PARALYSIS IN *Gallus gallus* AND *Cairina moschata* INDUCED BY LARVAE OF *Argas (Persicargas) miniatus*

FERNANDO E. P. DE MAGALHÃES[2], CARLOS L. MASSARD[3] e NICOLAU M. DA SERRA FREIRE[2,3]

SINOPSE.- Magalhães F.E.P., Massard C.L. & Serra Freire N.M. 1987. [**Paralisia em Gallus gallus e Cairina moschata induzida por larvas de Argas (Persicargas) miniatus.**] Paralysis in Gallus gallus and Cairina moschata induced by larvae of Argas (Persicargas) miniatus. *Pesquisa Veterinária Brasileira 7(2):47-49.* Depto Biologia Animal, Univ. Fed. Rural do Rio de Janeiro, Km 47, Seropédica, RJ 23851, Brazil.

A ação tóxica da larva de *Argas (P.) miniatus* procedente do Estado do Rio de Janeiro foi estudada em condições de laboratório com temperatura e umidade controlada, utilizando-se pintos (*G. gallus* e patos (*C. moschata*) como hospedeiros. Lesões cutâneas do tipo ulcerativo foram evidenciadas após fixação das larvas, sendo o grau de patogenicidade mais acentuado quanto maior fosse o estado de ingurgitamento larvar. Sintomas clínicos caracterizados como paralisia, causada pela ação tóxica das larvas do carrapato foram observados em 20 pintos e quatro patos infestados com larvas, 3 a 5 dias após fixação das larvas. Incoordenação motora, aumento da sensibilidade, paralisia das patas e asas e flexão da cabeça para baixo foram os sintomas mais freqüentes, finalizando com morte por parada respiratória. À necropsia as lesões macroscópicas mais evidentes foram: atrofia esplênica, modificação da coloração e friabilidade do fígado e estase biliar. Exames histopatológicos revelaram lesões em órgãos como: pele, pulmão, coração, baço, fígado, rins e glândulas ad-renais. Esta foi a primeira citação de *A.P. miniatus* como agente indutor de paralisia.

TERMOS DE INDEXAÇÃO: Paralisia por carrapato, toxicose, *Argas miniatus*.

ABSTRACT.- A study was made of the toxic action of *Argas (P.) miniatus* Koch, 1844, using material collected in the state of Rio de Janeiro, Brazil. This study was conducted in the laboratory under controlled temperature and humidity conditions, using chickens (*Gallus gallus*) and ducklings (*Cairina moschata*) as hosts. Cutaneous ulcerative lesions were observed after larval attachment. The degree of pathogenicity increased with increasing engorgement of the larvae. Clinical symptoms characterized as tick paralysis were observed in 20 chicks and four ducklings after 3 to 5 days of larval infestation. The most frequent symptoms were: motor incoordination, increased sensibility, paralysis of the feet and wings and ventral flexion of the head. Death resulted from respiratory failure. Post-mortem, the most obvious macroscopic lesions were: atrophy of the spleen, biliary stasis and yellowing of the liver. Histopathological studies showed lesions of the skin, lungs, heart, spleen, kidneys, adrenal glands and liver. This is the first reference to the toxic effect or tick paralysis due to the larvae of *A. (P.) miniatus*.

INDEX TERMS: Tick paralysis, toxicosis, *Argas miniatus*.

INTRODUCTION

Argas (Persicargas) miniatus Koch, 1844 is one of the most common ectoparasites of domestic birds in Brazil. The importance of this species relates not only to the general debilitating effects of parasitism but relates also to the transmission of pathogenic agents and to its toxic reaction on the host. *A. miniatus* was first described by Koch (1844), in examination of samples derived from Demerara in British Guiana. The host species, however, was not identified nor was any mention made about its toxicity or of the number of specimens examined. Rohr (1909) reported work with *Argas (Persicargas) persicus* (Oken, 1818) in Brazil without considering the morphological differences with *A. miniatus* and did not make reference to the toxicity of this species for birds.

Neitz (1962), in South Africa, reported that tick paralysis is an acute or subacute disease of certain mammals and birds and is caused by a neurotoxin found in at least 20 members of the family Ixodidae, and four species of the family Argasidae. The disease is characterized by flacid paralysis which progresses rapidly from the body extremities to the central nervous system, causing incoordination and sensorial changes. Death frequently results from respiratory paralysis. Only *A. persicus* was incriminated as causing this disease in birds.

