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Source: Journal of Wildlife Diseases, 25(1) : 70-75

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-25.1.70

Journal of Wildlife Diseases, 25(1), 1989, pp. 70–75

DISTEMPER-LIKE DISEASE AND ENCEPHALITOZOONOSIS IN WILD DOGS (*LYCAON PICTUS*)

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ABSTRACT: Clinical signs of a fatal disease resembling those of canine distemper were observed in two groups of captive wild dog (*Lycaon pictus*) pups 13 days after vaccination with a commercially available combination vaccine for dogs which contained a live attenuated strain of canine distemper virus. Histopathological examination of tissues revealed the presence of intranuclear inclusion bodies in neurons and lesions resembling canine distemper as well as colonies of an *Encephalitozoon* sp. in the central nervous system and kidneys. Lesions were observed in both organs which resembled those described in other species infected with *Encephalitozoon cuniculi*.

Key words: Distemper-like disease, canine distemper vaccine, wild dog, *Lycaon pictus*, *Encephalitozoon* sp., encephalitozoonosis.

INTRODUCTION

Clinical signs resembling those of canine distemper have been observed in free-living (Reich, 1981) and captive wild dogs (*Lycaon pictus*) (Van Heerden, 1985) as well as in captive animals recently vaccinated against canine distemper (Van Heerden et al., 1980; McCormick, 1983). Distemper-like inclusion bodies in cells have previously been reported in tissues examined from wild dog pups which have died of suspected canine distemper (McCormick, 1983; Van Heerden, 1985). Without exception, the disease was not confirmed virologically (Hofmeyr, 1956; Van Heerden et al., 1980; McCormick, 1983; Van Heerden, 1985).

Infection with *Encephalitozoon* sp. has been diagnosed in laboratory animals (rabbits, mice and rats) by Shadduck et al. (1979), captive muskrats (*Ondatra zibethica*) by Wobeser and Schul (1979), squirrel monkeys (*Saimiri sciureus*) by Anver et al. (1972) and Zeman and Baskin (1985), humans by Margileth et al. (1973), birds by Poonacha et al. (1985), captive suricates (*Suricata suricatta*), clouded leopards (*Neofelis nebulosa*) and arctic foxes (*Alopex lagopus*) by Vávra et al. (1971), dogs by Botha et al. (1979) as well as in the domestic cat by Van Rensburg and Du Plessis (1971). Clinical signs of disease are accompanied by immuno-incompetency (Margileth et al., 1973) in humans and in general with newborn or young (Anver et al., 1972; Brown et al., 1973; Zeman et al., 1985) captive animals (Vávra et al., 1971; Zeman and Baskin, 1985). Clinical signs of disease in domestic dog pups have been described by Botha et al. (1979). Experimental infection in laboratory animals (rabbits, mice, rats and squirrel monkeys) often does not result in clinical disease (Shadduck et al., 1979). Positive antibody titres to *Encephalitozoon cuniculi* have been found in apparently healthy animals (Stewart et al., 1979).

This report presents the first recorded occurrence of an *Encephalitozoon* sp. in wild dogs as well as the occurrence of distemper-like clinical signs in two unassociated groups of wild dog pups after vaccination against canine distemper with a commercially available combined vaccine.

MATERIALS AND METHODS

Group one

Six 8-wk-old apparently healthy wild dog pups were vaccinated with a live canine distemper/canine hepatitis/parainfluenza/parvovirus combination vaccine (Quantum 4, Pitman-

Moore, P.O. Box 344, Washington Crossing, New Jersey 08560, USA). These puppies were the remainder of a litter of 10 born in captivity at the Hartebeestpoortdam Snake Park (Box 109, Hartebeestpoortdam, Republic of South Africa) which were removed at the age of 1 wk from the mother and subsequently hand-reared. Four pups disappeared prior to removal of the litter. The pups were hand-reared at a private home where initially they were kept indoors and later in an outdoor enclosure with concrete flooring. They did have contact with domestic dogs. The parents were held in enclosures with partial concrete flooring. Their vaccination status was uncertain. A variety of carnivores, primates, antelopes and birds also were kept in the zoo. An *Encephalitozoon* sp.-like organism also has been identified in necropsy material of a marmoset (*Callithrix jacchus*) from the zoo (J. Van Heerden, 1985, unpubl. data).

Thirteen days after vaccination all the pups showed signs of disease such as crying, paresis and convulsions. The clinical signs had an acute onset. Within 36 hr of the onset, three dead puppies were presented at the Faculty of Veterinary Science, Medical University of Southern Africa (Medunsa, Republic of South Africa) for necropsy and another two for clinical investigation.

