


CONSULT THE EXPERT

CANINE HEARTWORM

**Andrew R. Moorhead, DVM, MS,
PhD, DACVM (Parasitology)**
University of Georgia

A microscopic image showing a long, thin, segmented heartworm (Dirofilaria immitis) in the center, surrounded by numerous small, round blood cells. The heartworm is dark purple and has a lighter, segmented interior. The blood cells are light purple with dark purple nuclei. A red vertical bar is on the right side of the image, containing text.

Dirofilaria immitis (ie, canine heartworm), a potentially deadly disease, is arguably the most important parasite that affects dogs in North America, with ≈100,000 new cases reported annually.¹ It is thus important that all clinicians (including those in historically nonendemic regions) be knowledgeable regarding the heartworm life cycle, as this will allow for better understanding of treatment and prevention strategies.

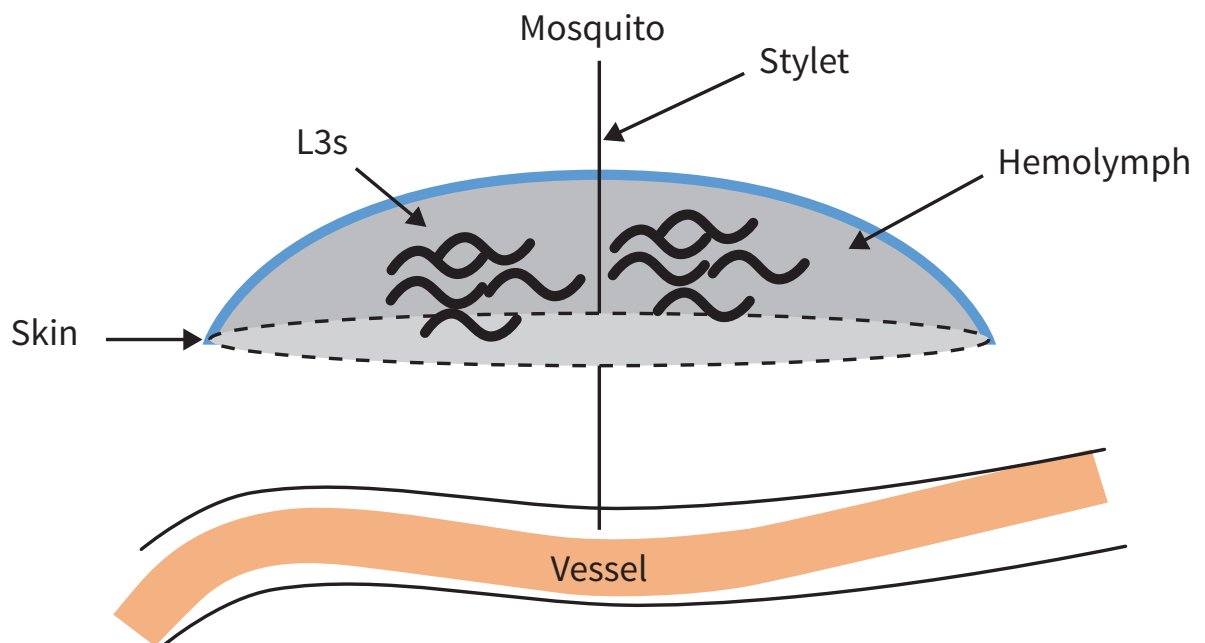
Life Cycle

Adult female heartworms can grow to a length of 10 to 12 inches; male heartworms typically reach 4 to 6 inches. Adult worms can live up to 5 to 7 years in dogs, where they mate and produce microfilariae ($>300\ \mu\text{m}$ in length) that circulate in the blood. Microfilariae are then ingested by an intermediate mosquito host—this is essential for heartworm development—in which they migrate for an average period of ≈ 14 days and develop to first-stage larvae (L1). The L1 then molt twice and become infectious third-stage larvae (L3) that are $\approx 1\ \text{mm}$ in length and exist in the head of the mosquito. The ambient temperature must be $>57.2^\circ\text{F}$ (14°C) for larval development in the mosquito.² When the mosquito lands and takes a blood meal, the L3 emerge from the proboscis (ie, mosquito mouthparts) and surround the stylet (ie, piercing part of the proboscis) in a pool of mosquito hemolymph (*Figure*). When the stylet is removed, the larvae enter the host through the hole created by the stylet. This process is in contrast to the commonly held belief that larvae are injected by the mosquito into the definitive host.³

