

Outcome of minimally invasive surgical treatment of heartworm caval syndrome in dogs: 42 cases (1999–2007)

Christina M. Bové, DVM; Sonya G. Gordon, DVM, DVSc, DACVIM; Ashley B. Saunders, DVM, DACVIM; Matthew W. Miller, DVM, MS, DACVIM; Risa M. Roland, DVM, DACVIM; Sarah E. Achen, DVM, DACVIM; Lori T. Drourr, DVM, DACVIM; May M. Boggess, PhD

Objective—To report the outcome of minimally invasive surgical treatment of heartworm caval syndrome in a series of dogs and to provide information on long-term survival of patients with this condition.

Design—Retrospective case series.

Animals—42 client-owned dogs with a diagnosis of heartworm caval syndrome.

Procedures—Information on history, clinical, laboratory, and diagnostic imaging findings and treatment was obtained from medical records. When possible, additional follow-up information was obtained through telephone interviews with referring veterinarians and owners.

Results—Of the 42 dogs with caval syndrome, 21 underwent minimally invasive surgical treatment consisting of transvenous heartworm extraction. Two of the 21 dogs died during the procedure, and after surgery, 4 died. Following induction of anesthesia, heartworms migrated into the distal portion of the pulmonary artery in 1 dog; therefore, extraction was not attempted. Transvenous heartworm extraction was completed successfully in 14 dogs, and all 14 of these dogs were discharged from the hospital. Mean follow-up time in these 14 dogs was 24.4 ± 17.7 months with a range of 2 to 56 months. At the time of final follow-up, 10 of these 14 dogs had survived at least 18 months and 7 had survived > 24 months. By the end of the study, 1 dog was lost to follow-up and 3 had been euthanized for unrelated reasons.

Conclusions and Clinical Relevance—Results of the study reported here suggest that dogs with caval syndrome that undergo successful transvenous heartworm extraction and survive to discharge have a good long-term prognosis. (*J Am Vet Med Assoc* 2010;236:187–192)

Heartworm disease is a serious and potentially fatal condition caused by the nematode, *Dirofilaria immitis*, that is transmitted to dogs by at least 10 to 15 species of mosquitoes.^{1,2} Heartworm disease is most common in tropical and subtropical climates, but occurs throughout the United States, with an incidence of 45% along the Atlantic and Gulf coasts and the Mississippi River and its major tributaries.^{1–5} The number of adult heartworms found in affected dogs varies dramatically, depending on the local concentration of infected mosquitoes and mean temperature in the region.^{3,4,6}

Clinical signs of HWD reflect the number of heartworms, the duration of infection, and the host immune response.^{2–4} The immunologic response to the parasite may be more important than the worm burden in deter-

ABBREVIATIONS

ALT	Alanine aminotransferase
CS	Caval syndrome
DIC	Disseminated intravascular coagulation
HWD	Heartworm disease

mining disease severity.⁷ Clinical signs that have been reported for affected dogs include cough, dyspnea, syncope, weight loss, and lethargy.^{2–5,8}

Caval syndrome is a life-threatening manifestation of HWD.^{4,9,10} Caval syndrome is thought to develop when a large number of heartworms mature over a short period and is therefore uncommon in geographic areas characterized by a low incidence of heartworm infection and low heartworm burdens.^{1–3} Caval syndrome is characterized by severe pulmonary hypertension and decreased cardiac output, with subsequent displacement and retrograde migration of adult heartworms into the main pulmonary arteries, right atrium, right ventricle, and, less commonly, the vena cava.^{3,11} Migrating heartworms may mechanically disrupt the tricuspid valve and physically obstruct blood flow entering the right atrium and right ventricle.^{1,10,11}

The number of heartworms required to cause CS is dependent on the size of the patient. In large dogs, CS

From Animal Emergency Clinic, 19311 State Hwy 249, Houston, TX 77070 (Bové); the Department of Small Animal Clinical Sciences and The Michael E. DeBakey Institute for Comparative Cardiovascular Sciences and Biomedical Devices, College of Veterinary Medicine and Biomedical Sciences (Gordon, Saunders, Miller), and the Department of Statistics, College of Science (Boggess), Texas A&M University, College Station, TX 77843; Metropolitan Veterinary Associates, 2626 Van Buren Ave, Norristown, PA 19403 (Roland); Michigan Veterinary Specialists, 29080 Inkster Rd, Southfield, MI 48034 (Achen); and 163 Prospect Ave, Sausalito, CA 94965 (Drourr).