Group two

A litter of four apparently healthy 11-wk-old wild dog pups were vaccinated with a modified live distemper/hepatitis/parvovirus-enteritis/canine adenovirus 2/parainfluenza/*Leptospira canicola*/*L. icterohaemorrhagiae* bacterin combination vaccine (Quantum 6, Pitman-Moore). These pups were housed with their parents in an enclosure in the National Zoological Gardens (Pretoria, Republic of South Africa). Within 13 days of vaccination, three pups (females) died and were presented for necropsy. The remaining pup (male) was presented in extremis at the same time for clinical examination and subsequent necropsy.

Laboratory and necrospy procedure

Individuals from both groups were examined physically. Specimens (cerebrospinal fluid, blood and serum) were collected from the two living puppies in Group one. Subsequently, they were killed with an intravenous overdose of barbiturates (Euthanaze, Centaur Labs, 36 Durban Street, Johannesburg, Republic of South Africa). Cerebrospinal fluid was analysed for protein concentration (ASTRA 8, CSF mode, Beckman Instruments Inc., Brea, California 92621, USA). Serum specimens were analysed for the concentrations of sodium, potassium, chloride, urea, creatinine, total proteins, albumin, phosphorus, bilirubin and the activities of alkaline phosphatase, aspartate transaminase, lactic dehydrogenase, creatine kinase and alanine transaminase according to described methods (Van Heerden, 1986). The haemoglobin concentration; haematocrit; and erythrocyte, white cell and platelet counts were determined from blood samples. Cerebrospinal fluid also was cultured in blood agar and MacConkey's agar (Biolab Chemicals, Box 14574, Verwoerdburg 0140, Republic of South Africa).

A complete necropsy was performed on each animal. Fixed organ specimens (10% buffered formalin) were processed routinely and stained with haemotoxylin and eosin and Gram's stain for light microscopy.

RESULTS

Clinical signs

Pups in Group one presented in a terminal stage of disease with tetraparalysis; intermittent, severe, generalized convulsions; intermittent clonic muscle contractions; opisthotonus; complete absence of pupillary reflexes; and diffusely spread papules and pustules over the skin of the inguinal and lower abdominal areas. The remaining pup in Group two showed circling, falling into objects, ataxia and incoordination. It emitted a high-pitched cry.

Clinical pathology

The protein concentration in the cerebrospinal fluid of one puppy was highly elevated (0.095 g/dl). The other specimen was contaminated with blood. An elevated urea concentration (64.2 mmol/dl) was found in the serum of one pup. Both specimens showed a mild hypergammaglobulinaemia (3.5 g/dl) caused by an increase in alpha and beta globulins and a leukocytosis ($29.0 \times 10^3/\mu l$ and $40.2 \times 10^3/\mu l$). The concentrations or activities of the other parameters were all within normal range for wild dogs (Van Heerden, 1986). No bacterial or mycotic growth was obtained on culturing of the cerebrospinal fluid.