Once inside the definitive host, the larvae follow a complicated migration pathway. The L3 remain at the site of entrance for ≈ 3 to 4 days; during which time they molt to fourth-stage larvae (L4). Molting is usually completed within 4 days but may not occur until day 12.³ L4 typically molt to the last stage (ie, juvenile adults) between day 50 to 58, then migrate through the subcutaneous tissue and musculature.⁴ Worms begin arriving in the pulmonary artery (ie, the final location) by days 67 to 70^{4,5}; most worms reach this location by day 120. By day 180, worms are sexually mature and begin to produce microfilariae; thus, completing the life cycle.⁴ This timeline can vary.

Pathophysiology

Heartworm disease can result in death, especially in cases with a large number of worms (ie, caval syndrome). Although this acute presentation can be remarkable, pathology is most often observed secondary to the prolonged presence of worms in the pulmonary arteries, which can occur as early as day 60 to 70 of infection.^{4,5} Severity of disease and extent of pathology are influenced by multiple



▲ **FIGURE** L3 in a hemolymph pool, with the mosquito stylet still inserted in the definitive host

factors (eg, number of adult worms, duration of infection, host immune response, presence of dead worms). The presence of dead worms is especially important, as they are carried by the blood flow further into the pulmonary vessels, resulting in pulmonary thromboembolism (PTE).

Heartworms are constantly pushed back and forth by the pulsing blood flow; this can cause trauma to the vessels that results in thickening of the tunica intima and inflammation of the vessel wall characterized by a pathognomonic roughened, stippled appearance.^{6,7} This prolonged damage can eventually lead to vessel inelasticity, resulting in increasing pressure on the main pulmonary artery, right heart, and vena cava. In turn, this pressure can lead to chronic, passive congestion and pulmonary hypertension that results in right-sided heart enlargement and (eventually) failure.⁸ Right-sided heart failure can cause liver congestion and ascites. In addition, renal lesions (eg, glomerulonephritis) can develop secondary to immune complex deposition.⁹ Damage increases with the persistence of worms in the vessels.

Although worms can cause damage, data suggest that endosymbiotic *Wolbachia* spp can cause some of the inflammation associated with heartworm disease, specifically after worm death. These bacteria are endosymbionts of many filarial worms, are transmitted vertically, and live within the worm; they do not result in overt pathology.¹⁰ When the worms die (either of a natural cause or due to drug treatment), the surface proteins of *Wolbachia* spp are exposed to the host, and the host's immune system responds.¹¹ It is therefore important to eliminate *Wolbachia* spp from the worms before treatment is initiated.

Clinical Signs

Dogs infected with *D immitis* may be presented with no to mild, moderate, or severe clinical signs or with caval syndrome. Although the severity of clinical signs depends on many factors, the presence or absence of clinical signs is important for staging the disease (see *Clinical Signs of Heartworm Disease*).

Diagnostics

A 3-dose melarsomine treatment protocol is safe,⁸ but complications are possible. Disease staging should thus be performed prior to treatment. Diagnostics should include physical examination, immunodiagnosis, microfilariae testing, radiography, and cardiac ultrasonography, as well as CBC, serum chemistry profile, and urinalysis. However, if diagnostic staging cannot be performed, the 3-dose protocol is recommended over other treatment protocols.

Physical Examination

Physical examination should occur first and clinical signs indicative of disease severity (see *Clinical Signs of Heartworm Disease*) should be assessed.