Address correspondence to Dr. Gordon (sgordon@cvm.tamu.edu).

is typically associated with heartworm burdens of 60 to 100.^{1,2} However, an experimental study¹² documented CS in dogs with a mean worm burden of 40 (range, 12 to 125 worms) and with as few as 12 worms in 1 dog. In general, prognosis for CS is thought to be poor without prompt heartworm extraction.^{2,5,10} The objectives of the present study were to describe the outcome of treatment of CS in dogs and to provide information on long-term survival of patients with this condition.

Material and Methods

Case selection—The electronic medical records database of Texas A&M University Teaching Hospital was searched to identify all dogs with a diagnosis of CS that were admitted between January 16, 1999, and June 25, 2007. Forty-two dogs referred for treatment of CS or subsequently determined to have CS were identified and the records reviewed for inclusion in the study. When possible, additional follow-up data were collected by telephone interviews with referring veterinarians and owners.

Medical records review—For each dog, signalment, historical information obtained from the owner or referring veterinarian, results of physical examination, results of clinicopathologic analyses (CBC, serum biochemistry panel, and coagulation panel), and results of diagnostic imaging (thoracic radiography and echocardiography) were reviewed. Dogs were categorized according to treatment as follows: dogs that had transvenous heartworm extraction attempted or dogs that had no treatment or procedure attempted. Additional information obtained from the medical records included preoperative medications, sedation and anesthetic protocols, intra- and postoperative medications, transfusions, and any other treatments administered to dogs that underwent treatment (ie, heartworm extraction). Finally, any complications, duration of hospitalization, and reasons for death or euthanasia in dogs that did not survive or were not treated were recorded.

Statistical analysis—A 2-sample proportion test was used to compare characteristics between 2 groups (eg, dogs that underwent treatment and survived vs dogs that underwent treatment and died). Linear regression (for normally distributed data) and Kruskal-Wallis tests (for data that were not normally distributed) were used to compare data among 2 groups. Kaplan-Meier product-limit estimates were used for nonparametric estimates of cumulative survival probabilities. A proportional hazards model with a Weibull distribution was used for parametric survival estimates. Model comparisons for different parametric distributions were made by use of the Akaike criterion. When data were normally distributed, they were reported as mean \pm SD. Other data were reported as median values and interquartile ranges. All statistical analyses were performed with a commercially available software package.^a Values of $P < 0.05$ were considered significant.

Results

Animals—The study population consisted of 42 dogs with a diagnosis of CS. Dogs comprised 11 (26%)

sexually intact females, 18 (43%) sexually intact males, 5 (12%) castrated males, and 8 (19%) spayed females. Breeds of dogs included Chihuahua ($n = 5$ dogs), Dachshund (5), mixed-breed dogs (4), American Staffordshire Terrier (3), Pug (2), Dalmatian (2), German Shepherd Dog (2), Rottweiler (2), English Bulldog (2), Boston Terrier (2), Toy Fox Terrier (2), Shetland Sheepdog (2), Miniature Poodle (2), Boxer (1), Welsh Sheepdog (1), Italian Greyhound (1), Chinese Crested (1), Yorkshire Terrier (1), Saluki (1), and English Springer Spaniel (1). Dogs ranged from 1.4 to 15.7 years of age (mean \pm SD, 7.0 ± 3.7 years). Median body weight was 9.3 kg (20.5 lb) and ranged from 2 to 43 kg (4.4 to 94.6 lb) with a mean body weight of 12.6 ± 9.0 kg (27.7 ± 19.8 lb).

Dogs had a variety of clinical signs at the time they were first examined at the veterinary teaching hospital (Table 1). All dogs underwent a complete physical examination, CBC determination, serum biochemical analysis, echocardiography, and heartworm antigen testing. The complete range of diagnostic tests was not performed on all dogs in the study. Some dogs underwent additional diagnostic tests, including a coagulation panel with measurement of antithrombin III (24/42 [57%]), urinalysis (40/42 [95%]), and thoracic radiography (35/42 [83%]). Additional clinical signs included coughing (18/42 [43%]), ascites (22/41 [54%]), jugular distension (15/41 [37%]), heart murmur (38/42 [91%]), exercise intolerance (25/41 [61%]), collapse (6/42 [14%]), pale mucus membranes (31/42 [74%]), weak femoral pulses (9/42 [21%]), and profound weakness with recumbency (9/42 [21%]).

