

OVERVIEW ON TICK BORNE DISEASES AND PARALYSIS WITH REFERENCE TO EGYPT

By

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Abstract

Ticks are small arachnids of order Ixodida along with mites, they constitute subclass Acarina. Three families of ticks, 1- Nuttalliellidae comprises a single species, *Nuttalliella namaqua*, 2- Ixodidae, hard ticks and 3- Argasidae, soft ticks. Ixodidae are distinguished from the Argasidae by the presence of a scutum or hard shield. Ixodidae nymphs and adults both have a prominent *capitulum* (head) which projects forwards from the body; in the Argasidae, conversely, the *capitulum* is concealed beneath the body. Ticks transmit many infectious diseases to mammals including man, birds, and some reptiles and amphibians. Toxins of various ticks caused a disease known as tick paralysis, which can be confused with infectious and noninfectious conditions.

Key words: Ticks, Tick paralysis, Egypt, Overview

Introduction

Ticks play a major role in transmitting humans and animals' infectious diseases. Economic loss due to tick-borne infectious diseases among ruminants in tropical and subtropical could be several billion dollars per year (Perveen *et al*, 2021). In the United States, ticks can cause human disease, as 1- Anaplasmosis, 2- Babesiosis, 3- *Borrelia mayonii*, 4- Lyme disease, 5- *Borrelia miyamotoi*, 6- Bourbon virus, 7- Colorado tick fever, 8- Ehrlichiosis, 9- Heartland virus, 10- Powassan disease, 11- *Rickettsia parkeri* rickettsiosis, 12- Rocky Mountain spotted fever (RMSF), 13-STARI (Southern tick-associated rash illness), 14- Tick-borne relapsing fever (TBRF), 15- Tularemia and 16- 364D rickettsiosis (CDC, 2020).

Review and Discussion

In Egypt, the documented ticks transmitted diseases were 1-Anaplasmosis bovine (El-Ashker *et al*, 2016). 2- Babesiosis in rodents, man, dog, and equines (El Bahrawy *et al*, 1993; El-Bahnasawy and Morsy, 2008; Mahmoud *et al*, 2016), 3- Crimean-Congo hemorrhagic fever virus imported (El-Bahnasawy *et al*, 2012a), 4-Lyme disease tick and

man (Adham *et al*, 2010; Elhelw *et al*, 2014; Saleh *et al*, 2016), 5-Tickborne relapsing fever (El-Bahnasawy *et al*, 2012b), 6-Theileriosis in ruminants, cattle, sheep and equines (Mazyad and Khalaf, 2002; El-sify *et al*, 2015; Mahmoud *et al*, 2016), and 7-Ghoneim *et al*. (2017) in Egypt studied role of camels and attached ticks in *Francisella* species epidemiology. They did not detect *F. tularensis*, but high sero-positivity among abattoir workers especially those always exposed to tick bites underlines the possible of ticks attached to camels role in zoonotic tularemia. Abdullah *et al*. (2018) in reported that *Hyalomma dromedarii* and *H. excavatum* by molecular analyses both camels and ticks could be sources for Q fever to Egyptian animals and man. Also, Selim and Ali (2020) reported seroprevalence of *C. burnetii* (Q fever) among camels and that appropriate control measures must be taken to reduce infection transmission to other animals or human. Eldin *et al*. (2017) in France stated that zoonotic *Coxiella burnetii*, agent of Q fever, or query fever was described in Australia 1937. They added that Q fever can be found worldwide, but its epidemiological

features varied due to geographic area considered, in endemic or hyperendemic situations, and occurrence of large epidemic outbreaks. Recently, a major breakthrough in understanding of the natural history of human infection with *C. burnetii* was the breaking of the old dichotomy between acute and chronic Q fever. Borawski *et al.* (2020) in Europe reported Q fever is an emerging infectious disease, and ticks may act as a vector to animals and humans.