Immunodiagnosis

Antigen testing is considered the gold standard for diagnosing heartworm disease. Antigen tests are highly sensitive and specific for infections of adult female worms >8 months of age because older

CLINICAL SIGNS OF HEARTWORM DISEASE

- ▶ No to mild signs: Patients are subclinical or have a mild cough but otherwise appear healthy.
- ▶ Moderate signs: Patients have a moderate cough, can have difficulty breathing, and can be slightly exercise intolerant (eg, inability to run, tire more easily than normal on walks).
- ▶ Severe signs: Patients are dyspneic and can be severely exercise intolerant (eg, difficulty walking or walking with labored breathing). Syncope and hemoptysis may be present. Signs of right-sided congestive heart failure (eg, ascites) are present.
- ▶ Caval syndrome: Patients typically have associated acute presentation of signs (eg severe dyspnea, collapse) normally related to a large numbers of worms obstructing blood flow through the tricuspid valve. Hemoglobinemia and pigmenturia are characteristic. Onset is rapid and results in death after 12 to 72 hours if not treated.⁸

PTE = pulmonary thromboembolism

worms tend to produce more antigens. However, single-sex infections of only males are undetectable using this method. Antigen tests do not routinely detect prepatent infections (worms <5 months of age) and are not quantitative. The color of any test is not correlated with worm burden. Antigen tests can also be used to determine the effectiveness of adulticidal treatment. If all worms are killed, adult antigens should be cleared from the blood by 9 months after treatment.⁸

In a subset of dogs with adult female heartworms, no detectable antigen is present. This may be due to immune complex formation between a heartworm-derived antigen and antibodies against the antigen. Therefore, it is crucial to test all dogs for microfilariae at the same time as antigen testing in case the antigen test is negative due to sequestration of the antigen by immune complex formation.¹² Immune complexes can be dissociated with heat treatment of the sample. Current recommendations for heat treatment of serum/plasma are when no antigen is detected but the patient is microfilaremic and/or has clinical signs.

Microfilariae Testing

Several methods can be used to evaluate for the presence of microfilariae, regardless of antigen status. Examination of a drop of blood or direct smear detects fewer infections than a concentration

technique (eg, Knott or filter test). For the aforementioned reasons regarding the formation of immune complexes, microfilarial testing should be performed simultaneously with antigen testing. Because as many as 20% of dogs may be amicrofilaremic, this is not recommended as a stand-alone diagnostic test.⁸

In addition, *D immitis* should be differentiated from *Acanthocheilonema (Dipetalonema) reconditum*, which is a nonpathogenic worm that is transmitted by fleas, lives in the subcutaneous tissue, and does not require treatment. The easiest way to differentiate these microfilariae in a clinical setting is to observe their movement under a microscope. A *reconditum* moves progressively, whereas *D immitis* are mobile but remain in a single location.

Radiography

Radiography enables assessment of damage that has already occurred.

Cardiac Ultrasonography

ECG is not a first-line diagnostic method for heartworm disease. However, visualization of heartworms via ultrasound can confirm infection; lack of visualization does not rule out infection.

CBC, Serum Chemistry Profile, & Urinalysis

Liver and kidney function should be assessed before administration of melarsomine. Existing disease should be considered prior to treatment.⁸

Treatment

Previously, intravenous thiacetarsamide sodium was used to treat *D immitis*, but this drug had serious adverse effects.¹³ Later, melarsomine dihydrochloride was introduced and considered to be a significant improvement for treatment, with early treatment protocols involving a straightforward 2-dose injection protocol and the 3-dose protocol being reserved for more complicated cases.¹⁴ Due to advancements in knowledge about *D immitis*, treatment protocol now involves additional components, including melarsomine dihydrochloride, macrocyclic lactones, corticosteroids, and doxycycline.

It is crucial to test all dogs for microfilariae at the same time as antigen testing.

PTE = pulmonary thromboembolism

Protocol

The timing of each component of heartworm disease treatment is detailed by the AHS.⁸ Macrocytic lactone and 4-week doxycycline treatment should be initiated at the time of diagnosis. After 1 month and then monthly, a macrocytic lactone should be administered unless a sustained-release moxidectin product was chosen as the macrocytic lactone in the beginning of the treatment protocol. Two months after diagnosis, the first dose of melarsomine should be administered, followed 1 month later by a second and third dose given 24 hours apart. It is thought that the month in between the end of doxycycline and the start of melarsomine therapy is necessary for worm-mediated degradation of *Wolbachia* spp killed by doxycycline,¹⁵ ensuring no immunogenic *Wolbachia* spp surface proteins are released into the bloodstream. Because *Wolbachia* spp is an endosymbiont, killing the bacteria should also weaken the worm, as *Wolbachia* spp is required for worm survival.

