heart rate more effectively than do the calcium channel blockers and are preferred by this author. If heart failure is also present, digoxin (0.007 mg/kg PO q48h) may be a useful alternative. At the same time, methimazole (Tapazole®) at 10-15 mg/day in divided doses is instituted bid-tid or, if clinical conditions permit, surgical excision of the adenomatous thyroid gland can be performed. Often it is preferable to delay surgery until the cat has recovered somewhat (one to three weeks) and tachycardia has been well controlled. An alternative definitive therapy is ^{131}I administration; this is generally confined to referral centers. Cardiac arrhythmias most often do not require specific therapy beyond that mentioned above. Ventricular and/or atrial ectopy often subside with control of heart rate and/or failure and with beta blockade. Diltiazem has been shown to reduce atrial and ventricular ectopy, as well as heart rate, in hyperthyroid human patients.

In over 80 percent of cats with heart failure, systolic myocardial dysfunction is present.

In over 80 percent of cats with heart failure, systolic myocardial dysfunction is present. For this reason,

digoxin therapy as described above is indicated. In addition, management of signs of congestion (pleural effusion) with thoracentesis, low doses of furosemide (1 mg/kg PO q24h-bid) to prevent recurrence, and an angiotensin converting enzyme inhibitor, such as enalapril (Enacard®) at 0.5 mg/kg q24-72h (for its neurohumoral and vasodilatory effects) may be employed. If pulmonary edema is present, more aggressive diuresis may be necessary (furosemide at 1-3 mg/kg PRN parenterally, followed by the lowest effective oral dose). Nitroglycerin paste may also be used to treat pulmonary edema, with 1/8-1/4 inch applied to the inner surface of the ear tid for the first 24 hours, then discontinued. It is important not to produce excessive preload reduction, as this will further reduce cardiac output and worsen signs of forward heart failure (azotemia, weakness, weak pulses, etc). Control of heart rate is likewise important in the cat with heart failure. However, because of their negative inotropic potential, beta and calcium channel blocking agents should be used carefully, and only after digoxin therapy has failed to reduce heart rate. If an echocardiogram reveals normal systolic function, heart failure can be assumed to be due to diastolic failure. In this instance, digoxin is not indicated and atenolol or diltiazem should be employed for their rate-reducing and positive lusitropic effects. In any case, heat (if hypothermic), cage rest, and supportive care are indicated.

Hypertension complicates over 80 percent of cases and should be controlled in order to slow or prevent progression of cardiac dysfunction and to prevent retinal detachment and progression of renal disease. No clear guidelines for the treatment of hypertension in cats have come forth, but thyrotoxic systemic hypertension is largely reversed by two to four months after control of the hyperthyroidism. Nevertheless, at least for the first two to four months of therapy, moderate salt restriction and some combination of beta blockers (atenolol), calcium channel blockers, or angiotensin converting enzyme inhibitors should be employed, with or without low dose diuretic therapy. Researchers have recently described success with the calcium channel blocker amlodipine, used alone at 0.625 mg PO q24h in a hypertensive cat with renal disease. Careful serial monitoring of blood pressure allows "fine-tuning" of the treatment regime chosen.

The prognosis for hyperthyroidism is generally good, if the diagnosis is made and treatment instituted early.

Prognosis

The prognosis for hyperthyroidism is generally good, if the diagnosis is made and treatment instituted early. This is particularly true if surgery or ^{131}I is employed. Methimazole may also be used successfully, but four percent of cats treated develop serious hematological aberrations. Cardiac changes are potentially reversible. However, researchers showed that chamber enlargement and wall thicknesses were maintained in over 50 percent of cases 10 months post-therapy, even though exuberant cardiac function had normalized. Contrarily, if heart failure has resulted, the prognosis is grim. Control of hyperthyroidism reverses hypertension.

Feline Heartworm Disease: What's Different in 2004?

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doses of aspirin did not prevent angiographically-detected vascular lesions.[22] Dosages of aspirin necessary to produce even limited histological benefit approached the toxic range. Despite this, because therapeutic options are limited; because at conventional doses (80 mg PO q72h), aspirin is generally harmless, inexpensive, and convenient; and because the quoted studies were based on relatively insensitive estimates of platelet function and pulmonary arterial disease (thereby possibly missing subtle benefits), the author continues to advocate aspirin for cats with HWI. Aspirin is not prescribed with concurrent corticosteroid therapy. Management of other signs of HWD in cats is largely symptomatic.

Prognosis

In the aforementioned study of 50 cats with natural HWI, at least 12 cats died of causes other than heartworm disease. Seven of these and two living cats were considered to have survived heartworm disease (lived >1000 days).[8] The median survival for all heartworm-infected cats living beyond the day of diagnosis was 1460 days (four years; range 2-4015 days), while the median survival of all cats (n=48 with adequate follow-up) was 540 days (1.5 years; range 0-4015 days). Survival of 11 cats treated with sodium caparsolate (mean 1669 days) was not significantly different from that of the 30 managed without adulticide (mean 1107 days). Likewise, youth (<3 years of age), presence of dyspnea, cough, ELISA-positivity for heartworm antigen, presence of echocardiographically-identifiable worms, or gender of the cat did not appear to affect survival.[8] The effect of HWI on survival has been compared to that of other cardiovascular diseases.[23]

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