Necropsy

The macroscopic lesions in Group one included marked pulmonary congestion

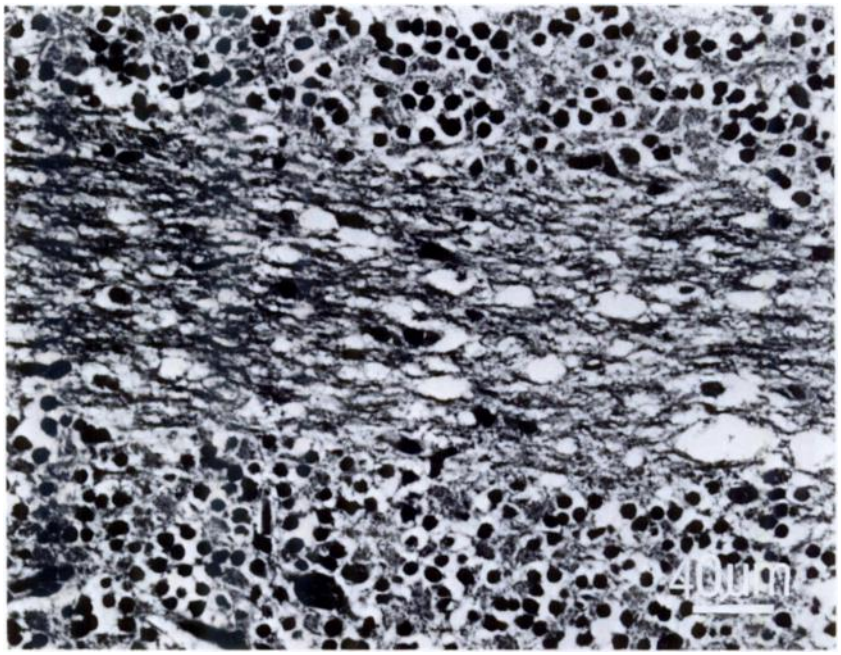

FIGURE 1. Low magnification of the status spongiosis observed in the white matter of the cerebellum of wild dog pups with distemper-like disease. H&E.

with multiple areas of red hepatization, slight hydropericardium, cerebral oedema, meningeal congestion and pustules in the skin of the ventral abdomen.

Histopathologically, the lesions were similar in all five pups in Group one. Multifocal lesions occurred in the brain stem, cerebrum and cerebellum. They consisted of neuronal swelling and malacia, gliosis and patchy demyelination (Figs. 1, 2). Eosinophilic, amorphous, intranuclear inclusions occurred in a few neurones in the affected areas. A severe meningitis was present over the cerebrum and the cerebellum. The cell reaction here was constituted by macrophages, lymphocytes and a few neutrophils. Elsewhere, and unrelated to the above, microgranulomas consisting of aggregates of glial cells, lymphocytes and a few macrophages were observed predominantly in the gray matter in the cerebrum and the cerebellum. Some of the granulomas occurred in close association with small blood vessels. *Encephalitozoon* sp.-like organisms were seen singly or in small groups in the microgranulomas or occurred as encysted colonies unassociated with a tissue reaction (Fig. 3). The organisms were observed with difficulty in the H&E stained sections. They were gram-positive, ovoid and ranged in size from 2.4 to 2.8 × 0.8 to 1.6 μm.

Small focal cortical and medullary lesions were present in the kidney. These consisted of small necrotic foci surrounded by a scant lymphocyte and macrophage cell reaction. Organisms similar to those in the brain occurred free or in small colonies in close association with the inflammatory foci.

Histological lesions in the other organs included an acute interstitial pneumonia, purulent dermatitis and severe lymphoid atrophy. No intranuclear or intracytoplasmic inclusions were observed in these organs.

Macroscopic findings in Group two wild dogs included pulmonary edema, hepatomegaly, enteritis and gastritis. Microscopic examination of the organs of females revealed severe splenic lymphoid atrophy and accompanying necrosis, an

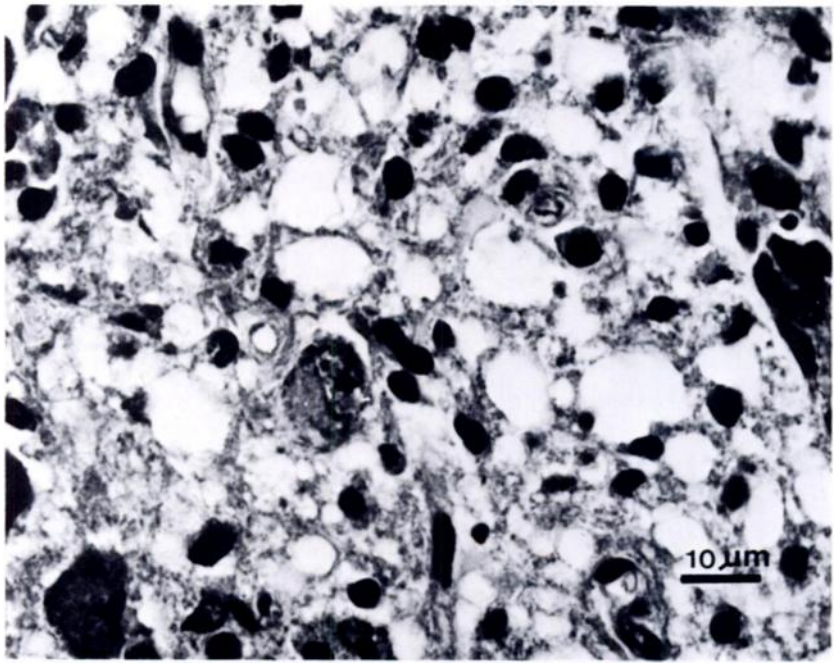

FIGURE 2. Higher magnification of an area manifesting a status spongiosis and a mild diffuse gliosis in wild dog pups with distemper-like signs. H&E.