Macrocytic Lactones

Macrocytic lactone preventive treatment should be started at the time of diagnosis (ie, 2 months prior to the first melarsomine treatment). This can help prevent further infections and help eliminate developing larvae.

Doxycycline

The antibiotic doxycycline (recommended dosage, 10 mg/kg every 12 hours for 28 days) is critical in the treatment of heartworm disease because of the drug's activity against *Wolbachia* spp.⁸

Melarsomine Dihydrochloride

The labeled dosage for melarsomine is 2.5 mg/kg IM twice in the epaxial muscles 24 hours apart.⁸ However, the AHS recommends an alternate or 3-dose regimen, in which 1 injection (2.5 mg/kg) is administered, then 2 doses are given 30 days later at a 24-hour interval. This regimen has increased safety and efficacy but includes an additional month of exercise restriction, increased total arsenical dose, and additional cost of a third injection.⁸

Corticosteroids

Prednisone (0.5 mg/kg every 12 hours the first week, 0.5 mg/kg every 24 hours the second week, 0.5 mg/kg every 48 hours the third and fourth weeks) can be administered.⁸ It is recommended to taper the anti-inflammatory dosage concurrent with the first and third injections of melarsomine. In addition, prednisone administered concurrently with doxycycline is recommended if there are clinical signs of heartworm disease.⁸

Slow-Kill Methods

Numerous studies have evaluated the efficacy of slow-kill modalities, which consist of a prophylactic dose of ivermectin, topical moxidectin, or injectable moxidectin and concurrent administration of doxycycline as an adulticidal protocol.¹⁵⁻¹⁸ These protocols have varying efficacy and require a longer time period than the 3-dose regimen of melarsomine. Although these protocols may be considered advantageous in certain settings, they are salvage procedures. The use of the slow-kill method versus the 3-dose regimen is analogous to femoral head osteotomy versus total hip replacement in a dog. The slow-kill method can be successful, but it is not ideal, as the worms remain in the vessels longer, and the time of worm death is variable as compared with melarsomine treatment.

Pulmonary Thromboembolism & Pulmonary Damage

PTE and pulmonary damage are inevitable consequences of successful adulticidal therapy. Although no current tests are predictive for PTE, the severity of clinical signs can be reduced via administration of doxycycline and corticosteroids during treatment.⁸ Clinical signs of embolism include fever, cough, hemoptysis, and exacerbation of right-sided heart failure. These signs are usually seen within 7 to 10 days but may occur for up to 4 weeks posttreatment. Therefore, exercise restriction for 6 to 8 weeks posttreatment (total of 10-12 weeks) is essential.⁸ This can be difficult, but cage rest (leash walk only) is as important as melarsomine injections.

Continues ►

Compromise may be necessary to ensure adherence to exercise restriction. If the dog is anxious in a crate, confinement in a small room may be preferred to ease anxiety. Assessing the ease at which the pet owner is able to restrict the dog can help with tailoring exercise restriction recommendations. For example, the owner may be asked to rate (on a scale of 1 to 10) the ease of crate resting the dog for 10 to 12 weeks (considering this duration includes 4 weeks after the first injection of melarsomine, then 6-8 weeks after the second and third injections of melarsomine). Recommendations can be given based on the answer.

Prognosis & Prevention

Prognosis is typically good with treatment and appropriate exercise restriction. To prevent reinfection, preventives labeled for use against heartworms should be adherently administered.

Clinical Follow-Up/Monitoring

Dogs should be tested for microfilariae 30 days posttreatment. If the test is positive, a microfilaricide should be given. A test for antigen should be given 9 months post-treatment. If the test is positive, the dog should be retreated with a 2-dose protocol.

Conclusion

Treatment recommendations for heartworm disease are constantly changing. It is thus important to keep current on developments. The 3-dose treatment protocol is the recommended approach to treatment in most cases. ■■■

See page 61 for references.



Brief Summary: Before using NexGard® (afoxolaner) Chewables, please consult the product insert, a summary of which follows.

