

Brief Report

A SIX-YEAR-OLD GIRL WITH TICK PARALYSIS

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TICK paralysis is a neurologic syndrome that is frequently confused with other acute disorders. In this syndrome, ascending paralysis is caused by a potent neurotoxin produced by an attached, engorged tick. Removal of the tick leads to prompt recovery. Although cases of tick paralysis were clearly described almost 90 years ago in the United States,¹ Canada,² and Australia,³ the syndrome is unfamiliar to many clinicians today. Since a delay in the diagnosis can have devastating consequences, physicians must be aware of the salient features of this syndrome. If the diagnosis of tick paralysis is being considered, detection is possible with nothing more than a fine-toothed comb.

We report severe tick paralysis in a child cared for at an academic medical center. Once the correct diagnosis had been established, intervention with the use of another common item — tweezers — resulted in rapid recovery.

CASE REPORT

A previously healthy, six-year-old girl reported a tingling sensation in her fingers and six hours later began to stagger and fall. The next day, she was unable to walk without assistance. She was brought to the Medical College of Georgia 30 hours after the onset of symptoms. On initial examination, the child, who had long hair, was alert and afebrile, but had truncal instability and a wide-based, ataxic gait. She was unable to walk without support. Muscle strength was 4/5 in the legs and arms, proximally and distally. Dysmetria of the arms was noted on finger-to-nose testing. Muscle-stretch reflexes were diminished at the left knee but were normal elsewhere. The findings on tests of sensory-nerve and cranial-nerve function were normal, as was the rectal-sphincter tone. Chest radiographs and routine laboratory studies showed no abnormalities. Tests for toxic substances and a stool culture were negative. The initial differential diagnosis included acute cerebellar ataxia, cervical-cord compression, and the Guillain-Barré syndrome.

Magnetic resonance imaging studies of the head and neck showed no abnormalities. Analysis of a cerebrospinal fluid specimen revealed a protein level of 29 mg per deciliter, a glucose concentration of 71 mg per deciliter (3.9 mmol per liter), and a lymphocyte

TABLE 1. RESULTS OF NERVE-CONDUCTION STUDIES.

NERVE	MUSCLE ACTION POTENTIAL	CONDUCTION VELOCITY
	mV	m/sec
Ulnar		
During paralysis	9.6	Not assessed
After recovery	14.4	63
Peroneal		
During paralysis	2.5	45
After recovery	5.8	53

count of 2 per cubic millimeter. Gram's staining and a culture of cerebrospinal fluid were negative. Nerve-conduction studies performed 24 hours before the maximal weakness revealed that the distal latency of the ulnar nerve was prolonged, at 6.0 msec (normal value, <2.5) and that the amplitude of the muscle action potential of the peroneal nerve was moderately reduced (Table 1). There was no evidence of conduction block or late components.

Forty-eight hours after the onset of symptoms, the child became lethargic and irritable. Symmetric leg weakness increased, with muscle strength of 3/5. Fine motor movements of the fingers were poor, and muscle-stretch reflexes at the knees and ankles were absent. The child could barely sit, even with support. Forced vital capacity was 20 ml per kilogram (normal value for age, >40). The end-tidal partial pressure of carbon dioxide was 53 mm Hg. Oxygen saturation was maintained at 98 percent with nasal administration of oxygen. Because of the ascending paralysis and hypoventilation, the child was transferred to the pediatric intensive care unit for monitoring and possible intubation. The clinical impression was that the child had Guillain-Barré syndrome.

By 72 hours after the onset of illness, motor strength in the arms and legs had decreased to 2/5. Listlessness, slurred speech, and bilateral ptosis were observed. Because of the rapid deterioration in association with a presumed diagnosis of Guillain-Barré syndrome, preparation for femoral-vein access was initiated for emergency plasmapheresis.

During placement of the central catheter, an astute pediatric resident, aware that the Guillain-Barré syndrome can be confused with tick paralysis, carefully inspected the child's hair with a fine-toothed comb. To the surprise of the three pediatricians, the pediatric neurologist, and the pediatric intensivist who were caring for the child, an engorged tick, 15 mm in diameter, was embedded in the left parietal area of the scalp (Fig. 1). It was identified as a female *Dermacentor variabilis*.⁴ The tick was promptly removed with tweezers, 78 hours after the onset of symptoms and 48 hours after admission to the hospital.

Within six hours after removal of the tick, the child's slurred speech, ptosis, and lethargy had resolved. Within eight hours, her strength had improved to 4/5 in the arms, and she could elevate her arms above her head. In addition, the forced vital capacity increased to 50 ml per kilogram, and the oxygen saturation increased to 99 percent while the child was breathing room air. Within 12 hours, she could sit up with minimal assistance and elevate her legs against moderate resistance. Areflexia persisted until 17 hours after removal of the tick, when muscle-stretch reflexes at the ankles and knees returned to normal. Within 24 hours, ataxia of the trunk, arms, and legs had fully resolved, and the child could walk without assistance (Fig. 2). The results of a neurologic examination were completely normal, and she was discharged 32 hours after removal of the tick. Data from follow-up nerve-conduction studies, performed after her strength had returned to normal, revealed marked improvement in the amplitude of the muscle action potentials and in the conduction velocity of the ulnar and peroneal

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Figure 1. Engorged Female *Dermacentor variabilis* in the Scalp of a Six-Year-Old Girl with Ascending Paralysis, Ataxia, Areflexia, Bulbar Signs, and Hypoventilation.

nerves (Table 1). The distal latency of the ulnar nerve also improved dramatically, with a decrease from 6.0 to 2.6 msec.

