

Tick paralysis in a free-ranging bobcat (*Lynx rufus*)

Meredith E. Persky DVM

Yousuf S. Jafarey DVM

Sarah E. Christoff

Dewey D. Maddox

Stephanie A. Stowell

Terry M. Norton DVM

From the Department of Animal Health, Jacksonville Zoo and Gardens, 370 Zoo Pkwy, Jacksonville, FL 32218 (Persky, Jafarey, Christoff, Maddox); Georgia Sea Turtle Center, 214 Stable Rd, Jekyll Island, GA 31527 (Stowell, Norton); and Jekyll Island Authority, 100 James Rd, Jekyll Island, GA 31527 (Norton).

Address correspondence to Dr. Persky (perskym@jacksonvillezoo.org).

CASE DESCRIPTION

A free-ranging male bobcat (*Lynx rufus*) was evaluated because of signs of pelvic limb paralysis.

CLINICAL FINDINGS

Physical examination of the anesthetized animal revealed tick infestation, normal mentation, and a lack of evidence of traumatic injuries. Radiography revealed no clinically relevant abnormalities. Hematologic analysis results were generally unremarkable, and serologic tests for exposure to feline coronavirus, FeLV, FIV, and *Toxoplasma gondii* were negative. Results of PCR assays for flea- and common tick-borne organisms other than *Bartonella clarridgeiae* were negative.

TREATMENT AND OUTCOME

Ticks were manually removed, and the patient received supportive care and fipronil treatment. The bobcat made a full recovery within 72 hours after treatment for ticks, and a presumptive diagnosis of tick paralysis was made. Identified tick species included *Dermacentor variabilis*, *Amblyomma americanum*, and *Ixodes scapularis*.

CLINICAL RELEVANCE

To the authors' knowledge, tick paralysis has not previously been reported in felids outside Australia. This disease should be considered a differential diagnosis in felids, including exotic cats, with signs of neuromuscular disease of unknown etiopathogenesis. (*J Am Vet Med Assoc* 2020;256:362–364)

A 7.53-kg (16.57-lb) free-ranging male bobcat (*Lynx rufus*), estimated to be 15 months old, was reported to be injured on Jekyll Island, Ga. Staff from the Jekyll Island Authority-Georgia Sea Turtle Center observed that the animal appeared unable to stand and that it had remained in 1 location overnight. The bobcat was restrained with a catch pole and was immobilized with ketamine hydrochloride^a (4 mg/kg [1.8 mg/lb], IM), dexmedetomidine hydrochloride^b (0.04 mg/kg [0.018 mg/lb], IM), and butorphanol tartrate^c (0.4 mg/kg [0.18 mg/lb], IM) by direct (hand) injection for transport to the Jacksonville Zoo and Gardens (day 0). The bobcat was recovering from anesthesia in a crate upon arrival at the zoo, limiting the clinician's ability to evaluate its mentation or attempts to ambulate. Further sedation was necessary to allow safe handling for examination and diagnostic testing.

The bobcat was anesthetized with a combination of tiletamine hydrochloride and zolazepam hydrochloride^d (10 mg/kg [4.5 mg/lb], IM) delivered by use of a pole syringe, and anesthesia was maintained with isoflurane^e in oxygen delivered via face mask at 2 L/min. On examination, thin body condition was noted, and no external signs of trauma were present. Although infested with ticks, the bobcat was afebrile (rectal temperature, 38.5°C [101.3°F]), and all palpable lymph nodes were considered to be of normal size. The remaining physical examination findings were unremarkable. Blood was collected for point-of-care testing with a handheld blood analyzer^f and laboratory assays^g including a CBC and serum biochemical analysis (with measurement of total thyroxine concentration), measurement of anti-feline coronavi-

rus antibody titers, testing for FeLV antigen and antibodies against FIV and *Toxoplasma gondii* (IgG and IgM), and PCR assays for feline flea- and tick-borne pathogens, including *Anaplasma phagocytophilum*, *Bartonella henselae*, *Bartonella clarridgeiae*, *Bartonella quintana*, *Ehrlichia* spp, *Mycoplasma hemofelis*, *Mycoplasma hemominutum*, *Mycoplasma turicensis*, *Rickettsia rickettsia*, and *Rickettsia felis*. Although bobcats are natural reservoir hosts for *Cytauxzoon felis*,^{1,2} no evidence of hemoparasites was observed on clinicopathologic assessment of the blood smear slide prepared for a CBC.⁸