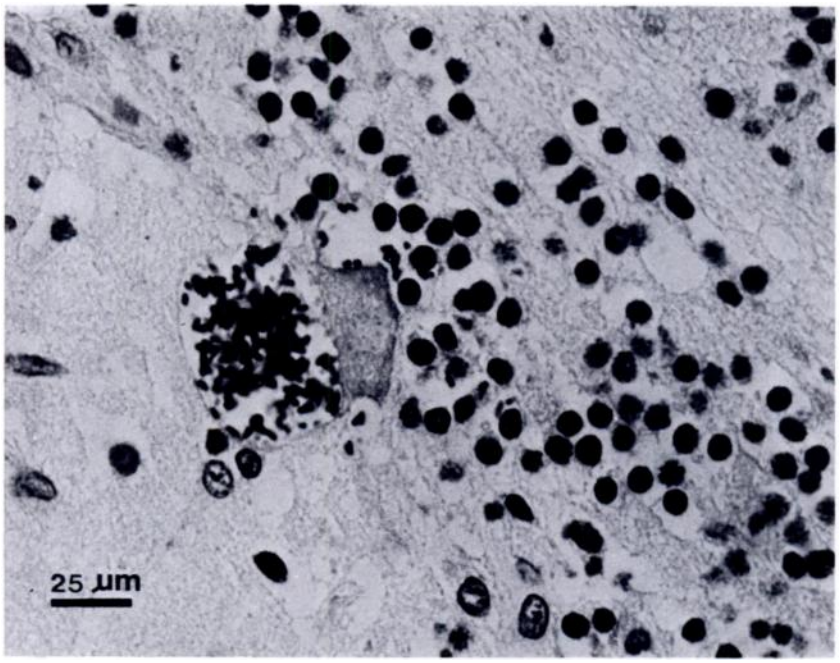

FIGURE 3. Intracytoplasmic *Encephalitozoon* sp. in Purkinje cells in the cerebrum of wild dog pups. Gram's stain.

acute mild to moderate necrotic enteritis and a focal acute necrotic bronchitis. Intracytoplasmic eosinophilic inclusions occurred in the bronchial epithelial cells in affected areas of the bronchi. Changes were not observed in the central nervous system.

DISCUSSION

These two case reports provide further circumstantial evidence for the etiological association between a live canine distemper vaccine and a neurological disease clinically resembling canine distemper. The antemortal nervous signs observed in Group one puppies were typical of a viral disease like acute canine distemper. Circumstantial evidence of acute fatal disease following within 13 days of vaccination indicate a vaccine-induced disease. However, this remains speculative until the question is resolved by viral isolation.

A live canine distemper vaccine has been used in well over 140 instances in captive wild dogs of all age groups without any observable side-effects (Van Heerden, 1985). However, in the present reports, a different strain of attenuated live virus was used. It is possible that this particular vaccine virus strain is still potentially pathogenic in wild dogs. These vaccines were developed for use in domestic dogs only. In Group one animals a combination of histopathological lesions including an acute lymphocytic diffuse meningitis, multifocal lymphocytic encephalitis, neuronal degeneration and necrosis, demyelination, neuronal intranuclear inclusion bodies and marked lymphoid atrophy is suggestive of a viral encephalitis similar to canine distemper in dogs. Although intranuclear inclusions in the central nervous system only occurred in neurons, they morphologically resemble those of canine distemper. The other viruses included in the vaccine are not known to cause intranuclear inclusions in neurons or astrocytes in the central nervous system.

Although widespread vaccination of wild dogs against canine distemper is practiced, the possibility of vaccine-induced disease remains and preference should be given to vaccines which appeared to have produced good results (Van Heerden, 1985). Concomitant infection with *Encephalitozoon* sp. may increase the risk of vaccine-induced disease.

Group one cases were complicated by the presence of a multifocal microgranulomatous encephalitis and nephritis associated with *Encephalitozoon* sp.-like organisms in the brain and kidney. Although dogs and other carnivores infected with *Encephalitozoon cuniculi* sometimes develop nervous signs (Botha et al., 1979; Mohn, 1982), it is not a consistant clinical finding. Clinical and blood chemistry results (Mohn, 1982; Botha et al., 1986) in dogs suffering from encephalitozoonosis are nonspecific; although nervous signs do occasionally occur, the general unthriftiness characterizing encephalitozoonosis makes clinical diagnosis difficult. Urine sediment smears may aid in confirming the diagnosis clinically. Anemia, which was not observed in these cases, has a late onset.