CAUTION: Federal (USA) law restricts this drug to use by or on the order of a licensed veterinarian.

Description: NexGard is a soft chewable for oral administration to dogs and puppies according to their weight. Each chewable is formulated to provide a minimum afoxolaner dosage of 1.14 mg/lb (2.5 mg/kg).

Indications: NexGard kills adult fleas and is indicated for the treatment and prevention of flea infestations (*Ctenocephalides felis*), and the treatment and control of *Ixodes scapularis*, *Dermacentor variabilis*, *Amblyomma americanum*, and *Rhipicephalus sanguineus* infestations in dogs and puppies 8 weeks of age and older, weighing 4 pounds of body weight or greater, for one month. NexGard is indicated for the prevention of *Borrelia burgdorferi* infections as a direct result of killing *Ixodes scapularis* vector ticks.

Dosage and Administration: NexGard is given orally once a month, at the minimum dosage of 1.14 mg/lb (2.5 mg/kg). See full product insert for dosing table and details.

Warnings: Not for use in humans. Keep this and all drugs out of the reach of children. In case of accidental ingestion, contact a physician immediately. Keep NexGard in a secure location out of reach of dogs, cats, and other animals to prevent accidental ingestion or overdose.

Precautions: Afoxolaner is a member of the isoxazoline class. This class has been associated with neurologic adverse reactions including tremors, ataxia, and seizures. Seizures have been reported in dogs receiving isoxazoline class drugs, even in dogs without a history of seizures. Use with caution in dogs with a history of seizures or neurologic disorders.

The safe use of NexGard in breeding, pregnant or lactating dogs has not been evaluated.

Adverse Reactions: In a well-controlled US field study, which included a total of 333 households and 615 treated dogs (415 administered afoxolaner, 200 administered active control), no serious adverse reactions were observed with NexGard.

Over the 90-day study period, all observations of potential adverse reactions were recorded. The most frequent reactions reported at an incidence of > 1% within any of the three months of observations are presented in the following table.

Table 1: Dogs with Adverse Reactions.

	Treatment Group			
	Afoxolaner		Oral active control	
	N ¹	% (n=415)	N ²	% (n=200)
Vomiting (with and without blood)	17	4.1	25	12.5
Dry/Flaky Skin	13	3.1	2	1.0
Diarrhea (with and without blood)	13	3.1	7	3.5
Lethargy	7	1.7	4	2.0
Anorexia	5	1.2	9	4.5

¹ Number of dogs in the afoxolaner treatment group with the identified abnormality.

² Number of dogs in the control group with the identified abnormality.

In the US field study, one dog with a history of seizures experienced a seizure on the same day after receiving the first dose and on the same day after receiving the second dose of NexGard. This dog experienced a third seizure one week after receiving the third dose. The dog remained enrolled and completed the study. Another dog with a history of seizures had a seizure 19 days after the third dose of NexGard. The dog remained enrolled and completed the study. A third dog with a history of seizures received NexGard and experienced no seizures throughout the study.

Post-Approval Experience (July 2018): The following adverse events are based on post-approval adverse drug experience reporting. Not all adverse events are reported to FDA/CVM. It is not always possible to reliably estimate the adverse event frequency or establish a causal relationship to product exposure using these data.

The following adverse events reported for dogs are listed in decreasing order of reporting frequency for NexGard: Vomiting, pruritus, lethargy, diarrhea (with and without blood), anorexia, seizure, hyperactivity/restlessness, panting, erythema, ataxia, dermatitis (including rash, papules), allergic reactions (including hives, swelling), and tremors.

Effectiveness: See full product insert for details regarding Effectiveness.

Animal Safety: In a margin of safety study, NexGard was administered orally to 8 to 9-week-old Beagle puppies at 1, 3, and 5 times the maximum exposure dose for a total of six treatments. There were no clinically-relevant effects related to treatment on physical examination, body weight, food consumption, clinical pathology (hematology, clinical chemistries, or coagulation tests), gross pathology, histopathology or organ weights. Vomiting occurred throughout the study, with a similar incidence in the treated and control groups, including one dog in the 5x group that vomited four hours after treatment.

In a well-controlled field study, no adverse reactions were observed from the concomitant use of NexGard with other medications.

