



Apparent *Ixodes* tick paralysis in a cat from North America

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Abstract

Case summary A 2-year-old castrated male domestic longhair cat presented for acute, diffuse, flaccid paralysis. Thoracic and abdominal radiographs, biochemistry panel and complete blood count were unremarkable. Titers to *Toxoplasma gondii*, myasthenia gravis radioimmunoassay testing and creatinine kinase levels were within normal limits. The most likely differentials included acute toxicity (coral snake envenomation, organophosphate toxicity), botulism and, less likely, acute polyradiculoneuritis. A thorough physical examination revealed a single engorged tick attached to the ventral neck of the cat, which was later identified as an adult female *Ixodes* species. Topical fipronil and (S)-methoprene was administered. Over the next 48 h, the cat recovered full motor function and at 5 days post-tick removal the cat had resumed all normal activities.

Relevance and novel information Tick paralysis is considered endemic in Australia by bites from, most commonly, the *Ixodes holocyclus* tick. However, this phenomenon is rarely reported in the USA. This is the first report of a domestic cat suffering from acute tick paralysis in North America.

Keywords: Tick paralysis; *Ixodes*; North America; flaccid paralysis

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Introduction

Although relatively rare in North America, tick paralysis is one of the main differentials for acute, diffuse, flaccid tetraparalysis of dogs and cats.¹ The clinical course typically begins as an ascending paresis that progresses cranially from the pelvic limbs.¹ Typically, the patient has complete flaccid tetraparesis or tetraplegia within 24–72 h, with occasional head and neck involvement.¹ Interestingly, tick paralysis typically does not result in cranial nerve deficits, whereas some other conditions that present similarly, such as botulism, can have cranial nerve deficits.¹ Often, respiratory paresis or paralysis may occur as a result of intercostal or phrenic nerve involvement.¹ When a female tick bites a host and remains attached for a prolonged period, paralysis due to salivary neurotoxins takes effect in approximately 3 days.² Treatments entail removing the offending tick,

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performing a thorough examination for additional ticks, possibly treating with a topical acaricide and/or treating with tick antiserum (TAS).² In Australia, tick paralysis in cats is commonly reported, especially with bites from *Ixodes holocyclus*, yet, in the USA, *Dermacentor variabilis* and *Dermacentor andersoni* are typically implicated in tick paralysis of many different animal species.²⁻⁴

To our knowledge, this is the first reported case of tick paralysis in a domestic cat in North America, although the condition is well reported in dogs, humans and other animal species.

Case description

A 2-year-old castrated male, domestic longhair cat weighing 5.66kg presented to the Texas A&M Small Animal Teaching Hospital for acute paralysis overnight and lying in an atypical location. The day prior, it had gone outside during the day and climbed trees as per usual. That evening, after going back inside, the cat continued to jump onto furniture. At this time, the only abnormality of note was slight hyporexia. The cat lives with three other cats, all of which remained healthy.

Upon physical examination, abnormalities included intermittent increased respiratory effort with normal bronchovesicular sounds; non-ambulatory flaccid paralysis; depressed but alert mentation; intact cranial nerves and bilateral pupillary light reflexes (both direct and indirect); and tapeworm segments were noted on the perineum. On intake, thoracic and abdominal radiographs, a complete blood count, blood chemistry, a urinalysis, ammonia levels and a feline immunodeficiency virus/feline leukemia virus immunodiagnostic test were performed. All were unremarkable. A consultation with the Neurology Service confirmed diffuse lower motor neuron disease. Differentials included a coral snake envenomation, organophosphate toxicity, botulism, acute polyradiculoneuritis and acute fulminant myasthenia gravis. Creatine kinase, myasthenia gravis titers and supportive care were recommended.

Upon re-examining the cat thoroughly for any wounds, an engorged tick was found attached to its ventral neck (Figure 1). The tick was removed, and the cat was treated with topical fipronil and (S)-methoprene (Frontline Plus; Merial) and fenbendazole (Panacur; Merck). The tick was identified as an engorged adult female *Ixodes* species by experienced parasitologists using pictorial keys (Figure 2). Molecular genetic species-level confirmation by PCR and sequencing was not performed.