An unexplained high mortality rate among wild dog pups, for which various theories have been proposed, has been reported in both captive and free-living populations (Van Heerden, 1986). It is possible that infection with *Encephalitozoon* sp. may be another contributing factor under certain circumstances. Further investigation of the possible prevalence and epidemiology of this condition in wild dogs is presently hampered by the lack of a specific technique for antibody determination of its occurrence in wild dogs.

LITERATURE CITED

ANVER, M. R., N. W. KING, AND R. D. HUNT. 1972. Congenital encephalitozoonosis in a squirrel monkey *Saimiri sciureus*. Veterinary Pathology 9: 475–480.

BOTHA, W. S., I. C. DORMEHL, AND D. J. GOOSEN. 1986. Evaluation of kidney function in dogs suffering from canine encephalitozoonosis by standard clinical pathological and radiopharmaceutical techniques. Journal of the South African Veterinary Association 57: 79–86.

———, A. F. VAN DELLEN, AND C. G. SWART. 1979. Canine encephalitozoonosis in South Africa.

Journal of the South African Veterinary Association 50: 135–144.

BROWN, R. J., D. K. HINKLE, W. P. TREVETHAN, J. L. KUPPER, AND A. E. MCKEE. 1973. Nosematosis in a squirrel monkey (*Saimiri sciureus*): First reported case. Journal of Medical Primatology 2: 114–123.

HOFMEYR, C. F. B. 1956. Two hundred and eighty-four autopsies at the National Zoological Gardens, Pretoria. Journal of the South African Veterinary Medical Association 27: 263–282.

MARGILETH, A. M., A. J. STANO, R. CHANDRA, R. NEAFIE, M. BLUM, AND R. M. MCCULLY. 1973. Disseminated nosematosis in an immunological compromised infant. Archives of Pathology 95: 145–150.

MCCORMICK, A. E. 1983. Canine distemper in African cape hunting dogs *Lycaon pictus*—Possibly vaccine induced. Journal of Zoo Animal Medicine 14: 66–71.

MOHN, S. F. 1982. Experimental encephalitozoonosis in the blue fox. Acta Veterinary Scandinavica 23: 503–514.

POONACHA, K. B., P. D. WILLIAM, AND R. D. STAMPER. 1985. Encephalitozoonosis in a parrot. Journal of the American Veterinary Medical Association 186: 700–702.

REICH, A. 1981. The behaviour and ecology of the African wild dog *Lycaon pictus* in the Kruger National Park. Ph.D. Dissertation. Yale University, New Haven, Connecticut, 425 pp.

SHADDUCK, J. A., W. T. WATSON, S. P. PAKES, AND A. CALI. 1979. Animal infectivity of *Encephalitozoon cuniculi*. The Journal of Parasitology 65: 123–129.

STEWART, C., W. S. BOTHA, AND A. F. VAN DELLEN. 1979. The prevalence of *Encephalitozoon* antibodies in dogs and an evaluation of the indirect fluorescent antibody test. Journal of the South African Veterinary Association 50: 169–172.

VAN HEERDEN, J. 1985. Reproduction in captive hunting dogs *Lycaon pictus*. South African Journal of Wildlife Research 15: 80–84.

———. 1986. Disease and mortality of captive wild dogs *Lycaon pictus*. South African Journal of Wildlife Research 16: 7–11.

———, W. H. SWART, AND D. G. A. MELTZER. 1980. Serum antibody levels before and after the administration of live canine distemper vaccine to the wild dog *Lycaon pictus*. Journal of the South African Veterinary Association 51: 283–284.

VAN RENSBURG, I. B. J., AND J. L. DU PLESSIS. 1971. Nosematosis in a cat: A case report. Journal of the South African Veterinary Association 42: 327–331.

VÁVRA, J., K. BLAZEK, N. LAVICKA, I. KOCZKOVA, S. KALAFA, AND M. STEHLIK. 1971. Nosematosis in carnivores. The Journal of Parasitology 57: 923–924.

WOBESER, G., AND J. C. L. SCHUL. 1979. Microsporidial encephalitis in muskrats. Journal of Wildlife Diseases 15: 413–417.

ZEMAN, D. H., AND G. B. BASKIN. 1985. Encephalitozoonosis in squirrel monkeys *Saimiri sciureus*. Veterinary Pathology 22: 24–31.

Received for publication 15 September 1987.