KATARZYNA BARTOSIK

Porażenie kleszczowe

Tick paralysis

Streszczenie

Toksykozy u żywicieli może powodować ponad 50 gatunków kleszczy z rodziny *Ixodidae* i *Argasidae*. Wśród nich porażenie kleszczowe jest najbardziej niebezpieczne dla ludzi i zwierząt. Toksyny wywołujące porażenie są wydzielane przez komórki „b" pęcherzyków gruczołów ślinowych w drugiej fazie żerowania kleszczy.

Porażenie kleszczowe charakteryzuje się brakiem koordynacji ruchowej, ataksją, osłabieniem mięśniowym oraz postępującym symetrycznym paraliżem. Pierwsze objawy pojawiają się w kończynach dolnych i w przeciągu 1-2 dni rozprzestrzeniają się na tułów, kończyny górne, plecy i twarz. Zazwyczaj nie obserwuje się zmian czuciowych i gorączki. Występują także obniżenie szybkości przewodnictwa nerwowego i napięcia mięśniowego. W większości przypadków objawy ustępują szybko po usunięciu kleszcza.

Zapobieganie ukłuciom kleszczy i szybkie usunięcie żerujących pasożytów minimalizuje ryzyko rozwoju porażenia kleszczowego.

Słowa kluczowe: ślina kleszczy, ukłucia kleszczy, porażenie wiotkie wstępujące.

Abstract

Over 50 tick species of *Argasidae* and *Ixodidae* family can cause toxicoses in hosts. Among them tick paralysis is the most dangerous for people and animals. Toxins causing the syndrome are secreted by "b" cells in the salivary glands during tick feeding.

Tick paralysis is characterized by incoordination, ataxia, muscular weakness and ascending symmetrical paralysis without antecedent symptoms. The first signs of paralysis occur in the lower limbs, and then they extend to the torso, upper limbs, neck and face. There are no sensory changes and no fever. There also occurs a decrease in conduction velocity and muscle action potential amplitude. Symptoms usually recede rapidly upon removal of the tick.

Prevention of tick bites and prompt removal of the attached tick restrict the development of tick paralysis.

Key words: tick toxicosis, tick saliva, tick bites, ascending flaccid paralysis.

Chair and Department of Biology and Parasitology, Medical University of Lublin

INTRODUCTION

Tick paralysis (TP) is the major neurotoxin-mediated poisoning associated with tick feeding and was the earliest form of toxicosis described for ticks.

TP has been described for 55 species of Ixodidae and 14 Argasidae species [1]. The most important paralysis-inducing ticks include the hard ticks *Ixodes holocyclus* (Australia), *Dermacentor andersoni, Dermacentor variabilis*, *Amblyomma americanum*, *Amblyomma maculatum*, *Ixodes scapularis*, *Ixodes pacificus* (North America), *Ixodes rubicundus* and *Rhipicephalus evertsi evertsi* (South Africa). Soft ticks for which paralysis has been documented or suspected include *Argas walkerae*, *Argas arboreus*, *Ornithodoros capensis*, *Ornithodoros lahorensis*, *Ornithodoros savignyi* and *Otobius megnini*. From the species mentioned Australian tick, *Ixodes holocyclus* is responsible for most cases and probably is the most potent of all the world's paralysing ticks [2].

Several tick species in Poland are potential causative agents of tick paralysis in humans and animals. These include *Ixodes ricinus* (L.), *Ixodes hexagonus* Leach, *Ixodes crenulatus* Koch, *Haemaphysalis punctata* Can. et Fanz. and *Argas reflexus* (Fabr.). *Ixodes ricinus* occurring across Poland and *Argas reflexus* present in many cities inhabited by their hosts – pigeons - have the greatest medical importance. However, due to the increasing tourism in regions that are habitats of dangerous ticks, there is elevated risk of infestations of humans by other tick species and appearance of symptoms of paralysis, often only after returning to the country. That is why the possibility of such a condition should be in the physician's mind during any case of ascending flaccid paralysis or acute ataxia.

TICK BIOLOGY AND ECOLOGY

Most species causing tick paralysis are three-host ticks (each stage sucks the blood of a different host and attacks many species of hosts including humans). *Argasids* exhibit narrower host-specificity than Ixodidae; birds are their major host. In the case of the absence of birds, *argasid* ticks get to human flats through cracks in walls and at windows, and attack humans. Man can be attacked by various stages of ticks, mainly by nymphs and females. In adults, ticks attach under the knees, on inner thighs, in the armpits, the abdomen and chest, while in children - along the hairline behind the ears and on the neck.