Also, Al-Hosary *et al.* (2021) in El-Faiyum Oasis, Assiut Governorate and El-Khariga Oasis examined ticks on bovine hosts for blood pathogenic agents. They found that *H. excavatum* ticks in a descending order of abundance were positive for *Anaplasma marginale*, *Theileria annulata*, *Midichloria mitochondrii*, *Babesia occultans*, *Ehrlichia chaffeensis*-like, *Ehrlichia minasensis*, and *Anaplasma platys*. In *R. annulatus* agents in a descending order were identified at different MIRs: *M. mitochondrii*, *T. annulata*, *A. marginale*, *A. platys*, *B. bovis* and *E. minasensis*. Pathogenic agents co-detected in tick pools were *A. marginale* and *T. annulata* (13.3%) samples followed by *A. marginale* and *M. mitochondrii* (8.4 %), and the triple agents were *A. marginale*, *T. annulata* and *M. mitochondrii* (5.3%) of tick pools.

Besides, 8- Dobler (2010) in Germany stated that tick-borne flaviviruses are among medically arboviruses in Europe and Asia. He concluded that changes in human behavior, land use, or climate may change the actual geographical distribution and transmission intensity so that tick-borne flaviviruses are potential winners of the changing environment and may increase in medical and veterinary importance.

Apart from tick borne infectious diseases, which are too much to mention, the toxins of various ticks' saliva can cause tick paralysis, which can be confused with both infectious and noninfectious conditions. Tick paralysis affects man and domestic and wild animals worldwide, but most cases occur in Australia and North America. Tick paralysis is im-

portant to recognize because it was fatal or nearly fatal particularly in dogs (Gerasimova *et al.*, 2018). Though, if diagnosed promptly, the disease was cured with the combination of proper tick removal and supportive care, yet in cases respiratory muscles paralysis without mechanical ventilation, it might lead to death (Borawski *et al.*, 2018).

Tick paralysis was first described by explorers in the 1824 Australian outback (Scott, 1921). Eighty-eight years later, it was recognized to occur in western Canada (Edlow and McGillicuddy, 2008). Tick paralysis has subsequently been found to affect humans and domestic and wild animals worldwide, although most cases occur in Australia and North America. Although it is a rare disease in humans, tick paralysis is important to recognize because it can be fatal or nearly fatal. However, if diagnosed promptly, this illness can be cured with the combination of tick removal and supportive care (Schaumburg and Herskovitz, 2000).

Epidemiology: There is no national surveillance system for tick paralysis and reliable information on incidence does not exist.

However, the disease appeared to be not common based upon available literature and clinical experience. As an example, only 33 cases were reported in Washington State in 50 years between 1946 & 1996, even though tick paralysis was a reportable disease in this state until 1998 (Dworkin *et al.*, 1999). Most cases in North America occur in the western regions of the United States and Canada, but the illness has also been seen in the eastern, southeastern, and south central United States. In addition, cases were reported in urban areas in travelers returning from endemic areas (Gordon and Giza, 2004).

Four cases of tick paralysis were reported in patients from north central Colorado. The clustering of these cases was unusual because in previous years, on average, only one case per year was reported from Colorado. Another unusual characteristic of this cluster was the age distribution of cases: only one of four cases occurred in a child. Previ-

ous case reports noted that tick paralysis was more likely to occur in children under the age of 10 years because tick toxins are more likely to cause symptoms in individuals with a smaller body mass (CDC, 2006).

Females are affected more often than males, possibly because ticks remained more likely undetected after attachment to persons with long hair. As with other tick-borne diseases, most cases occur in the spring and early summer months (CDC, 1996).

Tick vectors: There are more than 800 known species of argasid (soft-shelled) and ixodid (hard-shelled) ticks, and over 40 individual tick species are capable of producing salivary toxins that can cause paralysis in humans and animals (Gothe *et al*, 1979). The tick species that cause most cases of human tick paralysis in the United States and Canada were *Dermacentor andersonii* (Rocky Mountain wood tick), and *D. variabilis* (American dog tick). Others as *Amblyomma americanum* (Lone Star tick), *Ixodes scapularis* (Black-legged tick), and *I. pacificus* (Western black-legged tick) were associated with human tick paralysis (Inokuma *et al*, 2003). The primary cause of tick paralysis in Australia is the scrub tick, *I. holocyclus* (Lysyk, 2010). In Egypt, various tick species infested camels, cows, buffaloes, sheep, goats, rabbits, dogs, and cats, as well as birds were up to 32 genera of many tick species (Davis, and Hoogstraal, 1954; Hoogstraal, 1958; Hoogstraal *et al*, 1964; Arthur, 1965; Morsy *et al*, 1986; El Kammah *et al*, 2001; El-Bahnasawy and Morsy, 2008; Abdel-Shafy *et al*, 2012; Morsy, 2012, Saleh *et al*, 2016; Hassan *et al*, 2017). Generally, the ticks of medical and veterinary importance in the Egyptian localities belong to soft-tick family, Argasidae; genera *Argas*, *Ornithodoros* and *Otobius*), and hard-tick family, Ixodidae; genera *Amblyomma*, *Dermacentor*, *Haemaphysalis*, *Hyalomma*, *Ixodes*, and *Rhipicephalus* (Hoogstraal and Tatchell, 1985). The most common species were *O. savignyi* was described as African Ixodidae Egyptian tick. Also, genera *Hyalomma*, and *Rhipi-*