Contact Information: For a copy of the Safety Data Sheet (SDS) or to report suspected adverse drug events, contact Boehringer Ingelheim Animal Health USA Inc. at 1-888-637-4251. For additional information about adverse drug experience reporting for animal drugs, contact FDA at 1-888-FDA-VETS or www.fda.gov/reportanimalae.

The information provided here is not comprehensive. The full FDA-approved product insert is available at www.nexgardfordogs.com. Consult your veterinarian for further information.

Product approved by FDA under NADA # 141-406

Marketed by: Frontline Vet Labs™, a Division of Boehringer Ingelheim Animal Health USA Inc. Duluth, GA 30096

NexGard® is a registered trademark and FRONTLINE VET LABS™ is a trademark of the Boehringer Ingelheim Group.

©2020 Boehringer Ingelheim Animal Health USA Inc.

All rights reserved.

Reference package insert: 1050-4493-09 Rev. 11/2019

Brief summary preparation date: 08/2020

US-PET-0735-2020

References

- Self SW, Pulaski CN, McMahan CS, Brown DA, Yabsley MJ, Gettings JR. Regional and local temporal trends in the prevalence of canine heartworm infection in the contiguous United States: 2012-2018. *Parasit Vectors*. 2019;12(1):380.
- Bowman DD, Atkins CE. Heartworm biology, treatment, and control. *Vet Clin North Am Small Anim Pract*. 2009;39(6):1127-1158, vii.
- Anderson RC. The superfamily *Acuarioidae*. In: Anderson RC. *Nematode Parasites of Vertebrates: Their Development and Transmission*. 2nd ed. CABI Publishing; 2000:483-485.
- Kotani T, Powers KG. Developmental stages of *Dirofilaria immitis* in the dog. *Am J Vet Res*. 1982;43(12):2199-2206.
- Orihel TC. Morphology of the larval stages of *Dirofilaria immitis* in the dog. *J Parasitol*. 1961;47(2):251-262.
- Keith JC Jr, Schaub RG, Rawlings C. Early arterial injury-induced myointimal proliferation in canine pulmonary arteries. *Am J Vet Res*. 1983;44(2):181-186.
- Falcón-Cordón Y, Montoya-Alonso JA, Caro-Vadillo A, Matos-Rivero JI, Carretón E. Persistence of pulmonary endarteritis in canine heartworm infection 10 months after the eradication of adult parasites of *Dirofilaria immitis*. *Vet Parasitol*. 2019;273:1-4.
- American Heartworm Society. Current canine guidelines for the diagnosis, prevention, and management of heartworm (*Dirofilaria immitis*) infection in dogs, 2020. AHS website. <https://www.heartwormsociety.org>. Accessed March 6, 2020.
- Ludders JW, Grauer GF, Dubielzig RR, Ribble GA, Wilson JW. Renal microcirculatory and correlated histologic changes associated with dirofilariasis in dogs. *Am J Vet Res*. 1988;49(6):826-830.
- Kramer L, Grandi G, Leoni M, et al. *Wolbachia* and its influence on the pathology and immunology of *Dirofilaria immitis* infection. *Vet Parasitol*. 2008;158(3):191-195.
- Kramer LH, Tamarozzi F, Morchón R, et al. Immune response to and tissue localization of the *Wolbachia* surface protein (WSP) in dogs with natural heartworm (*Dirofilaria immitis*) infection. *Vet Immunol Immunopathol*. 2005;106(3-4):303-308.
- Little S, Saleh M, Wohltjen M, Nagamori Y. Prime detection of *Dirofilaria immitis*: understanding the influence of blocked antigen on heartworm test performance. *Parasit Vectors*. 2018;11(1):186.
- Hoskins JD, Hribernik TN, Kearney MT. Complications following thiacetarsamide sodium therapy in Louisiana dogs with naturally-occurring heartworm disease. *Cornell Vet*. 1985;75(4):531-539.
- Raynaud JP. Thiacetarsamide (adulticide) versus melarsomine (RM 340) developed as macrofilaricide (adulticide and larvicide) to cure canine heartworm infection in dogs. *Ann Rech Vet*. 1992;23(1):1-25.
- McCall JW, Genchi C, Kramer L, et al. Heartworm and *Wolbachia*: therapeutic implications. *Vet Parasitol*. 2008;158(3):204-214.
- Bazzocchi C, Mortarino M, Grandi G, et al. Combined ivermectin and doxycycline treatment has microfilaricidal and adulticidal activity against *Dirofilaria immitis* in experimentally infected dogs. *Int J Parasitol*. 2008;38(12):1401-1410.
- Savadelis MD, Ohmes CM, Hostetler JA, et al. Assessment of parasitological findings in heartworm-infected beagles treated with Advantage Multi® for dogs (10% imidacloprid + 2.5% moxidectin) and doxycycline. *Parasit Vectors*. 2017;10(1):245.
- Alberigi B, de Souza CDSF, Fernandes JI, Merlo A, Labarthe N. Use of slow-release injectable moxidectin for treatment of *Dirofilaria immitis* infection during pregnancy. *Front Vet Sci*. 2020;6:440.