Twenty-one of 42 (50%) dogs underwent heartworm extraction by means of a minimally invasive transvenous approach. Twenty-one (50%) dogs were not treated. Seventeen of the 21 dogs that did not undergo treatment were euthanized. Reasons for euthanasia included financial constraints (4/21 [24%]), poor prognosis (8/21 [47%]), and unknown (5/21 [29%]). Transvenous heartworm extraction or euthanasia was declined as an option for 3 dogs, and these dogs were discharged. All 3 dogs were subsequently lost to follow-up, although 2 dogs were expected to be seen by their primary care veterinarians soon after discharge for humane euthanasia. Transvenous heartworm extraction was not recommended as treatment for 1 dog because of a relatively low heartworm burden, severe pulmonary hypertension, and no evidence of hemolysis or DIC. This dog was discharged at the request of the owner to be euthanized by the referring veterinarian, and additional follow-up information was not available.

Dogs that underwent transvenous heartworm extraction (21/42 [50%]) received the following preoperative medications: cefazolin (10/21 [48%]), dalteparin sodium (7/21 [33%]), prednisolone (10/21 [47%]), diphenhydramine (9/21 [43%]), dexamethasone (6/21 [29%]), hydro-morphone (5/21 [24%]), albuterol aerosol (4/21 [19%]), glycopyrrolate (4/21 [19%]), prednisone (4/21 [19%]), pimobendan (1/21 [5%]), and sildenafil (1/21 [5%]). Two (10%) dogs received fresh frozen plasma transfusions prior to surgery for a diagnosis of DIC. Dogs were positioned in left lateral recumbency under sedation (5/21 [24%]) or general anesthesia (16/21 [76%]). Dogs were administered general anesthesia on the basis of their hemodynamic sta-

Table 1—Number (%) of dogs (unless otherwise indicated) with CS that had specific clinical findings and diagnostic test results (not all variables were recorded for all dogs).