DISCUSSION

Tick paralysis typically affects children, who present with paresthesias and leg weakness but without fever; they have difficulty walking and tend to fall. Adults are affected less frequently,⁵⁻⁷ presumably because the neurotoxin produced by the tick has a less pronounced effect because of their larger body mass. Within 24 to 48 hours, weakness in the legs ascends to the trunk musculature, and the patient can no longer sit or walk without assistance. Ascending paralysis, accompanied by hyporeflexia or areflexia, progresses to the arms, then to the bulbar structures involved in speech, swallowing, and facial expression, and eventually to the respiratory musculature. If the tick is not found and removed, respiratory weakness can lead to progressive hypoventilation, lethargy, coma, and death.

The clinical differentiation of tick paralysis from the Guillain-Barré syndrome and other causes of ascending paralysis is crucial because the therapies for these conditions differ. Features of four common syndromes of generalized paralysis in previously healthy patients are summarized in Table 2. The diagnostic features of the Guillain-Barré syndrome are a progressive paralysis in both legs, both arms, or all four limbs; areflexia or hyporeflexia; a cerebrospinal fluid protein level that exceeds 40 mg per deciliter (although the protein level may be normal initially), with a mononuclear-cell count of less than 10 per cubic millimeter; and a progression to peak neurologic impairment within four weeks after the onset of symptoms.⁸ In contrast, tick paralysis is characterized by



Figure 2. The Fully Recovered Patient, 24 Hours after Removal of the Tick.

normal cerebrospinal fluid and by a progression to peak paralysis over a period of hours to a few days. Nerve-conduction studies in patients with tick paralysis may resemble those in patients with the Guillain-Barré syndrome: findings in both conditions include prolonged latency of the distal motor nerves, diminished nerve conduction velocity, and reduction in the amplitudes of muscle and sensory-nerve action potentials, as observed in our patient. Acute lesions of the spinal cord are distinguished by sensory changes, urinary retention, fecal incontinence, and laxity of anal-sphincter tone. Poliomyelitis, with which tick paralysis was often confused 40 years ago, is now rare in North America and is usually associated with use of the trivalent oral vaccine or travel to areas where poliomyelitis is endemic. Patients with poliomyelitis usually present with fever, meningeal signs, asymmetric weakness, and a predominance of lymphocytes in the cerebrospinal fluid, all of which are absent in patients with tick paralysis. Botulism, with which tick paralysis can also be confused, is characterized by a slow, descending paralysis that involves the cranial nerves first, usually with extraocular palsy and large, poorly reactive pupils, unlike the rapid, ascending pattern of tick paralysis.⁹

TABLE 2. FEATURES OF FOUR SIMILAR SYNDROMES OF ASCENDING PARALYSIS.

FEATURE	TICK PARALYSIS	GUILLAIN-BARRÉ SYNDROME	SPINAL CORD LESION	POLIOMYELITIS
Ataxia	Present	Absent	Absent	Absent
Rate of progression	Hours to days	Days to weeks	Gradual or abrupt	Days to weeks
Muscle-stretch reflexes	Absent	Absent	Variable	Absent
Babinski sign	Absent	Absent	Present	Absent
Sensory loss	None	Mild	Present	None
Meningeal signs	Absent	Rare	Absent	Present
Fever	Absent	Rare	Absent	Present
Cerebrospinal fluid findings				
Protein level	Normal	High	Normal or high	High
White-cell count (per mm ³)	<10	<10	Variable	>10
Time to recovery	<24 hr after tick removal	Weeks to months	Variable, depending on cause	Months to years or no recovery (permanent paresis)

In studies in animals, the neurotoxin in tick paralysis reduces nerve conduction velocity and the amplitude of muscle action potentials,^{10,11} inhibits terminal-nerve conduction and acetylcholine release at the presynaptic neuromuscular junctions of muscle fibers,^{12,13} and causes total blockade of transmission at myoneural junctions.¹⁴ In the few published reports on paralysis in humans that was caused by *D. variabilis* or *D. andersoni*, the electrophysiologic abnormalities included reductions in motor- and sensory-nerve conduction velocities and the amplitude of muscle action potentials, without defects in neuromuscular transmission¹⁵⁻¹⁷; prolongation of the distal latency of the peroneal nerve, with unresponsiveness to repetitive stimulation¹⁸; and diminution of the conduction velocities of the median, ulnar, and peroneal nerves and marked reductions in the amplitude of muscle action potentials, with the restoration of normal neuromuscular transmission and rapid reversal of the abnormalities when the tick was removed.¹⁹ The last set of findings is consistent with the results of the nerve-conduction studies performed in our patient. The predominant abnormality is a reduction in muscle action potentials, perhaps caused by interruption of the sodium flux across axonal membranes at the nodes of Ranvier and nerve terminals, with little, if any, impairment of neuromuscular transmission.

