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Tick Paralysis: A Treatable Disease Not to Be Missed

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Abstract

Tick paralysis is a rare condition which can be treated easily but if undiagnosed can lead to significant morbidity and mortality. It is prevalent in certain areas especially in North America and Australia. It is characterized by acute onset symmetric lower extremity weakness and if not diagnosed early the weakness involves the upper extremities, cranial nerves, respiratory musculature leading to respiratory failure requiring mechanical ventilation. The clinical picture is often confused with other causes of acute onset paralysis eg., Guillain-Barre syndrome, the Miller Fisher variant, leading to unnecessary investigations and interventions complicating the clinical picture. The condition can be easily diagnosed by careful history and physical examination. Tick removal will lead to early and complete recovery of the condition.

It is very important for the clinicians to be aware of this condition so that it can be recognized early to avoid unnecessary and expensive investigations, interventions, morbidity and mortality.

Keywords: Tick paralysis; Morbdity; Mortality; Neurologic syndrome; Neurotoxin

Introduction

Tick paralysis is a rare neurologic syndrome characterized by ascending paralysis due to a potent neurotoxin produced by an attached engorged gravid female tick. Although tick paralysis is a well described phenomenon, it may still be unfamiliar to clinicians resulting in delayed diagnosis, so a differential diagnosis of tick paralysis should always be kept in mind while encountering a case of ascending paralysis. It can be fatal if not recognized early because fatal respiratory paralysis develops. Children are particularly vulnerable due to their small size and the fact that less neurotoxin is needed to produce the paralysis.

We report a case of 5-year-old girl who presented with ascending paralysis with ophthalmoplegia, and who completely recovered within 12 hours after removal of a tick from her scalp.

Clinical Presentation

A previously healthy 5-year-old girl, was seen in the emergency room for progressive gait difficulty noticed by her parents for over 5 days. She had a low-grade fever, was clumsy, and had not had a bowel movement for 4 days prior to her presentation. There was no history of seizures but she had decreased oral intake and speech over 3 days. Clinical presentation was complicated by a tooth removed the day prior to presentation and an abscess treated with amoxicillin.

On examination, she was afebrile and awake, with limited speech. There were no meningeal signs. Her pupils were symmetrical and reactive to light. Her fundus exam was normal. She had right eye ptosis. She complained of diplopia on lateral gaze and had ophthalmoplegia with limitation of lateral eye movements with nystagmus. Her facial movements and eye closure were symmetrical. She had fair strength in her arms against moderate resistance with decreased reflexes. She had weakness in both lower extremities with limited range of movements against gravity. Her deep tendon reflexes were absent in both lower extremities. Her cerebellar exam showed mild dysmetria which could be related to her visual impairment from the diplopia. Her gait could not be assessed due to her leg weakness.

Investigations included normal blood work, negative urine and serum toxicology screening. CSF glucose and protein levels were normal and no cells were found. Brain and spine MRI with and without contrast were normal. The child was admitted to the Pediatric Intensive Care unit for close monitoring due to concerns about a possible atypical variant of Guillain Barre syndrome (Miller Fisher syndrome) with plans for further investigations including electrodiagnostic studies.

Additional history about tick exposure in a family member led to detailed physical examination and revealed an engorged tick in her scalp (Figure 1) that was carefully removed applying sustained pressure with a tweezers.

The girl started talking within an hour of the tick removal and her motor strength improved overnight, and she could bear weight and walk within 12 hours.

Discussion

Tick paralysis is a disease that occurs worldwide. It was described more than 100 years ago in North America [1]. It typically affects children who presents with ascending weakness of the legs, progressing to trunk musculature, arms, bulbar structures which involves speech, swallowing and facial expression and finally involves the respiratory musculature. If the tick is not found and removed it leads to respiratory weakness leading to progressive hypoventilation, lethargy, coma and death [2]. Three tick species are responsible for the paralysis in humans: *Dermacentor andersoni Stiles* (the North American wood

tick), *Dermacentor variabilis* (the common dog tick), *Ixodes holocyclus* (the Australian marsupial tick) [3].

Figure 1: Engorged *Dermacentor variabilis* removed from the 5-year-old girl presenting with ascending paralysis and bulbar signs.

Dermacentor andersoni is the etiologic agent of four distinct pathologies in men, rocky mountain spotted fever, tularemia, Colorado tick fever and tick paralysis.