Variable	All dogs (n = 42)	No heartworm extraction (n = 21)	Dogs that underwent attempted heartworm extraction			
			All dogs (n = 21)*	Successful extraction and survived to discharge (n = 14)	Died during surgery (n = 2)	Died after surgery (n = 4)
DIC†	19/31 (61)	7/11 (64)	12/20 (60)	9/13 (69)	1/2 (50)	2/4 (50)
Serum ALT						
Mild increase (130–260 U/L)	7/31 (23)	2/12 (17)	5/19 (26)	2/12 (17)	0/2 (0)	2/4 (50)
Severe increase (> 260 U/L)	9/31 (29)	4/12 (33)	5/19 (26)	3/12 (25)	2/2 (100)	0/4 (0)
Serum alkaline phosphatase						
Mild increase (147–294 U/L)	10/31 (32)	3/12 (25)	7/19 (37)	3/12 (25)	1/2 (50)	3/4 (75)
Severe increase (> 294 U/L)	5/31 (16)	2/12 (17)	3/19 (16)	3/12 (25)	0/2 (0)	0/4 (0)
BUN						
Mild increase (29–58 mg/dL)	7/35 (20)	3/14 (21)	4/21 (19)	3/14 (21)	1/2 (50)	0/4 (0)
Severe increase (> 58 mg/dL)	17/35 (49)	8/14 (57)	9/21 (43)	5/14 (36)	1/2 (50)	3/4 (75)
Serum creatinine						
Mild increase (2–4 mg/dL)	5/33 (15)	3/12 (25)	2/21 (10)	0/14 (0)	1/2 (50)	1/4 (25)
Severe increase (> 4 mg/dL)	3/33 (9)	2/12 (17)	1/21 (5)	1/14 (7)	0/2 (0)	0/4 (0)
Cachexia (body condition score < 4‡)	9/18 (50)	4/8 (50)	5/10 (50)	3/7 (43)	1/1 (100)	1/2 (50)
Anemia (Hct < 31%)	20/36 (56)	6/15 (40)	14/21 (67)	8/14 (57)	2/2 (100)	3/4 (75)
Leukocytosis (> 17 × 10 ³ WBC/μL)	25/31 (81)	9/11 (82)	16/20 (80)	10/13 (77)	2/2 (100)	3/4 (75)
Hemolysis	13/32 (41)	4/11 (36)	9/21 (43)	7/14 (50)	0/2 (0)	1/4 (25)
Location of heartworms on initial echocardiogram						
Right atrium, ventricle, or both	31/31 (100)	12/12 (100)	19/19 (100)	12/12 (100)	2/2 (100)	4/4 (100)
Pulmonary artery	13/29 (45)	4/10 (40)	9/19 (47)	4/12 (33)	2/2 (100)	3/4 (75)
Pulmonary hypertension§						
Mild (27–49 mm Hg)	20/31 (65)	7/11 (64)	13/20 (65)	9/13 (69)	2/2 (100)	2/4 (50)
Moderate (50–75 mm Hg)	3/15 (20)	1/4 (25)	2/11 (18)	2/8 (25)	0/2 (0)	1/1 (100)
Severe (> 75 mm Hg)	5/15 (33)	1/4 (25)	4/11 (36)	3/8 (38)	1/2 (50)	0/1 (0)
Severe (> 75 mm Hg)	7/15 (47)	2/4 (50)	5/11 (45)	3/8 (38)	1/2 (50)	0/1 (0)
Positive for microfilaria	29/34 (85)	12/14 (86)	17/20 (85)	10/13 (77)	2/2 (100)	4/4 (100)
Positive for heartworm antigen	35/36 (97)	14/15 (93)	21/21 (100)	14/14 (100)	2/2 (100)	4/4 (100)
Dyspnea	24/42 (57)	12/21 (57)	12/21 (57)	9/14 (64)	2/2 (100)	1/4 (25)
Hemoglobinuria	29/40 (73)	11/19 (58)	18/21 (86)	11/14 (79)	2/2 (100)	4/4 (100)
Thoracic radiographs						
Right-sided cardiac enlargement	33/35 (94)	13/14 (93)	20/21 (95)	14/14 (100)	2/2 (100)	3/4 (75)
Dilatation of main pulmonary artery	31/35 (88)	12/14 (86)	19/21 (90)	13/14 (93)	2/2 (100)	4/4 (100)
Time to surgery after diagnosis (h)						
Mean ± SD	NA	NA	7.1 ± 5.1	6.6 ± 5.1	5.5 ± 3.5	6.5 ± 2.1
Range	NA	NA	1–19	1–16	3–8	4–9
Anesthesia time (min)						
Mean ± SD	NA	NA	67 ± 55	56 ± 29	34 ± 27	126 ± 99
Range	NA	NA	15–260	20–120	15–53	30–260
No. of heartworms removed						
Mean ± SD	NA	NA	29.9 ± 31.2	33.5 ± 24.0	0 ± 0	39.5 ± 53.9
Range	NA	NA	0–120	4–75	0–0	8–120
Residual heartworms¶	NA	NA	NA	10/13 (77)	NA	NA

*One dog in this group had the procedure discontinued after induction of general anesthesia, when heartworms had migrated to the distal portion of the pulmonary artery and became inaccessible. This dog was excluded from the successful heartworm extraction and survived to discharge group. See text for full description. †A diagnosis of DIC was made when dogs had any 3 of the following: thrombocytopenia, prolongation of prothrombin and activated partial thromboplastin times, decreased antithrombin III concentration, and increased concentration of fibrin degradation products. ‡Body condition score on a range from 1 to 9. §A diagnosis of pulmonary hypertension was made if Doppler ultrasonography estimated a right atrial to right ventricular pressure gradient of > 27 mm Hg. Five of the 20 dogs with documented pulmonary hypertension could not be categorized because the gradient was not recorded in the medical record. ¶As detected on follow-up echocardiography.

NA = Not applicable.

bility and general demeanor. Sedation or premedication was achieved with midazolam (11/21 [52%]) or diazepam (10/21 [48%]) and fentanyl (11/21 [52%]) via bolus injection, or midazolam and fentanyl via continuous rate infusion (11/21 [52%]). General anesthesia was induced with etomidate (16/21 [76%]) and maintained with sevoflurane in oxygen (17/21 [81%]).

Heartworms were removed from the right atrium and right ventricle by use of an endoscopic retrieval device^b that was introduced via right jugular venotomy. In 1 dog, the left jugular vein was used. Lidocaine was administered SC for local analgesia of the neck in 4 (19%) dogs. Intra-

operative medications included administration of lidocaine (8/21 [38%]), cefazolin (6/21 [29%]), albuterol aerosol (6/21 [29%]), dobutamine (3/21 [14%]), atropine (3/21 [14%]), epinephrine (3/21 [14%]), glycopyrrolate (2/21 [10%]), dopamine (2/21 [10%]), diphenhydramine (2/21 [10%]), mannitol (2/21 [10%]), and aminophylline (2/21 [10%]). Because of anemia, 2 dogs (Hct, 18.1%) received packed RBC transfusions and 1 dog (Hct, 17.2%) was given a hemoglobin-based oxygen-carrying solution^c IV.