Radiographs of the skull, vertebral column, and forelimbs were obtained. Growth plates were still visible, and no clinically relevant radiographic lesions were noticed. The initial differential diagnoses for pelvic limb paresis included trauma, intervertebral disk disease, fibrocartilaginous embolism, aortic thromboembolism, sarcocystosis, toxoplasmosis, botulism, myasthenia gravis, tick paralysis, and rabies. Approximately 20 ticks were easily seen on the animal and were manually removed. The initial treatment plan was tailored toward management of a potential spinal cord injury and supportive care; treatments included administration of a long-acting corticosteroid (methylprednisolone^h [2.7 mg/kg [1.2 mg/lb], IM, once), lactated Ringer solutionⁱ (50 mL/kg [22.7 mg/lb], SC), long-acting ceftiofur crystalline-free acid^j (6.6 mg/kg [3 mg/lb], SC), D- α -tocopherol^k (20 U/kg [9.1 U/lb], SC), ivermectin^l (0.2 mg/kg [0.09 mg/lb], SC), and sustained-release buprenorphine^m (0.12 mg/kg, [0.05 mg/lb], SC). The patient had prolonged recovery from anesthesia, and atipamezoleⁿ (0.3 mg/kg [0.14 mg/lb],

IM) was administered several hours later to reverse the effects of dexmedetomidine that was given when the bobcat was captured for initial transport. The animal was hospitalized for monitoring and further care.

The WBC count was within the reference range, although a relative mild basophilia (5% basophils; reference range, 0% to 1%)^o was noted. The PCR assay results⁸ were positive for *B. clarridgeiae*, which is commonly isolated from cats without clinical signs of disease,^{3,4} and negative for the remaining flea- and tick-borne pathogens tested on the panel. Hyperglycemia (blood glucose concentration, 387 mg/dL; reference range, 90 to 302 mg/dL)^o was present, which was attributed to stress related to handling and inability to stand. Mild hypochloridemia (blood chloride concentration, 105 mEq/L; reference range, 110 to 128 mEq/L)^o was reported but was not considered to be a clinically relevant finding. The remaining laboratory results were within the respective reference ranges.

The following day (day 1), the bobcat was lying in sternal recumbency with normal mentation and apparent control of its head. Paresis was noted upon stimulation of the pelvic limbs. Owing to suspected lower motor deficits and a lack of urine output, the bobcat was immobilized with ketamine, dexmedetomidine, and butorphanol as previously described to allow expression of the urinary bladder and a recheck physical examination. Blood and urine samples were collected at this time for additional analyses with a benchtop analyzer^p and dipstick test,^q respectively. Close inspection of the integument revealed additional ticks along the ears, neck, and interdigital regions that had not been detected for removal during the initial evaluation. Multiple ticks were collected and submitted to a consulting service^r for identification. Three species (*Dermacenter variabilis* [4 adults], *Amblyomma americanum* [5 adults, 5 nymphs, and 2 larvae], and *Ixodes scapularis* [3 adults and 2 nymphs]) were identified.

Tick infestation in conjunction with the observed clinical signs supported tick paralysis as the leading differential diagnosis in the bobcat. A copious amount of fipronil^s spray was applied topically over the animal's entire body. Penicillin G procaine^t (44,000 U/kg [20,000 U/lb], SC) and lactated Ringer solution (50 mL/kg, SC) were administered. Although the bobcat had received a long-acting steroid at the time of initial examination, a dose of dexamethasone sodium phosphate^u (0.2 mg/kg, IM) was administered for anti-inflammatory purposes because there had been minimal improvement in the bobcat's condition. In-house plasma biochemical analysis^p results revealed mild hyponatremia (sodium concentration, 141 mEq/L; reference range, 142 to 178 mEq/L)^o and potassium concentration at the upper limit of the reference range (6.1 mEq/L; reference range, 2.8 to 6.1 mEq/L).^o Glucosuria (1,000 mg of glucose/dL) was detected by means of in-house dipstick^q analysis, although the plasma glucose concentration was within the reference range^o on hematologic analysis. Dex-

medetomidine effects were partially reversed with atipamezole at the previously described dosage, and recovery from the immobilization was unremarkable.

