Case Report/Relato de Caso

Tick paralysis cases in Argentina

Casos de paralisia por carrapato na Argentina

Carlos Remondegui[1]

ABSTRACT

Tick paralysis (TP) occurs worldwide and is caused by a neurotoxin secreted by engorged female ticks that affects the peripheral and central nervous system. The clinical manifestations range from mild or nonspecific symptoms to manifestations similar to Guillain-Barré syndrome, bulbar involvement, and death in 10% of the patients. The diagnosis of TP is clinical. To our knowledge, there are no formal reports of TP in humans in South America, although clusters of TP among hunting dogs in Argentina have been identified recently. In this paper, clinical features of two cases of TP occurring during 1994 in Jujuy Province, Argentina, are described.

Keywords: Tick borne diseases. Tick paralysis. Rickettsioses. Argentina.

RESUMO

A paralisia por carrapatos (PC) ocorre mundialmente e é causada por uma neurotoxina secretada por carrapatos fêmea engolidores de sangue que afeta o sistema nervoso periférico e central. As manifestações clínicas variam de suave ou sintomas não específicos a manifestações semelhantes à síndrome de Guillain-Barré, envolvimento de bulbar, e morte em 10% dos pacientes. O diagnóstico de PC é clínico. A nosso conhecimento, não há nenhum relatório formal em seres humanos na América do Sul, embora grupos de PC entre cães de caça na Argentina, tenham sido identificados recentemente. Neste artigo, descrevo dois casos de PC ocorridos durante o ano de 1994 na província de Jujuy, Argentina.

Palavras-chaves: Doenças por carrapato. Paralisia por carrapato. *Rickettsias*. Argentina.

INTRODUCTION

Tick paralysis (TP) occurs worldwide and is caused by a neurotoxins secreted by engorged female ticks that affects the peripheral and central nervous systems; clinical manifestations range from mild or nonspecific symptoms to manifestations similar to Guillain-Barré syndrome, bulbar involvement, and death in a 10% of the patients[1]. Toxins may derive from a pathogen or symbiotic organism living within the host tick as with organisms that make tetrodotoxin, or from a combination of the host and a symbiotic organism creating a product together, or from the tick itself[2].

Although the precise mechanism of action of the neurotoxin is not understood, the tox-ins have been shown to block axonal sodium channels and also inhibit the release of acetylcholine at presynaptic motor nerve terminals, causing total neuromuscular blockade. Clunies Ross provided the first definitive evidence that paralysis was due to a toxin secreted by the tick[3]. Kaire was able to obtain a partially purified toxin by homogenizing 350-400 replete ticks. Sub-sequent work by Stone et al. has suggested that it is a protein neurotoxin with a molecular weight of 40,000-80,000. It has been named holocylotoxin or *ixovotoxin* but its chemical structure has not been fully identified[4].

The diagnosis of TP is clinical and is determined by the exclusion of other known etiologies presenting with the same symptoms[5]. Although persons of any age may be affected, small children are particularly susceptible[6].

In North America, TP is reported most commonly in the Rocky Mountains and northwestern regions of the United States and in Western Canada. To our knowledge, in South America there is a report of a case in Brazil[7] and recently was published TP in humans in Mexico[8].

At least 15 species of ixodid ticks, including many that harbor rickettsiae of undetermined pathogenicity, are reported to bite humans in Argentina[9].

Jujuy Province in Northwest Argentina is a subtropical region, bordering Chile and Bolivia. Geographically, it has four regions: Quebrada and Puna are high and arid areas, and the Valle and the Yungas are tropical rainforest regions. The predominant tick species in Jujuy is *Amblyomma cajennense* and already has been reported as a vector of authochthonous cases of Rocky Mountain spotted fever (RMSF) in this province[10]. It is extensively distributed throughout Northwestern Argentina, including Jujuy Province, and nymphs and adults are frequently identified from tick bite surveys of humans[9]. In Jujuy and throughout Argentina, tick-borne diseases were underestimated by much of the general population and even by the medical community, until we published descriptions of several cases of RMSF and serological evidence of *Ehrlichia chaffeensis*[10].