Transvenous heartworm extraction—Twenty-one dogs had transvenous heartworm extraction attempted.

In 1 dog, heartworms migrated deep into the pulmonary arteries following induction of general anesthesia, making heartworm extraction via catheter impossible. This dog was subsequently discharged from the hospital and was included in the treatment group, but was considered to have had an unsuccessful outcome because catheter extraction was not performed. Two (10%) dogs died during the procedure, 3 (14%) dogs died within 24 hours after surgery, and 1 (5%) dog died 72 hours after surgery. The 2 dogs that died during the procedure experienced cardiac arrest secondary to unresponsive bradycardia. Although normal sinus rhythm was successfully restored in these dogs, neither dog regained consciousness and both dogs were euthanized. The remaining 4 dogs died after surgery. Two of these dogs had evidence of DIC before surgery, and all 4 dogs were hypotensive throughout the procedure; 1 dog developed cardiac arrest that required resuscitation during surgery. One dog developed seizures following the procedure and was euthanized. One dog became agonal and died of aspiration pneumonia of unknown etiology, which was confirmed by necropsy. In one of the remaining dogs, some heartworms were attached to the tricuspid valve and could not be removed, and the other dog developed progressive DIC. Both of these dogs became nonresponsive; the first of these 2 underwent cardiopulmonary arrest, and the second was euthanized. The overall in-hospital mortality rate of dogs undergoing attempted transvenous heartworm extraction was 29% (6/21). The overall success rate of transvenous heartworm extraction defined as dogs that had successful heartworm extraction and survived to discharge was 14 (67%). These 14 dogs had uneventful anesthetic procedures with uneventful recoveries.

The 18 dogs that survived successful transvenous heartworm extraction were recovered in the intensive care unit with oxygen supplementation administered via oxygen cage ($n = 16$) and parenteral analgesia (hydromorphone and buprenorphine) as required. Additional medications included cefazolin ($n = 12$), dalteparin sodium (9), prednisone (14), pimobendan (7), theophylline (4), sildenafil (5), enrofloxacin (2), ampicillin (2), and metronidazole (1). Five dogs received fresh frozen plasma transfusions.

Fourteen of 21 dogs successfully underwent transvenous heartworm extraction and were discharged from the hospital. The mean hospitalization time was 3.6 ± 1.96 days (range, 1 to 8 days). Medications provided upon discharge included prednisone ($n = 14$), ivermectin heartworm preventative (7), sildenafil (6), amoxicillin trihydrate and clavulanate potassium (5), theophylline (3), dalteparin sodium (2), pimobendan (1), doxycycline (1), furosemide (1), enalapril (1), cephalexin (1), and clopidogrel bisulfate^d (1). Discharge instructions included strict rest for a minimum of 1 month and reevaluation in 1 to 3 months for possible initiation of adulticide treatment. The dog that was anesthetized but had no heartworms removed was discharged after 2 days of hospitalization. During hospitalization, a split-dose protocol of immiticide was initiated, and the dog was discharged. The owners were instructed to administer prednisone and heartworm preventative medication, provide strict rest, and follow-up with the primary

care veterinarian for the second and third adulticide injections in 30 to 45 days. This dog was lost to follow-up after discharge.

The treatment group ($n = 21$) was divided into 3 subgroups for comparison as follows: died during procedure (2), died within hours of the procedure (4), and successful heartworm extraction and survived to discharge (14). For the purpose of follow-up analysis, the 1 dog in which the heartworms migrated to the distal portion of the pulmonary artery following general anesthesia was not included in the successful heartworm extraction and survived to discharge group (Table 1). There was no significant difference in the time from admission to time the procedure was performed, or anesthesia time, when the 3 treatment subgroups were compared. The mean number of heartworms removed from the 21 dogs that had extraction attempted was 29.9 ± 31.2 with a range of 0 to 120. No heartworms were removed from the 2 dogs that died during surgery or from the dog with heartworm migration into the pulmonary arteries. The mean number of heartworms removed in the 14 dogs that survived to be discharged versus the dogs that died in the postoperative period was not significantly different. Thirteen of the 14 dogs with successful heartworm extraction that survived to discharge underwent postoperative echocardiography. Ten of these 13 dogs had residual heartworms in the pulmonary artery.