cephalus with the most important ixodid ticks infesting animals were mainly *H. excavatum*, *H. dromedarii*, *H. impeltatum*, *H. marginatum*, *R. annulatus* and *R. sanguineus*. The cattle tick, *R. annulatus* was the most important economic pest infesting cattle. The widely distributed Egyptian ticks were *H. dromedarii*, *H. impeltatum*, *H. excavatum*, *H. anatolicum*, *H. truncatum*, *H. marginatum*, *H. rufipes*, *H. turanicum*, *R. annulatus*, *R. sanguineus*, *R. turanicus*, *R. guilhoni*, *R. camicasi*, *A. lepidum*, *A. marmoreum*, and *A. variegatum*. *Hyalomma* species were found on camels and cows and *R. annulatus* was only on cows, and *Rhipicephalus* species infested camels, sheep, and dogs. *Amblyomma* species were found on the Sudanese imported camels.

In Egypt, four small children living in Giza rural farm were referred Cairo University's Hospitals with tick paralysis features. Children suffered from cough, vomiting, diarrhea, irritability and mild intermittent fever. Moreover, small girl and boy as well as their dogs suffered nervous manifestation confused with rabies; myasthenia gravis; botulism; diphtheritic polyneuropathy. By the removal of ticks and specific symptomatic treatment children were recovered. The incriminated ticks were *Rhipicephalus sanguineus* on dogs, *Hyalomma dromedarii* on camels and *H. anatolicum excavatum* and *Haemaphysalis* sp. on goats (Mosabah and Morsy, 2012). No doubt, the manifestations severity depended on tick saliva protein (Horka *et al*, 2012).

Pathogenesis: The onset of symptoms of tick paralysis occurs only after a female tick has attached and begun feeding. Symptoms do not typically develop until the tick has fed for four to seven days. The biological effect of tick salivary toxins is, in part, specific to the species of tick. Neurotoxins produced by *Dermacentor* ticks have a number of pathologic effects including (Felz *et al*, 2000): 1- Slowing of motor nerve conduction velocity, 2- Lowering of nerve height and muscle action potential, and 3- Impaired

propagation of afferent nerve fiber signals.

The precise mechanism for the development of the symptoms of tick paralysis was not fully understood. But, the *Dermacentor* spp. produced toxin interrupted sodium flux across axonal membranes in selected locations such as the nodes of Ranvier and nerve terminals (Krishnan *et al*, 2009). This led to weakness through impairment of the neural transmission to motor nerve terminals (Cooper and Spence, 1976).

Neurotoxins produced by *I. holocyclus* act on presynaptic motor nerve terminals and inhibit the release of acetylcholine. This inhibition can result in total blockade of transmission at myoneural junctions and is temperature-dependent. Low-amplitude compound muscle action potentials can be detected in neurophysiologic testing; motor conduction velocities, sensory studies, and repetitive stimulation are normal. The *I. holocyclus* toxin was not well characterized, as it gave clinical similarities to botulism toxin (Grattan-Smith *et al*, 1997). Carrillo-Marquez (2016) reported that botulism was a rare, severe, and potentially lethal condition caused by botulinum toxin. Signs and symptoms might include: 1-Difficulty swallowing, 2- Muscle weakness, 3-Double vision, 4-Drooping eyelids, 5-Blurry vision, 6- Slurred speech, 7-Difficulty breathing, and 8-Difficulty moving that began 18to 36hrs in foodborne botulism of contaminated food (CDC, 2021) .