Tick paralysis is caused by over 40 species of ticks worldwide. Hard and soft bodied female ticks are thought to produce a neurotoxin capable of causing paralysis. In North America, tick paralysis in humans is usually caused by *Dermacentor andersoni* or *Dermacentor variabilis*[5]. In Australia, *Ixodes holocyclus* is responsible for most cases[10]. In Mexico tick paralysis caused by *Amblyomma maculatum*[8]. In Brazil TP was reported in cattle, sheep and goats, cajennense as the causative agent[11]. Clusters of TP among hunting dogs in Misiones Province, Argentina, have been also identified recently (Available from: http://www.misionesonline.net).

CASE REPORT

In this paper, clinical features of two cases of TP occurring during 1994 in Jujuy Province are described. Both patients were medical doctors who developed neurological symptoms associated with tick bite.

Patient 1 was a 40 year-old woman who experienced headache, facial numbness, dizziness, and malaise four days after visiting a tick-infested area in Jujuy. Physical examination revealed no fever, a right Bell`s palsy, and a small crusted lesion on her scalp where she had removed a tick two days earlier. A complete blood cell count, erythrocyte

1. Servicio de Infectologia y Medicina Tropical, Hospital San Roque, Ministerio de Salud de la Provincia de Jujuy, Jujuy, Argentina.
Address to: Dr. Carlos Remondegui. San Martin 330/4600, San Salvador de Jujuy, Jujuy, Argentina.
Phone: 55 388 4221 307
e-mail: remondegui@arnet.com.ar; infectologiajujuy@arnet.com.ar
Received in 30/08/2011
Accepted in 14/12/2011

sedimentation rate, chemistry profiles, and urinalysis were normal. Clinical improvement occurred after the fourth day of tick removal.

Patient 2 was a 41-year-old woman who experienced dizziness, irritability, slurred speech, and partial recent memory loss five days after removing a tick. She subsequently developed gait instability, inability to walk and drive her car, followed by severe malaise, arthromyalgias, headache, hypoacusia, blurred vision, and diplopia. On physical examination, she was afebrile, alert, and cooperative but bradypsychic, hypoacusic, and dysarthric. Her third, sixth, and eighth cranial nerves were affected. Horizontal nystagmus was present. There was a small scar behind the right ear where she had removed the tick. No meningeal signs were present; she was bradykinetic and ataxic, with abnormal gait and unable to walk and with a positive Romberg`s test and abnormal coordinating tests. A mild left upper limb palsy, hyporreflexia and mild hepatomegaly was present. A complete blood cell count, erythrocyte sedimentation rate, chemistry profiles, and urinalysis were normal. Blood cultures were negative. She has recovered after twenty days after onset of symptoms. See below neurological manifestations (**Table 1**).

TABLE 1 - Neurological manifestations.

Manifestation	Case patient 1	Case patient 2
Initial symptoms	facial numbness	headache
Consciousness	alert	alert, bradypsychic
Headache	yes	yes
Partial loss of recent memory	no	yes
Dysarthria	no	yes
Diplopia	no	yes
Horizontal nystagmus	no	yes
Impaired vision	no	yes
Hypoacusia	no	yes
Dizziness	yes	yes
Cranial nerve involvement	seventh	third, sixth, and eighth
Ataxia	no	yes
Finger to nose and fine motor testing	no	yes
Gait instability	no	yes
Vestibular syndrome	no	yes
Meningeal syndrome	no	no
Positive Romberg test	no	yes
Motor neurological deficit	no	yes, ULL
Deep tendon reflexes	normal	hyporreflexia ULL
Muscular strength	normal	decreased 3/5 ULL
Babinski sign	no	no

ULL: upper left limb.

DISCUSSION

In contrast to the dramatic, life threatening symptoms that follow soon after snake or spider bite, tick paralysis evolves slowly but it can be equally deadly in a 10% of the patients[1].

Tick paralysis has been frequently misdiagnosed, and this envenomation syndrome must be included in the differential diagnoses of any patient presenting with an ascending symmetrical paralysis.