For a considerable time there has been controversy in respect of the differential identification of *A. miniatus* and *A. persicus*, which are common avian parasites in Europe, Asia and Africa. Kohls et al. (1970) in the investigation of species of the subgenera *Persicargas* in the Americas, showed that *A. miniatus*

[1] Accepted for publication on March 5, 1987.
Part of the M.Sc. Thesis presented by the first author at the Universidade Federal Rural do Rio de Janeiro (UFRRJ), under the auspices of CNPq and Embrapa.

[2] Laboratório Nacional de Referência Animal – Lanara, Cx. Postal 50, Pedro Leopoldo, Minas Gerais 33600, Brazil.

[3] Departamento de Biologia Animal, UFRRJ, Km 47, Seropédica, Rio de Janeiro 23851, Brazil.

is indeed a separate species from *A. persicus*. The species *A. persicus* appears to be rare in the new world countries although it has been found in Maryland, Pennsylvania, Georgia, California and Paraguay (Kohls et al. 1970). *A. miniatus* is now reported from Panama (Dunn 1923, Kohls et al. 1970), Venezuela (Vogelsang & Dias 1960, Jones et al. 1972), Brazil (Aragão & Fonseca 1961, Kohls et al. 1970), British Guyana, Colombia and Trinidad (Kohls et al. 1920) and Cuba (Cruz 1974). However, Rosenstein (1976), in Pennsylvania, examined 20 week-old Leghorn pullets, heavily infested with *A. persicus* larvae. It was noted that the birds were severely depressed and recumbent. The signs were quickly reversed when the tick larvae were removed. Findings were negative following detailed diagnostic procedures and this, together with the rapid recovery of disinfested animals, is suggested as indicative that the tick larvae caused the paralysis.

The present work has been undertaken to investigate the specific toxic activity of *A. miniatus* in its larval stage, in infestations of poultry in Brazil.

MATERIALS AND METHODS

Day-old Leghorn chicks were kindly supplied by the Ornithological Section of the Animal Health Research Unit (UAPNPSA) of EMBRAPA, Itaguaí/RJ, and ducklings one week old were supplied by the UFRRJ, Estação para Pesquisas Parasitológicas W.O. Neitz. Ticks identified as *A. miniatus* were obtained from naturally occurring infestations of poultry in the state of Rio de Janeiro. Larvae from these chicks were obtained under controlled laboratory conditions (28-30°C and 70-80% relative humidity) and were used for the experimental infestation of the two host species. Experiments were made to confirm that paralysis can be induced in chickens and ducklings following artificial infestation with larvae of *A. miniatus*.

Twenty chicks and four ducklings were each infested with 100 to 200 larvae. The larvae were placed directly on the skin of the birds and the latter were then enclosed in small cloth bags leaving only the head protruding. The infestations were made at night over a period of twelve hours during which time the birds remained enclosed in their bags to facilitate the contact between host and parasite. The bags were then removed and the birds were observed daily. An additional 20 chicks and 4 ducklings, which had not been infested were maintained under similar conditions. The degree of engorgement, the natural fall-off of the larvae, the cutaneous lesions and the symptoms of the hosts were recorded.

Blood was collected from birds which showed signs of paralysis for intraperitoneal inoculation of sensitive birds of the same species. All the larvae were removed on the 5th day from 2 chickens which had developed paralysis and did not die. These birds were observed daily thereafter for 15 days.

Post-mortem examinations were made on 9 of the birds (7 chicks and 2 ducklings) which had shown signs of paralysis and died, and in the 2 chicks which were killed 15 days after removal of the larvae before engorgement. Internal organs were examined macroscopically and fragments were immersed in 10% formalin for fixation for subsequent histopathological examinations. The preparation and examination of specimens submitted for histopathology was very kindly undertaken by the Department of Veterinary Surgery of the Federal University of Minas Gerais.

RESULTS

Without exception, all the birds (20 chicks and 4 ducklings) subjected to infestation of tick larvae developed symptoms of disease, characterized principally as a progressive paralysis, on the 2nd and 3rd day after infestation. In one group of 10 chickens and 1 duckling the numbers of larvae used for individual infestation (number applied) and the numbers of larvae, which reached the engorged state (number collected), are recorded in Table 1. In all cases, the first signs of disease which were observed was the onset of motor incoordination. This was followed by dysphagia, hypersensitivity and paralysis of the wings and legs. As the latter condition developed, the birds assumed first a sitting position and later presented a picture of apathy with the head and neck flexed in a drooping position. Finally, the birds reached a state of torpor shortly before death. The disease had a course of 7 days.

