Identification of *Heterobilharzia americana* infection in a dog residing in Indiana with no history of travel

Jessica Y. Rodriguez DVM  
Joseph W. Camp PhD  
Stephen D. Lenz DVM, PhD  
Kevin R. Kazacos DVM, PhD  
Karen F. Snowden DVM, PhD

From the Department of Veterinary Pathobiology, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX 77843 (Rodriguez, Snowden); and the Department of Comparative Pathobiology (Camp, Lenz, Kazacos) and Indiana Animal Disease Diagnostic Laboratory (Lenz), College of Veterinary Medicine, Purdue University, West Lafayette, IN 47907.

Address correspondence to Dr. Rodriguez (jyrodriguez@cvm.tamu.edu).

A 1-year-old 28.5-kg (62.7-lb) castrated male Great Dane–German Shepherd mix was evaluated by its veterinarian because of chronic intermittent vomiting, with apparently normal appetite and bowel movements. On physical examination, the dog was presumed to have gastroenteritis as a result of NSAID administration. The dog was treated with sucralfate (35 mg/kg [15.9 mg/lb], PO, q 12 h for 14 days), famotidine (0.35 mg/kg [0.16 mg/lb], PO, q 12 h for 14 days), and metoclopramide (0.35 mg/kg, PO, q 12 h for 7 days). Three months after completion of this combination treatment, clinical signs returned; fecal samples were analyzed, but no parasites were detected via fecal flotation or microscopic examination of direct fecal-saline smear. The same dosages of sucralfate and famotidine were prescribed for another 10-day period.

Metronidazole (26.3 mg/kg [12 mg/lb], PO, q 12 h for 10 days) was added to the treatment regimen. One day after completion of this treatment, the dog was returned to the veterinary clinic because of recurrence of vomiting. The owner reported that the dog was well while receiving the medications but that the vomiting returned after the end of treatment. The dog also now had diarrhea with noticeable blood content. Abdominal radiography revealed no major abnormalities.

Treatment with porcine pancreatic enzyme concentrate (32 mg/kg [14.5 mg/lb], PO, q 12 h for 21 days), metronidazole (28.2 mg/kg [12.8 mg/lb], PO, q 12 h for 10 days), and amoxicillin (18.8 mg/kg [8.5 mg/lb], PO, q 12 h for 10 days) was initiated. However, the dog was returned to the veterinary clinic 2 days after the 10-day treatment period because of relapse of clinical signs (vomiting and frequent excretion of small amounts of loose feces). Contrast radiography was performed after administration of 20 mL of barium orally; radiographic views were obtained immediately and at 10 minutes, 2 hours, and 4 hours after...
contrast. At 4 hours, barium retained in the intestinal tract revealed a possible partial obstruction at the ileocecal junction. The dog underwent an exploratory laparotomy the following day.

During surgery, diffusely distributed, nodular, intramural lesions were observed in the jejunum. Two biopsy samples were collected from representative lesions. Given that the pancreas appeared grossly swollen, the dog was presumed to have pancreatitis. No other abnormalities were observed in the abdomen. The biopsy specimens were submitted to the Indiana Animal Disease Diagnostic Laboratory at Purdue University. After recovery from anesthesia, repeated treatment with metronidazole (28.2 mg/kg, PO, q 12 h) and sucralfate, with the addition of tramadol (1.8 mg/kg [0.8 mg/lb], PO, q 12 h), for 10 days was initiated.

Microscopic examination of the jejunal biopsy specimens revealed granulomatous mural enteritis, characterized by coalescing granulomas in the submucosa and muscular tunics. The granulomas were composed of epithelioid macrophages, lymphocytes, fewer plasma cells and eosinophils, and scattered multinucleated giant cells. Granulomas surrounded eggs with a yellow-brown shell that enclosed a miracidium with multiple nuclei (Figure 1). Mineralized and effaced trematode eggs were also present in granulomas. *Heterobilharzia americana* infection was suspected, and this diagnosis was confirmed at the College of Veterinary Medicine and Biomedical Sciences, Texas A&M University by PCR amplification and sequencing a portion of the 18S ribosomal RNA gene of the parasite from DNA extracted from formalin-fixed, paraffin-embedded small intestinal tissue samples. The 487-bp sequence shared 100% identity with other published sequences of this gene region of *H americana* in GenBank.