Neurotoxin produced by the *Ixodes holocyclus* tick, found in Australia, interferes with acetylcholine release at the neuromuscular junction, as does botulinum toxin.^{9,11,20} Extracts of homogenized *I. holocyclus* ticks produce paralysis when injected in dogs.²¹ The neurologic impairment caused by *I. holocyclus* ticks in Australia is more severe than that caused by dermacentor species in North America. With exposure to *I. holocyclus* neurotoxin, the weakness and bul-

bar symptoms often intensify during the first 24 to 48 hours after the tick has been removed, and clinical recovery is much slower than with dermacentor-related paralysis. For these reasons, *I. holocyclus* antitoxin must be administered before the tick is removed, and longer observation is required.²⁰

The onset of tick paralysis occurs five to seven days after a female tick attaches itself to the skin. Engorgement of the feeding tick is relatively limited until it mates with a male. Mating leads to rapid engorgement, the fertilization of eggs, and the production of neurotoxin. The term "ixovotoxin" has been applied to the substance responsible for the development of tick paralysis.²² The beginning of neurotoxin production coincides with the initial paralytic symptoms in affected patients. Continued feeding by the tick apparently accelerates toxin production, accounting for the rapid clinical deterioration. Once engorgement is complete, the female tick disengages from its host and drops off to deposit the eggs. The *D. variabilis* tick removed from our patient deposited approximately 200 eggs 17 days after removal, an observation that supports prior claims that the fertile dermacentor female causes tick paralysis in the United States.^{23,24}

Tick paralysis is easily confused with a variety of other conditions (Table 3).²⁵ Ours is not the first instance in which careful examination with a fine-toothed comb led to the correct diagnosis; in other cases, patients were initially thought to have cerebellar ataxia,²⁶ poliomyelitis,^{27,28} or "idiopathic paralysis."⁶ In each of these cases, the engorged tick was detected in scalp hair by a nurse using a comb, after extensive clinical evaluation by one or more physicians and hours to days of observation and laboratory testing.

In several reported cases involving patients who died of unexplained paralytic illnesses, an engorged

TABLE 3. DIFFERENTIAL DIAGNOSIS OF TICK PARALYSIS.*

Guillain-Barré syndrome
Acute spinal cord lesion
Cerebellar ataxia
Poliomyelitis
Botulism
Myasthenia gravis
Electrolyte disorder
Periodic paralysis
Diphtheria
Heavy-metal intoxication
Insecticide poisoning
Porphyria
Solvent inhalation (glue sniffing)
Hysterical paralysis

*The list of diagnoses is from Jones.²⁵

tick was found on the head or neck at autopsy or by an undertaker.²⁹⁻³³ In Rose's review³² of 332 cases of tick paralysis, the mortality rate was 11.7 percent. Death was attributed to the lack of examination by a physician, the delay in an initial examination until the terminal stage of illness, or the failure to look for or detect the engorged tick. It is astonishing that a 1-g tick, so easily overlooked, can generate a toxin of such potency.

One case of tick paralysis involved a two-year-old child in New York who had weakness and ataxia.³⁴ The child underwent extensive evaluation and, after observation for two days, was transferred to a tertiary medical facility, where she was found to have an engorged "wood tick" behind the right ear, at the hairline. Within 24 hours after removal of the tick the child had fully recovered. Other cases of tick paralysis include that of a two-year-old girl in Washington who had an unsteady gait and areflexia and was initially given a diagnosis of Guillain-Barré syndrome³⁵ and that of an 18-month-old girl in Virginia who had ascending flaccid paralysis with areflexia.³⁶ Rural residents of southern Georgia were once noted to be "so familiar with this syndrome that whenever their children or domestic animals display these symptoms, they search at once for ticks on their bodies."³⁷

One of us has treated five cases of tick paralysis during 25 years of practice. All five of these cases were in young girls with long hair and were initially thought to be the Guillain-Barré syndrome. In two of the cases, an engorged tick was detected on the scalp by a parent or a technician during preparation for electroencephalography. In all the patients, removal of the tick was followed by clinical recovery within 8 to 24 hours. We speculate that, as in these instances, certain patients who are presumed to have the Guillain-Barré syndrome actually have undiag-

nosed tick paralysis and that their clinical condition may improve after the spontaneous detachment of an undetected, fully engorged female dermacentor tick.

Whenever physicians evaluate children or adults with acute ataxia or ascending paralysis, they must search carefully for engorged female ticks, primarily on the scalp but also in the axilla and perineum. A clinical diagnosis of Guillain-Barré syndrome should not be accepted until a careful search has excluded the possibility that a tick is present. One may literally have to comb for the evidence. For patients with tick paralysis, timely examination with a fine-toothed comb to detect the tick, with the use of tweezers for its removal, can result in rapid recovery and may well be one of the simplest, most effective medical interventions available.

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