The progress of the 14 dogs that underwent successful heartworm extraction and survived to discharge was followed. Mean follow-up time for these dogs was 24.4 ± 17.7 months, with a range of 2 to 56 months. At the time of final follow-up, 11 of the 14 dogs discharged from the hospital had survived at least 18 months and 6 of these dogs had survived > 24 months. By the end of the study, 1 dog was lost to follow-up and 3 had been euthanized for unrelated issues. The probability that a dog undergoing transvenous heartworm extraction would live at least 6 months was 65% (95% CI, 41% to 82%), and the probability that it would survive 50 months was 33% (95% CI, 2% to 73%; Figure 1).

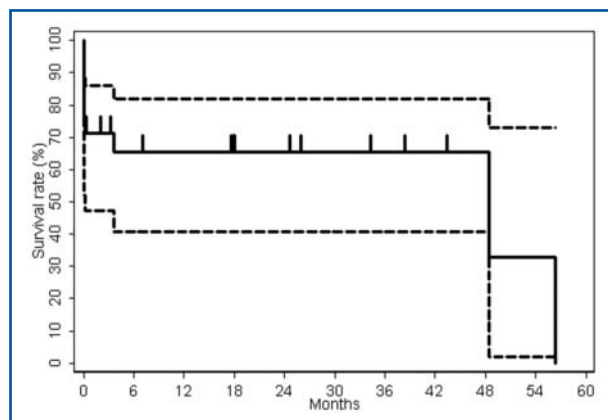


Figure 1—Kaplan-Meier survival curve for 20 dogs with CS treated by means of transvenous catheter extraction. Vertical hatch marks indicate dogs that were lost to follow-up or died of causes unrelated to HW or CS. The single dog that underwent the procedure but failed to have any heartworms removed and survived to discharge was not included in the model. Values are shown with 95% confidence intervals (dashed lines).

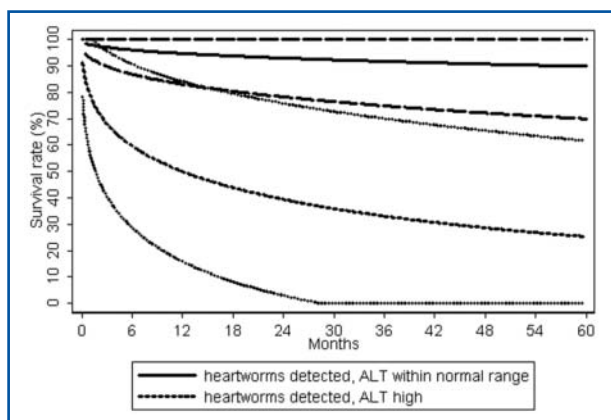


Figure 2—Effect of high preoperative serum ALT activity and the presence of heartworms in the pulmonary arteries (as detected by echocardiography prior to surgery) on survival rate for 18 dogs with CS. Values are shown with 95% confidence intervals (dashed lines).

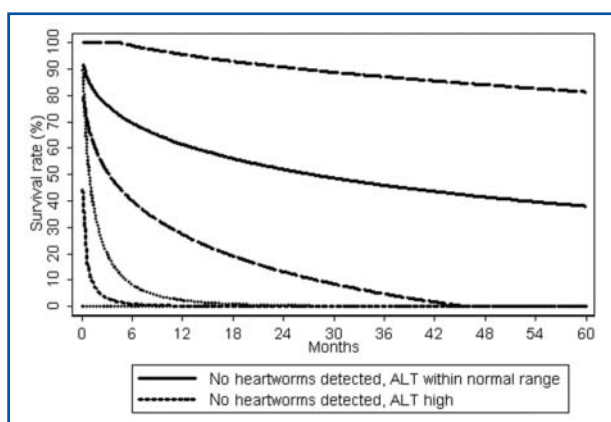


Figure 3—Effect of high preoperative serum ALT activity on survival rate for 18 dogs with CS but without heartworms detected in the pulmonary arteries. Values are shown with 95% confidence intervals (dashed lines).