Individual tick species have different temperature and humidity requirements. Environmental factors determine ticks' spread, survival, and activity. Toxin-producing Ixodidae usually prefer high humidity conditions. *Ixodes ricinus* can only be found in humid habitats of various, mainly mixed and deciduous, types of forest [3] and in urban woodland [4,5]. The tick is largely located along paths visited by its hosts. The development of *Argas reflexus* proceeds in low humidity conditions. The tick lives close to pigeons' nests in dry attics, garrets, or church towers.

Ticks are characterized by diurnal and seasonal activity. The diurnal rhythms are synchronized with host activity and diurnal changes in environmental conditions (temperature, humidity, and insolation). Larvae of the common tick exhibit the highest activity during the morning (about 8 a.m.), afternoon and night (from 1 p.m. to midnight) [6]. Nymphs and females are the most active in the morning and afternoon [7]. In turn, *argasids* display intensive activity at night. Even hungry individuals suck hosts' blood only after dusk [8]. Activity of adult stages of *Ixodes ricinus* in Poland usually extends from mid-April to early November and has two peaks: one in May and the other in September. Nymphs forage concurrently with adult stages, whereas larvae infest hosts in the period between May and September, most frequently in June, July and August. The time of the activity periods differs in other regions of Europe. Nymphs and adults of *Argas reflexus* from the Polish population posing a substantial toxicological threat [9] exhibit increased activity between April and June, which slightly declines from June to mid-October. The first larval activity peak is observed during the warm months (May-July), the second – between August and November [10].

Seasonal activity is also typical in the life cycle of the most dangerous species from the genus *Dermacentor*, which has to be borne in mind while planning visits in their geographical range. In the mountaineous regions of the USA, adult *Dermacentor andersoni* parasitizing a variety of domestic and wild animals, first of all cattle, horses, goats, sheep, roe deer, deer, and humans, are active in spring and early summer [11]. In the east Atlantic coast, California and several other western states, *Dermacentor variabilis* infests dogs, cattle, horses, hedgehogs, sheep, coyotes, foxes, skunks, rabbits and humans [12]. The activity of its larvae in these regions begins in March or April, increases in July and August, and declines in September or October. The activity of adult stages, which starts in March and April, reaches the peak in June or July, and ceases in August and September [11].

TICK TOXINS

According to Buczek et al. [4] compounds causing tick paralysis are produced in "b" cells in the salivary glands of the tick. Other kinds of cell (a,c,d,e,f) secrete substances important in feeding and causing the inflammatory reaction in the host's tissue.

The paralysis-causing toxin is part of the 40-100 kDa protein fraction [13,14]. The level of toxins rises during the foraging period and reaches a peak during intensive blood ingestion, i.e. ca. on day 5-7 after attachment to the host skin [15,16].

Tick feeding activity exhibits two phases. During the first, longer, usually 1-6-day long phase, ticks suck blood slowly and take up small amounts of blood. The second phase, the so-called rapid feeding phase, lasts from several hours to 1-2 days, after which ticks detach from the host. During the entire feeding process, the body weight of ticks from the family *Ixodidae* increases approximately 150-200-fold, while in ticks from the family *Argasidae* it is merely 10-15-fold greater. Occurrence of paralysis symptoms is strictly related to the increased count of granules in "b" cells of the salivary glands. The paralysis symptoms are most severe when "b" cells of the feeding tick contain the maximum

number of granules. The granules are <1μm in diameter and are PAS-positive [15].

CLINICAL SYMPTOMS

The course of tick paralysis depends on various factors, primarily on the tick species and stage of development, tick attachment site, body weight and individual sensitivity of the host. In most cases TP is recorded in children mainly in girls [17] and several per cent of them die as a result of paralysis of respiratory muscles. Children under 5 constitute a highest risk group, as in 70% of children ticks are usually attached in barely visible sites, i.e. on the neck, along the hairline and in hair [16,18,19]. As many as 16.4% cases of death (the highest mortality rate) have been reported when the tick was attached in those body regions [20]. In adults, deaths resulting from tick paralysis occur less frequently.

The incubation period of tick-borne paralysis lasts 3-8 days (usually 5) and depends on the type and amount of introduced toxins [16,21]. The symptoms of the first phase of the disease include malaise, fatigue, severe headaches, tingling sensation in extremities (lips and face) and projectile vomiting [22]. Subsequently, paralysis affects the lower extremities and within 1-2 days spreads to the upper parts of the body such as the torso, arms, back and face. There is a lack of coordination, ataxia (dysmetria and asynergy) and myasthenia. Patients cannot maintain an upright posture (astasia), walk (abasia) or move the arms [23]. Occasionally, myasthenia does not appear [24]. The course of TP may also include dysphagia; difficulty in chewing, speaking, and breathing; nystagmus and diplopia [25-28]; sometimes tachycardia and tachypnea [29]. The characteristic feature of TP is maximal neurologic impairment occurring within hours to days after the onset of symptoms. Body temperature and sensory responses to touch, prick, and pressure may remain unchanged. Atypical presentations including prominent bulbar palsy and isolated facial weakness without generalised paralysis as well as brachial plexus involvement caused by tick bite were also reported [30-32].