The spectrum of neurological manifestations described for both of the patients, occurring two days after tick bite or exposure, the absence of fever, and the presence of normal laboratory values are keys to diagnosis TP. It is important to emphasize that the diagnosis is entirely clinical, as are the diagnoses of Kawasaki disease or rheumatic fever and several other syndromes. In Jujuy Province, we also must consider other endemic diseases, including lactrodectism, rickettsioses, Guillain-Barré syndrome, rabies, heavy metal intoxication, and insecticide poisoning.

The two patients visited tick-infected areas within 10km of each other as was described in Colorado, USA. Case 1 presented with an isolated facial Bell`s palsy, which has been described in other cases of TP[6]. Case 2 presented with a more severe clinical picture that included gait instability, inability to walk, muscular hypotonia, and hyporeflexia as reported previously[1]. The clinical course of paralysis produced by this tick is different from that of ticks in other continents. Australian TP differs from the North American variety in that patients are more acutely ill, paralysis may continue and progress for 48 hours after tick removal, and recovery also is prolonged[5]. Some patients become worse after tick removal[1]. Both cases described here showed features compatible with Australian variety of TP[5].

Cases of tick paralysis are not very common in humans compared to animals[2]. Many times, the tick is not identified therefore, just demonstrating the presence of a tick on the body is not sufficient to make a defin-itive diagnosis of tick paralysis[3]. Until now we do not know the cusative agent of TP in Argentina.

ACKNOWLEDGMENTS

I thank Christopher D. Paddock, Centers for Disease Control and Prevention, Atlanta, Georgia, USA. Research Medical Officer, Infectious Disease Pathology Branch, Centers for Disease Control and Prevention, Atlanta, Georgia, for critically reviewing the manuscript.

REFERENCES

1. Van Gerpen JA, Caruso S. Tick Paralysis. Mayo Clinic Proc 2005; 80:938.
2. Mans BJ, Gothe R, Neitz AW. Biochemical perspectives on paralysis and other forms of toxicoses caused by ticks. Parasitol 2004; 129 (supl):95-111.
3. Ross IC. An experimental study of tick paralysis in Australia. Parasitol 1926; 18:410-429.
4. Stone BF. Tick paralysis, particularly involving *Ixodes holocyclus* and other *Ixodes species. In*: Harris KF, editor. Advances in disease vector research, Vol. 5. New York: Springer-Verlag 1988; p. 61-85.
5. Purwar S. Editorial. Tick Paralysis: An Uncommon Dimension of Tick-Borne Diseases. Southern Medical J 2009; 102:190-192.
6. Pearn J. Neuromuscular paralysis caused by tick envenomation. J Neurol Sci 1977; 34:37-42.
7. Almeida RA, Haddad Jr V, Ferreira MA, Barraviera B. Human Tick Paralysis in Brazil: report of a case. Abstracts Book (Poster). IX Congresso Brasileiro de Toxinologia; 2010; Araxá: Sociedade Brasileira de Toxinologia.
8. Espinoza-Gomez F, Newton-Sanchez O, Flores-Cazares G, De la Cruz-Ruiz M, Melnikov V, Austria-Tejeda J, et al. Tick paralysis caused by *Amblyomma maculatum* on the Mexican Pacific Coast. Vector Borne Zoonotic Dis 2011; 11:945-946.
9. Guglielmone AA, Beati L, Barros-Battesti DM, Labruna MB, Nava S, Venzal JM, et al. Ticks (Ixodidae) on humans in South America. Exp Appl Acarol 2006; 40:83-100.
10. Ripoll CM, Remondegui CE, Ordonez G, Arazamendi R, Fusaro H, Hyman MJ, et al. Evidence of rickettsial spotted fever and ehrlichial infections in a subtropical territory of Jujuy, Argentina. Am J Trop Med Hyg 1999; 61:350-354.
11. Hall-Mendelin S, Craig SB, Hall RA, O' Donoghue P, Atwell RB, Tulsiani SM, et al. Tick paralysis in Australia caused by *Ixodes holocyclus* Neumann. Ann Trop Med Parasitol 2011; 105:95-106.
12. Serra-Freire NM. Tick paralysis in Brazil. Trop Animal Health Production 1983; 15:124-126.