Clinical Features: Tick paralysis by *Dermacentor* spp., symptoms and signs usually begins with paresthesia and a sense of fatigue and weakness, although some patients might appear irritable or restless and complain of muscular pain. Fever is characteristically absent, and there is no change in the sensorium or headache unless severe hypoxia or hypercarbia are present. Despite patients' reports of paresthesia, the sensory exam is typically normal. Most patients eventually develop an unsteady gait that progresses to an ascending complete paralysis. Deep tendon reflexes were characteristically absent. Respiratory paralysis and death can occur in

severe cases (Rose, 1954). Among 332 tick paralysis cases published over 60 years ago, fatality rate was 12%. By contrast, a more contemporary 33 series cases fatality rate was 6%. In most fatal cases, tick paralysis diagnosis was not considered ante-mortem (Vedanarayanan *et al*, 2004).

A number of clinically important but unusual features of tick paralysis deserve emphasis: a- Involvement of the facial, ocular, lingual and pharyngeal muscles may produce diplopia, dysphagia, and/or dysarthria. Bulbar muscle involvement may also result in unusual presenting symptoms such as drooling (Bow and Brown, 1946), b- Paralysis may be localized to one arm or leg, and isolated facial paralysis has occurred in patients who have ticks attached to their external ear canal. Patients with unilateral arm weakness have also been described, suggesting a brachial plexopathy associated with an engorged tick in the subclavian fossa (Pearn, 1977), c- Patients may present with findings such as ataxia of the arms and legs that spuriously suggest the presence of a cerebellar lesion (Engin *et al*, 2006), & d- Patients rarely present with ascending weakness and autonomic dysfunction that led to an incorrect diagnosis of Guillain-Barré syndrome.

The onset of tick paralysis caused by *I. holocyclus* is characteristically slow; patients may have gradually worsening symptoms over 48 to 72hrs until complete paralysis occurs. Patients from Australia with tick paralysis may simultaneously suffer from internal and external ophthalmoplegia (Chronic progressive), and pupillary reflexes may be absent, or minimally reactive early in illness course (Crawford and Mitchell, 2009).

Laboratory findings: White blood cell count and cerebrospinal fluid are normal. Hypoxia and hypercarbia may be present if respiratory muscles are involved.

Imaging findings: Imaging studies were characteristically in the normal tick paralysis patients, but in one case report an attached tick was first detected on scalp by magnetic resonance imaging of a child with acute tick

paralysis (Burke *et al*, 2005).

Differential Diagnosis: Tick paralysis can be confused with an array of disorders including Guillain-Barré syndrome (GBS), botulism, cerebellar ataxia, myasthenia gravis, poliomyelitis, acute spinal cord lesions, periodic paralysis due to hypokalemia, insecticide poisonings, exposure to buckthorn, shellfish poisoning, and even hysterical paralysis (Vedanarayanan *et al*, 2002).

Guillain-Barré syndrome is a group of neuropathic conditions characterized by progressive weakness and diminished or absent myotatic reflexes, resulted from an aberrant immune response attacking nerve tissue. Commonest one was acute inflammatory demyelinating polyradiculoneuropathy, presented as progressive motor weakness, usually in legs and advancing proximally with peak within four weeks, then plateau before resolving. Autonomic symptoms occurred, such as cardiac arrhythmias, blood pressure instability, or urinary retention, with advancing symptoms of respiration and vital functions (Walling, and Dickson, 2013)

Myasthenia gravis basic abnormality reduction in acetylcholine receptors (AChRs) at neuromuscular junctions due to the effects of autoantibodies that are directed against the AChRs in most patients or against neighboring proteins involved in the clustering of AChRs (MuSK, LRP-4, or agrin). Clinically, MG is characterized by muscle weakness and fatigue, often in the typical patterns (Drachman, 2016).

Marine bio-toxins usually reach human consumers by ingestion of contaminated seafood, although other exposure routes like inhalation or contact were reported and may cause serious illness including paralytic toxins, amnesic toxins, ciguatoxins, brevetoxins, tetrodotoxins, diarrhetic toxins, azaspiracids and palytoxins. Also, many thousands of swimmers were stung by jellyfish every year, with effects ranging from mild discomfort to serious injury or even death (Morsy *et al*, 2020). A review of 50 well-documented cases reported in USA from 1946 to 2006

concluded that the more recently diagnosed cases were more likely to be misdiagnosed as GBS. Misdiagnosis of GBS often leads to expensive and unnecessary therapy such as plasmapheresis and intravenous immunoglobulin infusions (Diaz, 2010).