Tick paralysis caused by *Dermacentor andersoni* is characterized by reduction in motor nerve conduction [29,33,34], and reduction in the action potential [35] and muscle tone [29]. Electromyographic changes may occur in all prespinal and limb muscles. Spread of impulses in the sensory fibres is also retarded [29,36]. Reduction of conduction in the sensory and motor fibres is caused by disturbance in the release of acetylcholine in the neuromuscular junctions without inhibition of its synthesis [37]. The toxin of *Dermacentor* has a greater effect on the amplitude of the potential rather than the rate of conduction, which is related to changes in ions fluxes across cell membranes. It acts similarly to tetrodotoxin produced by fish from the family *Tetraodontidae*.

Studies on animals infested by the larvae of *Argas* (Persicargas) *persicus* demonstrated disturbed peripheral nerve conduction, especially in the fast-conducting nerve fibres [38]. The maximum conduction velocity in motor nerves decreased to 55% of the baseline. Toxins of ticks from the genus *Argas* induce paralysis of respiratory muscles and, consequently, hypoventilation, i.e. higher retention of CO_2 in the blood. Respiratory acidosis develops as a result of breathing disturbances, which in the absence of compensation impairs the function of vitally important organs and leads to death [39].

TREATMENT

Treatment of tick paralysis patients is mostly supportive and symptomatic. Finding the tick involves careful inspection of the entire body, especially of the areas covered with hair, axillae, the groins and beneath the breasts. In children ticks are usually found on the scalp, the occipital region, behind the ears, in external auditory canal and on the neck [19,28,40].

Removal of the tick results in rapid improvement and if the paralysis is not in advanced full recovery within hours to days depending on tick species (Australian TP Syndrome differs from North American one as the course of intoxication is more acute, paralysis may continue and progress for 2 days after tick removal and recovery is prolonged [18,41-43]. Due to the similarity of the clinical picture of TP to other diseases involving the peripheral nervous system, e.g. Guillain-Barré syndrome and toxic polyneuropathy, diagnosis of TP should be preceded by searching the entire body for a tick.

The differential diagnoses includes Guillain-Barré syndrome, botulism, shellfish poisoning, epidural abscess transverse myelitis and myasthenia gravis [44]. A differentiating point is the nature of development; ascending paralysis is seen in tick paralysis and Guillain-Barré syndrome, when the opposite is seen in paralytic shellfish poisoning and botulism. In contrast to Guillain-Barré syndrome there is an early onset of ocular symptoms in tick paralysis, ranging from dilated pupils that are unresponsive to light, to poor ocular convergence, and horizontal and vertical nystagmus as well as diplopia.

PREVENTION

Tick paralysis can be prevented by following several basic rules of protection against tick bites, which include:

1. Avoidance of tick habitats.
2. Proper clothing preventing ticks from contact with human skin. Wearing long trousers with legs either equipped with an elastic band or tucked into socks, long-sleeved blouses and rubber boots is recommended.
3. Careful checking the clothes and body after outings; before they find a convenient feeding site, ticks stay for some time on clothes or skin surface.
4. Use of tick repellents.

Prompt removal of ticks from the skin prevents introduction of large amounts of toxins into the blood during ingestion. Ticks should be removed mechanically by grasping them with tweezers close to the skin and pulling out with a swift twisting motion. The teeth-equipped tick's hypostome, anchored in the skin, offers lower resistance then. Although widely used, neither application of fat or other substances over the attached tick nor burning the parasite are recommended; thus irritated ticks inject more saliva into the wound. This in turn offers a possibility of rapid introduction of pathogens present in the salivary glands of these arthropods into the human body.

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Informacje o Autorce

Dr n. med. Kataryzna Bartosik – asystent, Katedra i Zakład Biologii i Parazytologii, Uniwersytet Medyczny w Lublinie.

Adres do korespondencji

Katarzyna Bartosik
Katedra i Zakład Biologii i Parazytologii,
Uniwersytet Medyczny w Lublinie
ul. Radziwiłłowska 11, 20-080 Lublin
tel/fax 81 528 84 63
e-mail: katarzyna.bartosik@umlub.pl