A parametric proportional hazards model with a Weibull distribution was performed to evaluate the effect of the preoperative clinicopathologic or diagnostic imaging results on survival time. High serum ALT activity ($P = 0.02$) and the presence of heartworms in the pulmonary artery ($P = 0.03$) before surgery were found to have significant effects on survival rate (Figures 2 and 3). Dogs with high serum ALT activities had a lower overall survival rate than dogs with ALT activities within reference limits. Based on the model, if a dog had both high serum ALT activity and heartworms in the pulmonary artery, predicted survival would be 0%. However, because of the sample size of only 18, with 9 deaths and 9 censored observations, these findings may not be representative of a larger population.

Discussion

Heartworm CS, while relatively uncommon, affects 16% to 20% of dogs with HWD.⁴ However, it is a life-threatening and expensive complication of heartworm infection that requires rapid decisive action by owners and veterinarians. The goal of this study was to offer new data on prognosis and survival rate of dogs with CS.

The clinical signs of dyspnea, hemoglobinuria, jugular distension, pale mucus membranes, weak femoral pulses, and presence of a heart murmur that were observed in our patients on first examination were consistent with previously reported clinical signs of CS.^{4,5} The presence of hepatic and renal dysfunction, anemia, leukocytosis, hemolysis, and DIC was also consistent with previous reports.^{1,3-5,9} In the present study, there was a significant reduction in survival rate in dogs with high serum ALT activities. In CS, elevations in ALT activity may be associated with passive hepatic congestion, inadequate cardiac output, DIC, hepatic thrombosis, or an overall proinflammatory state. In our retrospective study, high serum ALT activity was significantly associated with a decreased survival rate. Additionally, based on the model, if a dog had both a high serum ALT activity and heartworms in the pulmonary artery on initial examination, mortality rate was predicted to be 100%. Documentation of heartworms in the pulmonary artery before surgery may be associated with higher heartworm burdens and may reduce the number of accessible heartworms that can be retrieved during a transvenous procedure. This may then result in higher postretrieval heartworm burdens, increasing complication rates, and ultimately reduced survival. As in any retrospective study, interpretation of the results is at best speculative and represents the limitations of this study.

Most published studies^{12,13} do not report mortality rates, but it has been suggested that mortality rates of 30% to 40% are to be expected in dogs with CS,⁵ with a poor to guarded prognosis even with heartworm extraction.^{5,10} Disseminated intravascular coagulation and multiorgan failure have been reported as the primary causes of death, either before or after transvenous heartworm extraction.^{5,13} In the present study, the in-hospital mortality rate for dogs undergoing attempted transvenous heartworm extraction was 29% (6/21), which is similar to a previous report.⁵ Fourteen (67%) dogs successfully underwent transvenous heartworm extraction and were discharged from the hospital. However, most of these dogs had residual heartworms in the pulmonary arteries. Mean follow-up time in the 14 discharged dogs was 24.4 ± 17.7 months, with a range of 2 to 56 months. To date, of the 14 dogs discharged from the hospital, 11 have survived at least 18 months, and 6 of these have survived > 24 months. Medical treatment with an adulticide was recommended in most of these 14 dogs, but follow-up information was not available because ongoing treatment was administered by referring veterinarians. Of the remaining 3 dogs, 2 were lost to follow-up and 1 was euthanized because of intervertebral disc disease. These results suggest that dogs with CS that undergo successful minimally invasive surgical treatment via transvenous heartworm extraction and survive to be discharged from the hospital have a good long-term prognosis.

- StataCorp LP, College Station, Tex.
- Endoscopic retrieval basket (4-wire basket, 2.8-mm external diameter, 165-cm length), Olympus, San Jose, Calif.
- Oxyglobin, Biopure, Cambridge, Mass.
- Plavix, Bristol-Myers Squibb, New York, NY.