The dog was prescribed fenbendazole suspension (48 mg/kg [21.8 mg/lb], PO, q 24 h) for 10 days; cephalaxin (18.5 mg/kg [8.4 mg/lb], PO, q 12 h for 10 days) and carprofenb (3.7 mg/kg [1.7 mg/lb], PO, q 24 h for 7 days) were prescribed because of the risk of possible incidental infection and associated inflammation. Metronidazole, sucralfate, and tramadol were continued at their previous dosages for 10 days. The dog began vomiting and defecating soft stools 9 days after the treatments started. Two days later, treatment with fenbendazole was repeated for 7 days. Nine weeks after fenbendazole treatment ceased, the dog was returned for evaluation because of intermittent vomiting and diarrhea of 3 weeks’ duration. The client remarked that the dog was well while receiving treatment with fenbendazole. A physical examination, CBC, and serum biochemical analyses revealed no important abnormalities. Fenbendazole treatment was repeated at the same dosage as before for 10 days. One week after completing the fenbendazole treatment, a sample of feces was analyzed by fecal saline sedimentation (performed at Purdue University) and yielded several *H americana* eggs. The dog was prescribed praziquantel (25 mg/kg [11.4 mg/lb], PO, q 8 h) for 2 days. Four months after the praziquantel treatment ceased, the dog underwent a routine examination and received vaccinations, at which time it had no gastrointestinal signs.

After the diagnosis of *H Americana* infection, the owner was questioned regarding the history of the dog. At 12 weeks of age, the dog was adopted from an animal shelter in Fort Wayne, Ind, although the owner lived in Roanoke, Ind. At that time, the dog had 2 sources of possible water exposure: Roanoke Creek and a home aquarium that contained aquatic plants from Lake Tippecanoe, Ind. Ten months after adoption, the owner moved to a home in Fort Wayne, Ind, that had a pond on the property. The original owner of the dog, whose bitch gave birth to the adopted dog, verified that the adopted dog had been born in Indiana and had never traveled outside the state. A shelter employee verified that the dog did not leave the animal shelter until the time of adoption by the current owner.

**Discussion**

*Heterobilharzia americana* (Family: Schistosomatidae) is a trematode parasite that lives in the mesenteric vessels of various wild and domestic mammals. A larval form (miracidium) hatches from the egg on contact with a freshwater source (eg, lakes, ponds, and irrigation areas) and penetrates a lymnaeid snail (intermediate host). After asexual reproduction and development in the snail, fork-tailed cercariae are released in water and penetrate the skin of the mammal host. After migration through the lungs and development in the liver, adult worms live and mate in the host’s mesenteric veins. Parasite eggs migrate from the vasculature into the intestinal lumen and exit in the feces. In dogs, eggs along...
with associated granulomatous inflammation have also been located in the liver, pancreas, mesenteric lymph nodes, lungs, spleen, and stomach.3 Common clinical signs in infected dogs include diarrhea, weight loss, anorexia or hyporexia, vomiting, hematochezia, lethargy, polyuria, and polydipsia.3

According to the owner of the dog of the present report, clinical signs of infection were noticeable 2 months after the move to Fort Wayne, Ind. Thus, the pond on that new property was the likely source of exposure, given the parasite’s prepatent period of 61 to 75 days.2,4,5 More than 100 lymnaeid snails were collected from the pond during midsummer after diagnosis of *H americana* infection in the dog; however, no cercarial shedding from the snails was observed for a 2-week period. It is possible that at the time of collection, the snails were not infected or that not enough snails were collected.

For the dog of this report, the return of clinical signs after 3 fenbendazole treatments was due to either reinfection or persistent infection. During the snail collection, the dog was observed in the water; therefore, it is possible that the dog became reinfected if it entered the pond at a time when the snails were shedding cercariae. The apparent success of the praziquantel treatment supported the idea that the dog was persistently infected despite the fenbendazole treatments.