On day 2, the bobcat was able to raise itself into a sitting position and had urinated on its own. Dexamethasone sodium phosphate (0.2 to 0.5 mg/kg [0.09 to 0.23 mg/lb], IM) was administered via pole syringe every 24 hours over the next 3 days because the bobcat was not reliably eating medicated food items. On day 3, within 48 hours after the second examination and treatment for tick infestation, the bobcat was able to raise its hind quarters from a seated position and take a few steps with weak movement of the pelvic limbs. A deep pain response and withdrawal reflex were successfully elicited for both pelvic limbs. Considering the marked improvement in its attitude and pelvic limb movement, the bobcat was transferred to a larger enclosure with video surveillance to assess its ability to ambulate on day 4. The animal was observed walking within this hospital stall within 72 hours after the treatment for ticks (day 4).

Because rickettsial disease had not been definitively ruled out at this time and the bobcat began consuming medicated food items, prophylactic treatment with doxycycline hyclate^v (6.7 mg/kg [3 mg/lb], PO, q 12 h for 7 days) was initiated on day 4. Corticosteroid treatment was also transitioned from dexamethasone sodium phosphate to prednisolone^w (1 mg/kg [0.45 mg/lb], PO, q 24 h for 5 days, then 0.67 mg/kg [0.3 mg/lb], PO, once). The bobcat was kept hospitalized for treatment and monitoring for 1 additional week before release. On day 11, the bobcat was immobilized as previously described via pole syringe. Blood was collected for a CBC⁸ and serum biochemical⁸ analysis. The WBC count was within the reference range,^o and basophilia had resolved. The serum creatinine concentration was moderately low (0.4 mg/dL; reference interval, 0.9 to 3.9 mg/L),^o and remaining serum biochemical values were within respective reference intervals. A final dose of dexamethasone sodium phosphate (0.2 mg/kg, IM, once) was administered.

A microchip was placed in the interscapular region, and the bobcat was administered a rabies vaccine,^x was fitted with a satellite collar, had its ear notched, and was placed in a kennel for transport back to Jekyll Island. Naltrexone^y (2.4 mg/kg [1.1 mg/lb], IM) and atipamezole (as previously described) were administered. The bobcat recovered well from the immobilization and was returned to its original location, where it was released uneventfully. Satellite collar tracking and camera surveillance subsequently identified the same bobcat in multiple locations within its habitat on Jekyll Island.^z

Discussion

Tick paralysis is an acute ascending neuromuscular paralysis that is transmitted to human or animal hosts by approximately 60 species of ticks worldwide.⁵⁻⁷ The neurotoxin in the salivary glands of the

tick blocks the release of transmitters from motor nerve terminals, resulting in a flaccid paralysis that is frequently first observed as hind limb incoordination.^{1,5,7,8} Ataxia of the hind limbs progresses to paralysis and ascends to the forelimbs, neck, head, and respiratory muscles, leading to death if left untreated.^{1,5,8,9} Mydriasis, vomiting, regurgitation, dysphonia, hypotonus, and dysfunction of the urinary bladder can also occur.^{2,5,6,9} A presumptive diagnosis is made on the basis of clinical signs and the detection of ticks, along with a positive response to treatment.^{2,5,6,8} Treatment involves tick removal, supportive care, and administration of tick antitoxin serum.^{1,2,5,9} In many cases, removal of the ticks can result in full recovery in the following 24 to 72 hours.^{5,6}

Tick paralysis has been reported in domestic dogs and cattle and, rarely, in free-ranging wildlife, including mouflon (*Ovis ammon musimon*), various avian species, gray foxes (*Urocyon cinereoargenteus*), red wolves (*Canis rufus*), California mule deer (*Odocoileus hemionus californicus*), black-tailed deer (*Odocoileus hemionus columbianus*), bison (*Bison bison*), black bears (*Ursus americanus*), and a western harvest mouse (*Reithrodontomys megalotis*).^{1,6–8,10,11}

Although, to the authors' knowledge, tick paralysis has not previously been reported in cats in the United States, it is commonly diagnosed in domestic felids in Australia that are infested with *Ixodes holocyclus* and *Ixodes cornuatus*.^{2,5,9} Three different species of ticks presumed to cause tick paralysis,^{1,5} *D variabilis*, *A americanum*, and *I scapularis*, were removed from the bobcat of this report. Given the wide distribution of tick species in North America, it may be prudent to include tick paralysis as a differential diagnosis in felids, including exotic cats, that have progressive neuromuscular signs of idiopathic etiopathogenesis.

