A 4-year-old Boy with Ascending Weakness and a New ‘Skin Tag’

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A 4-year-old boy from Singapore, while on vacation in the United States (first to Georgia and then Washington, D.C.), was evaluated for a subacute progressive ascending weakness. Five days before, he had noted the onset of vague paresthesias and muscular pains beginning in the distal left leg and later in the distal right leg. There was a gradual progression of difficulties with walking during the next 5 days.

Upon initial presentation, the patient was complaining of mostly lower extremity but also some upper extremity weakness and was unable to stand on his own. There were no problems with breathing, talking, or swallowing, and no complaints of back pain or difficulties with bowel or bladder function.

The patient was seen by a physician at an acute-care facility. The examination revealed lower extremity weakness with less involvement of the arms. Reportedly, the patient was so weak he was unable to get out of bed, stand, or walk. No cranial nerve or sensory abnormalities were noted, but the patient was noted to be hyporeflexic, lower extremities more prominently than the upper extremities. In addition, the examining physician found on the back of the patient’s neck what the mother described as a “new skin tag.” This had been noticed 1 week before onset of the patient’s symptoms.

Upon closer examination, however, this skin tag was recognized instead to be an attached arachnid (see Figure, page 240). It was carefully removed and placed in a container, and the patient was admitted for further evaluation and observation. Within 5 hours, the patient was nearly back to his baseline.

The skin tag was identified as an engorged, female tick of the species *Dermacentor variabilis*, possibly contracted while spending time in wooded areas in Georgia.
DISCUSSION

Tick paralysis is a toxin-mediated disorder, seen most often in children, caused by injection of a neurotoxin from a feeding, engorged, and usually gravid female of certain species of ticks. Tick paralysis is most commonly reported in North America and Australia. There are about 50 species of ticks known to cause tick paralysis. In North America, tick paralysis is caused by several species, but is most commonly caused by *D. andersoni*, the Rocky Mountain wood tick, especially in the Pacific Northwest. *D. variabilis*, the dog tick, is a common cause on the eastern shore of the United States. Although *Ixodes scapularis*, the most common vector for Lyme disease in the eastern United States, has been reported to cause tick paralysis, it is much less commonly implicated. In Australia, *I. holocatus*, the scrub tick, is the most common culprit.

Clinically, tick paralysis is characterized by a subacute onset of an ascending flaccid paralysis. Non-specific symptoms, such as irritability or fatigue, are sometimes observed in the days before onset of neurologic symptoms. Distal extremity paresthesias may be felt before the onset of weakness, which progresses over hours to days from the lower to the upper extremities, often initially manifesting in children as difficulties with gait, specifically gait ataxia.

In severe cases, cranial nerve involvement may occur with ocular, facial, or bulbar involvement. There appear to be some differences in the clinical presentation depending on the species (which is dependent on the continent and geographical location), as *I. holocatus* tends to cause a higher incidence and an earlier onset of cranial nerve dysfunction.\(^1\)

The prevalence of tick paralysis is uncertain, but it appears to be rare in humans. It is most commonly encountered during the spring and summer, corresponding to the peak seasons for the prevalence of adult ticks. Tick paralysis occurs most often in children, with 80% occurring in children younger than 8 years.\(^2\) This is likely related to the smaller body size that is more susceptible to the neurotoxin. In addition, 75% of cases in children occur in girls,\(^2\) which may be related to the fact that the offending tick may be better hidden by long hair more often encountered with girls. Although the most common sites for attachment are the scalp and behind the ear, attachment may be anywhere on the body.

The exact chemical structure of the neurotoxin has not been isolated. However, based on neurophysiological studies, it is thought most likely to act at the distal nerve ending,\(^3\) although some evidence suggests possible effects at the neuromuscular junction.\(^4\) Toxin production is likely related to feeding, as it takes approximately 5 days of feeding before the clinical effects of the neurotoxin are seen.\(^1\)

The diagnosis of tick paralysis is made with a thorough skin examination, performed based on recognition of the clinical presentation: an ascending flaccid paralysis. Other disease processes can present similarly, particularly Guillain-Barré syndrome (GBS), an immune-mediated radiculoneuropathy. A lumbar puncture revealing an albuminocytologic dissociation is supportive of GBS, and treatment usually consists of intravenous immunoglobulins (IVIg) or plasmapharesis. Because of this similar presentation, several patients with tick paralysis have been reported to have been initially misdiagnosed and treated with IVIg.\(^3\) On the contrary, a descending paralysis with early cranial nerve involvement is characteristic of botulism.

Finally, patients with acute spinal cord dysfunction may present with acute or subacute paralysis. A sensory level abnormality, early bowel and bladder involvement, or the presence of upper motor neuron signs should alert the clinician to a possible spinal cord process.

Once identified, the treatment of tick paralysis involves removal of the tick. The recommended method is to grasp the tick as closely as possible to the point of attachment.
with tweezers or forceps, using slow steady pressure to remove the entire tick, including the mouthparts.  

The expected clinical course after tick removal is fairly rapid, with continued improvement in neurological function during the next few hours. This is especially the case in North America. However, in Australia, it is not unusual for there to be a continued worsening of symptoms for a few days before clinical improvement, even with complete removal of the offending tick.  

For the rare patient with phrenic or bulbar involvement, supportive respiratory ventilation may be required. Although an anti-serum prepared from dogs is available, its efficacy and safety are uncertain in humans, and its use should be reserved for severely ill patients. Despite previously reported mortality rates of more than 10%, in the modern era of intensive care and increased recognition, the expected prognosis is for a full recovery.  

**CONCLUSION**

A thorough skin examination should be performed on any child presenting with gait ataxia or an ascending paralysis resembling GBS. Tick paralysis is easily treated with removal of the offending tick, and its early diagnosis and treatment could prevent unnecessary testing and treatments.

**REFERENCES**