Table 1. *Experimental larval infestation of 10 chickens* (Gallus gallus) *and one duckling* (Cairina moschata) *with* A. (P.) miniatus

Host[a]	Number of larvae	
	Applied	Collected up to death of host
D 01	160	28
C 01	104	39
C 02	109	26[b]
C 03	129	31
C 04	153	31[b]
C 05	130	27
C 06	194	12
C 07	200	57
C 08	100	16
C 09	100	18
C 10	100	20

[a] D = duckling, C = chicken.

[b] Larvae were removed on 5th day, before complete engorgement.

During their infestation on the host, the larvae caused cutaneous ulcerative lesions which increased in size and severity during the growth period of the larvae. However, a continuous regression of the lesions commenced after the engorged larvae naturally detached from the host, or after partially engorged larvae were experimentally removed, leaving only a minimal scar to mark their presence.

No evidence of disease was observed after the intraperitoneal injection of blood from diseased birds into sensitive animals of the same species.

In the case of 2 chickens, which had shown typical progressive signs of tick paralysis arising from partially engorged larvae, a return to normal health was made after the larvae had been removed. At post-mortem examination the most evident macroscopic lesions were: splenic atrophy, yellowish discoloration of the liver, biliary stasis and an increase of fluid in the anterior portion of the alimentary tract. Histopathological examinations showed the presence of lesions in the skin, cerebrum and cerebellum, lungs, heart, spleen, kidneys, adrenal gland and liver. In comparison with tissues from normal chickens of a similar age, the skin lesions were hypertrophy and erosion of the epidermis in the region of fixation to the host and, in some cases, ulceration of the tissue; oedema and haemorraghes were observed in the tissues adjacent to the subcutaneous lesions. Proliferation of mononuclear cells and formation of fibrous tissue was observed only in 1 case. Cerebrum and cerebellum: areas of focal cellular proliferation and discrete oedema were observed in one sample of cerebral cortex. Lungs: reduction of the lumen of bronchii, congestion and discrete haemorrhagic foci ot the alveolae. Heart: cardiac fibres with nuclear hypertrophy and peripheral condensation of chromatin, necrotic areas with dissociation of cardiac fibres and coagulation, cloudy swelling and interstitial oedema. Spleen: follicular hypertrophy with absence of germinative centres. Kidneys: cloudy swelling of tubular epithelium and deposits of proteic material in the lumen. Adrenal gland: cortical hypertrophy. Liver: hypertrophy of the liver cells, formation of fat globules and cloudy swelling;

necrotic foci with cellular dissociation and coagulation were observed in one sample.

In the 2 chicks, which after removal of the larvae returned to normal health, no lesions were found.

DISCUSSION

The present study of tick paralysis caused by the toxic action of tick larvae in infestation of chickens and ducklings illustrates two original aspects: 1) this report appears to be the first recorded description of the disease in birds born and reared in Brazil; and 2) the causative agent of the disease has been identified as *Argas (Persicargas) miniatus*, although *A. persicus* has been identified with tick paralysis on several previous occasions in various countries. A review of the literature was presented by Neitz (1962) and Rosenstein (1976).

In a more recent review Gothe et al. (1979) showed that the capacity for ticks to produce paralysis has been demonstrated, described or suspected in a total of 43 different species in 10 genera. In the genera *Argas* the species reported were: *A. arboreus* Kaiser, Hoogstraal and Kohls; *A. persicus* (Oken); *A. radiatus* Railliet; *A. reflexus* (Fabricius); *A. sanchezi* Dugés, and *A. walkerae* Kaiser and Hoogstraal.

In this study symptomatic picture of tick paralysis caused by *A. miniatus* larvae, form the first manifestations of muscular incoordination to the final lethal effect, is very similar to that described by Neitz (1962), by Emmel (1945) and by Gothe (1971) in their reports of *A. persicus* infestations.

The high mortality caused by the toxicosis of *A. miniatus* and the rapid recovery of the hosts when the ticks are removed before becoming completely engorged is similar to tick paralysis provoked in birds by other species of ticks. Rosenstein (1976) and Gothe (1971) were unanimous in affirming that the removal of the larvae before complete engorgement favoured recuperation of the host and agreed with respect to the relationship of the number of parasites and the severity of the symptoms. Individual birds infested with 100 to 200 larvae showed almost the same picture. The small differences that were observed are not regarded as significant in the clinical or pathological aspect, except that with the greater number of larvae the number of cutaneous lesions were also proportionately greater.