Acknowledgments

The authors thank Dr. Rick Alleman for confirming tick identification; Yank Moore and Joseph Colbert, Jekyll Island Authority, for the transport and follow-up tracking of the bobcat; and Melody Woods and Jessica Robinson, Jacksonville Zoo and Gardens, for patient monitoring and care.

Footnotes

- a. Ketaset, Fort Dodge Animal Health, Fort Dodge, Iowa.
- b. Putney, Portland, Me.
- c. Torbugesic, Fort Dodge Animal Health, Fort Dodge, Iowa.
- d. Telazol, Zoetis, Parsippany, NJ.

- e. Isoflo, Abbott Laboratories, Chicago, Ill.
- f. iSTAT1 Handheld Analyzer, Abaxis, Union City, Calif.
- g. Antech Diagnostics, Regional Laboratory, Jacksonville, Fla.
- h. Depo-Medrol, Zoetis, Parsippany, NJ.
- i. Hospira Inc, Lake Forest, Ill.
- j. Excede, Zoetis, Parsippany, NJ.
- k. Vitamin E-300, VetOne, MWI Animal Health, Boise, Idaho.
- l. Vetrimec 1%, VetOne, MWI Animal Health, Boise, Idaho.
- m. Buprenorphine SR, ZooPharm Formulation, Windsor, Colo.
- n. Antisedan, Orion Pharma, Zoetis, Kalamazoo, Mich.
- o. Zoological Information Management System (ZIMS) [database online]. Minneapolis: Species360, 2019. Available at: zims.Species360.org. Accessed Jul 16, 2019.
- p. VetScan VS 2 Chemistry Analyzer, Abaxis, Union City, Calif.
- q. VetStix 11 SG Animal Health Urine Test Strips, VetOne, MWI Animal Health, Boise, Idaho.
- r. Lighthouse Veterinary Consultants, Alachua, Fla.
- s. Frontline Spray for Dogs and Cats, Merial, Duluth, Ga.
- t. Penject, Butler Schein Animal Health, Dublin, Ohio.
- u. Dexaject SP, Henry Schein Animal Health, Dublin, Ohio.
- v. Sun Pharmaceutical Industries, Mumbai, India.
- w. PrednisTab, Lloyd Inc, Shenandoah, Iowa.
- x. Imrab 3 rabies vaccine killed virus, Merial, Duluth, Ga.
- y. ZooPharm Formulation, Windsor, Colo.
- z. Joseph Colbert, Wildlife Manager, Jekyll Island Authority Conservation Department, Jekyll Island, Ga: Personal communication, 2018.

References

1. Bowman DD, Lynn RC, Eberhard ML, et al. *Georgis' parasitology for veterinarians*. 8th ed. St Louis: Saunders, 2003;1–114.
2. Nagamori Y, Reichard MV. Parasitology expertise from the NCVP: feline tick-borne diseases. *Today's Vet Pract* 2015;5:69–74.
3. Chomel BB, Kasten RW. Bartonellosis, an increasingly recognized zoonosis. *J Appl Microbiol* 2010;109:743–750.
4. Shaw SE. Haemobartonella and bartonella: two very different diseases!, in *Proceedings*. World Small Anim Vet Assoc World Cong 2003;118.
5. Atwell R. Merck veterinary manual. Overview of tick paralysis. Available at: www.merckvetmanual.com/nervous-system/tick-paralysis/overview-of-tick-paralysis. Accessed Dec 10, 2018.
6. Luttrell MP, Creekmore LH, Mertins JW. Avian tick paralysis caused by *Ixodes brunneus* in the southeastern United States. *J Wildl Dis* 1996;32:133–136.
7. Konjević D, Janicki Z, Severin K, et al. An outbreak of tick paralysis in free-ranging mouflon (*Ovis ammon musimon*). *J Zoo Wildl Med* 2007;38:585–587.
8. Beyer AB, Grossman M. Tick paralysis in a red wolf. *J Wildl Dis* 1997;33:900–902.
9. Schull DN, Litster AL, Atwell RB. Tick toxicity in cats caused by *Ixodes* species in Australia: a review of published literature. *J Feline Med Surg* 2007;9:487–493.
10. Botzler RG, Albrecht J, Schaefer T. Tick paralysis in a western harvest mouse. *J Wildl Dis* 1980;16:223–224.
11. Jessup DA. Tick paralysis in a grey fox. *J Wildl Dis* 1979;15:271–272.