Currently suggested treatments for *H americana* infection are fenbendazole granules (40 mg/kg [18.2 mg/lb], PO, q 24 h for 10 days) and praziquantel (25 mg/kg, PO, q 8 h for 2 days). The fenbendazole dosage is based on data from an experimentally infected and treated dog.3 The praziquantel dosage is extrapolated from a dog with paragonimiasis that was successfully treated with the drug.2,6 Several other case reports9–12 involving dogs describe various treatments that include combining the 2 drugs; however, data from clinical trials performed to compare different treatment protocols have not been published. In a case series of 238 infected dogs, 26 had *H americana* infection detected at least 1 more time after initial diagnosis and treatment.3 Given that the return of clinical signs in the dog of the present report appeared to be attributable to persistent infection despite treatment, repeated treatments of *H Americana*-infected dogs seem warranted. A study13 of humans infected with a related parasite, *Schistosoma mansoni*, revealed that repeated dosing with praziquantel at 2-week intervals increased the cure rate. The success of this regimen is thought to be due to the presence of juvenile worms that are not initially susceptible to the drug, although additional treatments also may be needed in patients with high numbers of parasites.14

In the case described in this report, the apparent success of the praziquantel treatment, compared with fenbendazole treatment, suggested that praziquantel is more effective. The apparent poorer performance of the fenbendazole may be related to the formulation that was used. In the experimentally treated dog from which the dosage of fenbendazole is based, the formulation used was fenbendazole granules® that are to be mixed with food.6 The dog of the present report was treated with fenbendazole suspension. It is possible that the dog was not appropriately administered each dose of the liquid formulation. Also, it is not known whether the dog was administered the fenbendazole with or without food. Systemic concentrations of the drug are significantly higher when fenbendazole is administered with food.15

The geographic distribution of *H americana* primarily involves the Gulf Coast and south Atlantic states.15 *Heterobilharzia americana* infection was recently reported in raccoons and dogs in Kansas.11,17 These were the first reports of this parasitic infection in animals in the Midwest. The case described in the present report expands the distribution of this parasite in this region. The presence of this parasite in the Midwest was speculated to be more likely a result of translocation of raccoons from endemic areas by private hunters than a result of natural northerly spread of the parasite. The reasoning behind this speculation was based on the absence of prior reports of *H americana* infection from Oklahoma and Arkansas, the 2 states located between Kansas and the endemic region.13 Recently, however, canine cases of *H americana* infection in Oklahoma and Arkansas have also been documented.3,18

The clinical effects of *H americana* infection in dogs are nonspecific and can range from no apparent clinical signs to death.3 Owing to the parasite’s potential for continuing geographic expansion, infection with *H americana* should be considered by veterinarians in both endemic and nonendemic regions to facilitate timely and appropriate diagnosis and treatment of affected dogs.

**Acknowledgments**

There were no sources of funding, financial conflicts of interest, or disclaimers.

**Footnotes**


c. Panacur granules 22.2%, Merck Animal Health, Summit, NJ.

**References**


From this month’s *AJVR*

**Effects of anesthetic induction with a benzodiazepine plus ketamine hydrochloride or propofol on hypothermia in dogs undergoing ovariohysterectomy**

Jennifer L. Bornkamp et al

**OBJECTIVE**
To assess the effect of anesthetic induction with a benzodiazepine plus ketamine or propofol on hypothermia in dogs undergoing ovariohysterectomy without heat support.

**ANIMALS**
23 adult sexually intact female dogs undergoing ovariohysterectomy.

**PROCEDURES**
Baseline rectal temperature, heart rate, and respiratory rate were recorded prior to premedication with buprenorphine (0.02 mg/kg, IM) and acepromazine (0.05 mg/kg, IM). Anesthesia was induced with midazolam or diazepam (0.25 mg/kg, IV) plus ketamine (5 mg/kg, IV; n = 11) or propofol (4 mg/kg, IV; n = 12) and maintained with isoflurane in oxygen. Rectal temperature was measured at hospital intake, prior to premedication, immediately after anesthetic induction, and every 5 minutes after anesthetic induction. Esophageal temperature was measured every 5 minutes during anesthesia, beginning 30 minutes after anesthetic induction. After anesthesia, dogs were covered with a warm-air blanket and rectal temperature was measured every 10 minutes until normothermia (37°C) was achieved.

**RESULTS**
Dogs in both treatment groups had lower rectal temperatures within 5 minutes after anesthetic induction and throughout anesthesia. Compared with dogs that received a benzodiazepine plus ketamine, dogs that received a benzodiazepine plus propofol had significantly lower rectal temperatures and the interval from discontinuation of anesthesia to achievement of normothermia was significantly longer.

**CONCLUSIONS AND CLINICAL RELEVANCE**
Dogs in which anesthesia was induced with a benzodiazepine plus propofol or ketamine became hypothermic; the extent of hypothermia was more profound for the propofol combination. Dogs should be provided with adequate heat support after induction of anesthesia, particularly when a propofol-benzodiazepine combination is administered. (*Am J Vet Res* 2016;77:351–357)