The general opinion is that there is a strong relation between the state of engorgement of the larvae, the degree of toxicosis and the regression of cutaneous lesions after removal of the parasites. This same relationship was observed in this study.

In all cases, chickens and ducklings alike, in which the disease progresses, death is caused by respiratory failure. This observation confirms the findings of Gothe et al. (1971) and of Kunze & Gothe (1971), who using clinical studies supported with electrocardiographic and electromyographic investigations, demonstrated that the respiratory muscles are primarily affected while the heart functions are only influenced indirectly at a later stage. Also Rosenstein (1975) showed that birds which died from tick paralysis caused by *A. persicus* were negative in tests using material collected from hosts for research of possible virus or bacterial infections. Resulting from these observations it can be affirmed that respiratory failure is the immediate cause of death, even though lesions are present in the principal organs.

Acknowledgements.- The authors wish to record their gratitude to the late Professor W. O. NEITZ for his help in the orientation of this research and his collaboration regarding the basic scientific literature. They are also grateful to Dr. Jack Rodican for his stimulus and collaboration in the preparation of this paper.

REFERENCES

Aragão H. & Fonseca F. 1961. Notas de Ixodologia. VIII. Lista e chave para os representantes de Fauna Ixodológica Brasileira. Mem. Inst. Oswaldo Cruz 59(2): 155-179.

Cruz J. 1974. Notas adicionales a la fauna de garrapatas (Ixodoidea) de Cuba. I. Argasidae de las aves. Poeyana (La Habana) nº 129, p. 1-3.

Dunn L.H. 1923. The ticks of Panama, their hosts, and the diseases they transmit. Am. J. Trop. Med. and Hygiene 3, p. 91-92.

Emmel M.W. 1945. So-called tick paralysis in chickens. J. Am. Vet. Med. Ass. 106: 108.

Gothe R. 1971. Die durch *Argas (Persicargas) persicus* Larven bedingte Paralyse der Huehner. I. Ueber den Einfluss des Saugzustandes und der Infestationsrate auf die klinische Manifestation. Z. Parasitenk. 35: 298-307.

Gothe R., Kunze K. & Mechow O. 1971. Untersuchungen ueber die Todesursache bei der durch *Argas (Persicargas) persicus* Larven bedingten Zeckenparalyse der Huehner. Tropenmed. u. Parasitol. 22(4): 430-435.

Gothe R., Kunze K. & Hoogstraal H. 1979. The mechanisms of pathogenicity in the tick paralysis. J. Med. Entomol. 16(5): 357-369.

Jones E.K., Clifford C.M., Keirans J.E. & Kohls G.M. 1972. The ticks of Venezuela (Acarina: Ixodidea) with a key to the species of *Amblyomma* in the Western Hemisphere. Brigham Young Univ. Sci. Bull. Biol. Ser. 17(4): 1-40.

Koch C.L. 1844. Systematische Uebersicht ueber die Ordnung der Zecken. Arch. Naturgesch. 10(1): 217-239.

Kohls G.M., Hoogstraal H., Clifford C.M. & Kaiser M.N. 1970. The subgenus *Persicargas* (Ixodoidea, Argasidae) Argas. 9. Redescription and new world records of *Argas (P.) persicus* (Oken), and ressurection, redescription and records of *A. (P.) radiatus* (Railliet), *A. (P.) sanchezi* (Dugés) and *A. (P.) miniatus* (Koch), new world ticks misidentified as *A. (P.) persicus*. Ann. Entomol. Soc. Am. 63(2):590-606.

Kunze K. & Gothe R. 1971. Die durch *Argas (Persicargas) persicus* Larven bedingte Paralyse der Huehner. III. neurophysiologische Untersuchungen. Z. Parasitenk. 36:251-264.

Neitz W.O. 1962. The different forms of tick toxicosis: a review. 2nd Meeting of the FAO/OIE Expert Panel on Tick-born Diseases of Livestock, Cairo, 3-10 December, p. 20.

Rohr C.J. 1909. Estudos sobre Ixodidas do Brasil. Trabalho do Instituto Oswaldo Cruz, Ed. Gomes, Irmão & Cia, Rio de Janeiro, 226 p.

Rosenstein M. 1976. Paralysis in chickens caused by larvae of the poultry tick, *Argas persicus*. Avian Diseases 20(2): 407-409.

Vogelsang E.G. & Dias J.A.T.S. 1960. Nova contribuição para o estudo da fauna ixodológica da Venezuela. Anais dos Serviços de Veterinária e Indústria Animal de 1953-1954 (Mozambique?), p. 21-25.