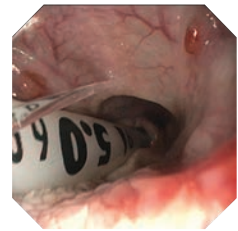
CASE IN POINT ► CONTINUED FROM PAGE 60

References

- Lloret A, Addie DD, Boucraut-Baralon C, et al. Cytauxzoonosis in cats: ABCD guidelines on prevention and management. *J Feline Med Surg*. 2015;17(7):637-641.
- Thomas JE, Ohmes CM, Payton ME, Hostetler JA, Reichard MV. Minimum transmission time of *Cytauxzoon felis* by *Amblyomma americanum* to domestic cats in relation to duration of infestation, and investigation of ingestion of infected ticks as a potential route of transmission. *J Feline Med Surg*. 2018;20(2):67-72.
- Cohn LA, Birkenheuer AJ, Brunker JD, Ratcliff ER, Craig AW. Efficacy of atovaquone and azithromycin or imidocarb dipropionate in cats with acute cytauxzoonosis. *J Vet Intern Med*. 2011;25(1):55-60.
- Cohn LA. Cytauxzoonosis. In: Sykes JE, ed. *Canine and Feline Infectious Diseases*. Elsevier Saunders; 2014:739-744.
- Nagamori Y, Slovak JE, Reichard MV. Prevalence of *Cytauxzoon felis* infection in healthy free-roaming cats in north-central Oklahoma and central Iowa. *JFMS Open Rep*. 2016;2(1):2055116916655174.
- Allen KE, Thomas JE, Wohltjen ML, Reichard MV. Transmission of *Cytauxzoon felis* to domestic cats by *Amblyomma americanum* nymphs. *Parasit Vectors*. 2019;12(1):28.
- Reichard MV, Thomas JE, Arther RG, et al. Efficacy of an imidacloprid 10%/flumethrin 4.5% collar (Seresto, Bayer) for preventing the transmission of *Cytauxzoon felis* to domestic cats by *Amblyomma americanum*. *Parasitol Res*. 2013;112(Suppl 1):S11-S20.
- Reichard MV, Rugg JJ, Thomas JE, et al. Efficacy of a topical formulation of selamectin plus sarolaner against induced infestations of *Amblyomma americanum* on cats and prevention of *Cytauxzoon felis* transmission. *Vet Parasitol*. 2019;270(Suppl 1):S31-S37.

WHAT ARE YOU BILLING?

Fill in what you would charge in the spaces next to the procedure and total it up:



- Emergency Exam..... _____
- Radiographs, Abdomen _____
- IV Catheter..... _____
- Anesthesia Level II..... _____
- Endoscopy, Minor (< 1 hr)..... _____
- CBC/Chem/LTES..... _____
- Labs/Injectables/Other..... _____

COMPARE THIS TO THE LEASE BELOW

TOTAL: \$ _____

Basic OLYMPUS 190 System w/Instruments
AVERAGE MONTHLY LEASE COST: \$1,543.00

FOR MORE INFO:  **endoscopy support services, inc.** *Better Products, Better Medicine* endoscopy.com

Ph: (845) 277-1700 • E: sales@endoscopy.com