References

1. Calvert CA, Thomason JD. Heartworm disease. In: Tilley LP, Smith FW, Oyama M, et al, eds. *Manual of canine and feline cardiology*. 4th ed. St Louis: Elsevier, 2008;183–199.
2. Atkins C. Canine heartworm disease. In: Ettinger SJ, Feldman EC, eds. *Textbook of veterinary internal medicine*. 6th ed. Philadelphia: Elsevier Inc, 2005;1118–1136.
3. Calvert CA, Rawlings CA, McCall J. Canine heartworm disease. In: Fox PR, Sisson D, Moise NS, eds. *Textbook of canine and feline cardiology*. 2nd ed. Philadelphia: WB Saunders Co, 1999;702–726.
4. Hoch H, Strickland KN. Canine and feline dirofilariasis: life cycle, pathophysiology, and diagnosis. *Compend Contin Educ Pract Vet* 2008;30:133–140.
5. Kittleson MD. Heartworm infection and disease (dirofilariasis)—caval syndrome. In: *Small animal cardiovascular medicine textbook*. 2nd ed. St Louis: Mosby, 1998;370–401. Available at: www.vin.com/Members/Proceedings/Proceedings.plx?CID=SA RCARDIO&PID=12537%O=VIN. Accessed Sep 30, 2008.
6. Nelson CT, McCall JW, Rubin SB, et al. 2005 Guidelines for the diagnosis, prevention and management of heartworm (*Dirofilaria immitis*) infection in dogs. *Vet Parasitol* 2005;133:255–266.
7. Dunavent B, Keister DM, Tanner PA, et al. Correlation between heartworm disease classification, serum antigen concentration and associated clinical pathology parameters. In: Soll MD, Knight DH, ed. *Proceedings of the heartworm symposium '95*. Batavia, Ill: American Heartworm Society, 1995.
8. Polizopoulou ZS, Koutinas AF, Saridomichelakis MN, et al. Clinical and laboratory observations in 91 dogs infected with *Dirofilaria immitis* in northern Greece. *Vet Rec* 2000;146:466–469.
9. Strickland KN. Canine and feline caval syndrome. *Clin Tech Small Anim Pract* 1998;13:88–95.
10. Hoch H, Strickland KN. Canine and feline dirofilariasis: prophylaxis, treatment, and complications of treatment. *Compend Contin Educ Pract Vet* 2008;30:146–151.
11. Atkins CE, Keene BW, McGuirk SM. Investigation of caval syndrome in dogs experimentally infected with *Dirofilaria immitis*. *J Vet Intern Med* 1988;2:36–40.
12. Kitagawa H, Sasaki Y, Ishihara K, et al. Cardiopulmonary function values before and after heartworm removal in dogs with caval syndrome. *Am J Vet Res* 1991;52:126–132.
13. Kitagawa H, Kitoh K, Ohba Y, et al. Comparison of laboratory test results before and after surgical removal of heartworms in dogs with vena caval syndrome. *J Am Vet Med Assoc* 1998;213:1134–1136.



From this month's AJVR

Effect of exercise and osteochondral injury on synovial fluid and serum concentrations of carboxy-terminal telopeptide fragments of type II collagen in racehorses

Orlaith B. Cleary et al

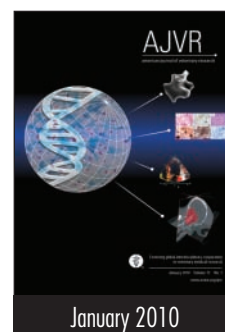
Objective—To investigate the effects of exercise and osteochondral injury on concentrations of carboxy-terminal telopeptide fragments of type II collagen (CTX-II) in synovial fluid (SF) and serum of Thoroughbred racehorses and to compare findings with radiographic and arthroscopic scores of joint injury severity.

Animals—78 Thoroughbreds with (n = 38) and without (40) osteochondral injury.

Procedures—Serum and metacarpophalangeal or carpal joint SF samples were collected from noninjured horses before and at the end of 5 to 6 months of race training (pre- and postexercise samples, respectively) and from horses with osteochondral injury (1 joint assessed/horse). Synovial fluid and serum CTX-II concentrations were determined by use of an ELISA. Radiographic and arthroscopic scores of joint injury severity were determined for the injured horses.

Results—The CTX-II concentrations in SF and SF:serum CTX-II ratio were significantly higher for horses with joint injuries, compared with pre- and postexercise findings in noninjured horses. Serum CTX-II concentrations in postexercise and injured-horse samples were significantly lower than values in pre-exercise samples. On the basis of serum and SF CTX-II concentrations and SF:serum CTX-II ratio, 64% to 93% of serum and SF samples were correctly classified into their appropriate group (pre-exercise, postexercise, or injured-joint samples). In horses with joint injuries, arthroscopic scores were positively correlated with radiographic scores, but neither score correlated with SF or serum CTX-II concentration.

Conclusions and Clinical Relevance—Results suggest that serum and SF CTX-II concentrations and SF:serum CTX-II ratio may be used to detect cartilage degradation in horses with joint injury. (*Am J Vet Res* 2010;71:33–40)



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