In 24h, the patient was able to support its head on its own and make small movements with its paws and tail. Creatine kinase levels were mildly elevated (1405U/l, reference interval 107–1300U/l). Ancillary testing of IgG and IgM serum titers to *Toxoplasma gondii* were negative (<64) and myasthenia gravis radioimmunoassay testing was



Figure 1 An engorged adult female *Ixodes* species tick found on the cat's neck



Figure 2 A stereomicroscope photograph of the tick specimen, that was then identified as an engorged, adult, female *Ixodes* species

negative (0.06nmol/l, negative is <0.3nmol/l). In 48 h, the patient was able to sit upright, stand with support and move around its cage a couple steps at a time. Prior to discharge, the patient was treated with topical emodepside/praziquantel (Profender; Bayer HealthCare). Three days post-tick removal, the patient resumed all its previous activities, including jumping on furniture and walking around the house.

Additionally, to assist in identifying possible pathogens that could be transmitted, the tick was submitted to the Texas A&M Veterinary Medical Diagnostic Laboratory for molecular testing (TickPath Layerplex panel)⁵ for multiple pathogens, including *Cytauxzoon felis*, *Bartonella* species, *Anaplasma* species, *Ehrlichia* species, *Mycoplasma haemofelis*, *Candidatus Mycoplasma haemominutum* and

Candidatus Mycoplasma turicensis. Of these, the only pathogen detected was '*Candidatus M haemominutum*', which may cause anemia. No anemia was present and this was considered an incidental finding.

Discussion

Tick paralysis is a relatively rare disease in North America and is typically found in dogs, livestock and humans.^{6,7} The most common tick species that cause tick paralysis in North America are *D andersoni* and *D variabilis*.^{4,6-9} *D andersoni* is distributed in the Rocky Mountain states and Pacific northwest of the USA, whereas *D variabilis* is found throughout the eastern half of the USA and along the West Coast.⁷ In addition, tick paralysis by *Ixodes brunneus* has been reported in birds in southeastern USA.¹⁰ The black-legged tick, *Ixodes scapularis*, however, is the most commonly reported species of this genus in cats and dogs across eastern and southern USA.¹¹ Disease occurs by a salivary neurotoxin that is released into the host from the engorged female tick from the *Ixodes* genus.² In Australia, the *I holocyclus* tick causes a unique, more severe version of tick paralysis.^{2,4} The mechanism of the holocyclotoxin found from *I holocyclus* ticks is not completely elucidated, but it seems to block calcium influx at the nerve ending thereby preventing acetylcholine release to the neuromuscular junction.^{2,4} It is possible that this is similar to how other *Ixodes* species' neurotoxins function.

Treatment for tick paralysis entails removal of the tick and supportive care (fluids and respiratory support, as needed).² To our knowledge, the TAS that is available in Australia is specifically targeted for treating tick paralysis induced by *I holocyclus*. The best way to prevent tick paralysis from occurring is to perform daily examinations for ticks and ensure patients are on an appropriate tick preventive. Currently in the USA, options for feline tick preventive include fluralaner (Bravecto; Merck), topical fipronil and (S)-methoprene (Frontline Plus; Merial), selamectin/sarolaner (Revolution Plus; Zoetis), or a flumethrin and imdacloprid collar (Seresto collar; Bayer).

Other notable differentials for diffuse lower motor neuron flaccid paralysis include acute fulminant myasthenia gravis, toxoplasmosis, acute polyradiculoneuritis, botulism and coral snake envenomation.^{12,13} Fortunately, the history, clinical presentation and differential diagnostics can help to discern between all of these. The importance of a good history cannot be overemphasized. Onset, duration and progression of clinical signs aids greatly in shortening the list of differential diagnoses. Myasthenia gravis and toxoplasmosis were excluded as differentials based on the titers performed. Acute polyradiculoneuritis was considered highly unlikely as recovery from this disease typically requires 4–6 weeks of supportive care.^{14,15} Botulism was considered unlikely as dogs and cats are relatively resistant and tend to recover in a few weeks after the source of the toxin is removed.^{13,14} Lastly, coral snake envenomation was excluded as typically puncture

wounds are noted on physical examination and clinical findings include altered mental status with rapid progression to respiratory failure.¹⁶

Conclusions

Considering the possible differentials for acute, flaccid tetraparalysis in conjunction with the rapid clinical improvement seen after tick removal, the likely explanation for this cat's clinical signs is tick paralysis.

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Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval This work involved the use of non-experimental animals only (including owned or unowned animals and data from prospective or retrospective studies). Established internationally recognized high standards ('best practice') of individual veterinary clinical patient care were followed. Ethical approval from a committee was therefore not necessarily required.

Informed consent Informed consent (either verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (either experimental or non-experimental animals) for the procedure(s) undertaken (either prospective or retrospective studies). No animals or humans are identifiable within this publication, and therefore additional informed consent for publication was not required.

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