Differentiating tick paralysis from these conditions should be relatively easy if the medical features were carefully considered or sought: a- A meticulous search will usually disclose the presence of a tick in cases of tick paralysis, b- Examination of the CSF is typically abnormal in patients with GBS, poliomyelitis, and in those with acute spinal cord lesions, whereas it is normal in patients with tick paralysis, c- Fever is not typically present in patients with tick paralysis. When fever is a prominent part of the prodromal or neurologic illness, tick paralysis is not a likely diagnosis, d- Unlike tick paralysis, botulism usually causes a descending paralysis, and the cranial nerves was early affected in illness. Pupillary abnormalities were common in botulism and uncommon in tick paralysis, e- Tick paralysis due to *Dermacentor* spp. bite generally progresses over time periods ranged from one or two hours to one or two days, whereas weakness and GBS paralysis, poliomyelitis or spinal cord lesions progressed more gradually, often over days to weeks, and f- Sensation is intact with patients with tick paralysis, but it is either mildly or obviously abnormal in patients with Guillain-Barré syndrome or spinal cord lesions.

Diagnosis and Management: When diagnosis of tick paralysis is suspected, a careful and meticulous examination should be performed to look for a tick. Special attention should be given to the scalp, axillae, ears, labia, buttocks, and the interdigital spaces. The use of a fine-tooth comb can detect ticks embedded in the scalp of people with long hair; such a comb should be used when the diagnosis of tick paralysis is considered likely and a tick cannot be found after a normal examination.

Response following tick removal: Most paralysis patients due to *Dermacentor* spp.

recovered or improved within a few hours after tick removal. However, weakness and paralysis may worsen for 24 to 48hrs after *I. holocyclus* was removed. So, patients with paralysis or weakness due to *I. holocyclus* must be observed carefully following tick removal.

Ixodes holocyclus antitoxin: A hyperimmune serum prepared from dogs was widely used to treat tick paralysis animals, but its use in humans was limited as risk of immune-mediated reactions included serum sickness. Thus, human paralysis due to *I. holocyclus* was usually managed with supportive care that may include mechanical ventilation until recovery occurs (Greenstein, 2002).

Conclusion

Ticks are obligate hematophagous ectoparasites and important vectors of a greater variety of medical and veterinary significance pathogens, causing economic losses associated with their infestation. Tick paralysis may be a rare disease caused by a toxin in tick saliva secreted while taking the blood meal. Symptoms more or less are typical poisoning with other neurologic disorders or diseases (Guillain-Barré syndrome or botulism or cerebellar ataxia, or myasthenia gravis, or poliomyelitis or diphtheritic polyneuropathy, and insecticide toxicity, exposure to buckthorn, shellfish poisoning, and even hysterical paralysis). The tick paralysis risk may be greatest for children in rural areas.

Recommendations

First of all control of ticks by environmental safe acaricides is a must. Neurotoxins pathologic effects including slowing of motor nerve conduction velocity, lowering of the height of the nerve and muscle action potential and impaired propagation of afferent nerve fiber signals.

Tick paralysis usually begins with paresthesia and a sense of fatigue and weakness, although patients sometimes appear irritable or restless with muscular pain, with or without fever, and without change in the sensorium or headache unless severe hypoxia or hypercarbia was present. Despite patients'

reports of paresthesia, the sensory exam is typically normal. Most patients eventually develop an unsteady gait that progresses to an ascending complete paralysis. Deep tendon reflexes are characteristically absent. Respiratory paralysis and death can occur in severe cases.

Clinically important but unusual tick paralysis features were involvement of facial, ocular, lingual, and pharyngeal muscles may produce diplopia, dysphagia, and/or dysarthria. Bulbar muscle involvement may also cause unusual symptoms such as drooling.

Paralysis may be on one arm or leg, and isolated facial paralysis occurred in patients with ticks attached to external ear canal. Patients with unilateral arm weakness have also been described, suggesting a brachial plexopathy associated with an engorged tick in the subclavian fossa. Patients may present with ataxia of arms and legs that spuriously suggest the presence of a cerebellar lesion.

When the diagnosis of tick paralysis is suspected, a careful and meticulous examination should be performed to look for a tick. Special attention should be given to the scalp, the axillae, the ears, labia, buttocks, and the interdigital spaces.

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