In the USA, the epidemiology of tick paralysis has a predictable pattern, commonly occurring in spring and summer in the North Western and South-Western region when the tick arouses from hibernation to mate and to feed blood [2,4]. It affects more commonly in children compared to adults [2,4]. Female children are more likely to be affected because of their long hair where ticks find a safe place to mate and feed [2,4]. Felz and Durden demonstrated that 59% of *Dermacentor variabilis* attached to the human head and neck [4].

The pathophysiology of paralysis due to *Dermacentor* is currently unknown. The current hypothesis is that sodium flux across axonal membranes at the node of Ranvier is blocked, whereas neuromuscular transmission is not impaired [5,6]. In contrast, the toxin from *Ixodes holocyclus* appears to block the release of acetylcholine at the level of the neuromuscular junction, similarly to the mechanism of action of botulinum toxin [7].

The clinical manifestations of tick paralysis usually occur within 4 days to 5 days after a female tick attaches itself to the skin [5,8]. The tick rapidly engorges and the neurotoxin produced causes the neurological decline [5].

Clinical features are helpful in distinguishing tick paralysis from other similar disease processes. North American tick paralysis is usually characterized by two distinct clinical phases: (1) a nonspecific prodromal phase of fatigue and weakness; and (2) a subsequent neurotoxic phase of acute ataxia, progressing to symmetric ascending flaccid paralysis with loss of deep tendon reflexes, involvement of bulbar structures (dysphagia, dysarthria, facial weakness, or ocular weakness), respiratory muscle weakness, lethargy, coma and death. The Sensory system seems to be spared [4,8-10].

In tick paralysis routine laboratory studies and CSF analysis are normal [2,6,10]. The nerve conduction abnormalities include reduced amplitude of Compound Muscle Action Potentials (CMAPS), Normal Sensory Action Potentials (SNAPs) and normal response to repetitive nerve stimulation [6,10].

Tick paralysis is most commonly misdiagnosed as Guillain-Barre syndrome (GBS) particularly the Miller Fisher's subtype especially if the cranial nerves are involved [11,12]. In GBS the paralysis of the extremities is symmetric, ascending, progressive. and usually it peaks within several weeks of symptoms onset. In GBS CSF protein level is elevated, usually >40 mg/dl. Tick paralysis is, instead, associated with normal CSF proteins and the peak in neurological deterioration occurs within hours to days after onset of symptoms [3,12]. Tick paralysis and GBS have similar electrophysiological features [3].

The other differential diagnoses to consider are botulism, poliomyelitis, polio-like syndromes and electrolyte disturbances [8,11,12]. Botulism presents as slow descending paralysis with primary bulbar involvement and poorly reactive pupils. Poliomyelitis is listed here for its historical importance since the last case of wild poliovirus was documented in 1979. Poliomyelitis presented with meningeal signs, fever, asymmetric weakness and CSF lymphocytosis. Other differential diagnoses of tick paralysis include acute spinal cord lesion, cerebellar ataxia, myasthenia gravis, diphtheria, heavy metal intoxication, insecticide poisoning, accidental ingestion, porphyria and hysteria [8,10].

The mainstay of treatment of tick paralysis is tick removal and supportive care including mechanical ventilation for respiratory failure. After finding the tick, the tick should be grasped with tweezers as close to the head as possible and removed by gently pulling straight out [13,14]. Twisting and squeezing the body of the tick is not recommended as more toxin may be released [13,14]. Even pressure should be applied when removing ticks as mouth parts can break off and may result in granulomas [15]. Gloves or tissues should be used to protect the skin from the infectious agents that reside in the tick's saliva: hands should be thoroughly washed with soap and hot water. The bite area should be judiciously disinfected and the tick should be disposed properly in an alcohol solution.

Exposure to ticks and other arthropods can be reduced by avoiding tick infested areas, wearing protective clothing that covers exposed parts of the body, using insect repellant and performing routinely a careful examination of the child's head, neck and body. A history of tick exposure should lead to careful search of the scalp, neck, axilla, auditory meatus, umbilicus, vulva, and note to ensure that multiple ticks have not attached themselves.

Conclusion

Tick paralysis although rare should not be missed as it is an easily reversible condition. Children are particularly vulnerable due to their smaller body size and the fact that less toxin is needed to provoke the respiratory arrest. Limited awareness of this diagnosis among physicians may lead to misdiagnosis of other conditions and delay diagnosis with progression of symptoms, unnecessary and invasive investigations, treatments such as immunotherapy with immunoglobulins and sometimes even plasmapheresis. This case highlights the importance of good clinical history and thorough skin examination in all cases of paralysis and altered mental status in all children to successfully identify and remove the tick, even in those cases were fever, bulbar involvement and other misleading symptoms may be present.

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