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Neosporosis and toxoplasmosis-associated paralysis in dogs in Costa Rica

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Summary

Clinical neosporosis and toxoplasmosis are reported in 2 separate adult dogs from Costa Rica.

Introduction

Neospora caninum is a recently recognized parasite of livestock and companion animals. Until 1988, it was misdiagnosed as *Toxoplasma gondii* (DUBEY et al. 1988; BJERKÅS & DUBEY 1991; DUBEY 1992; DUBEY & LINDSAY 1993). Since it was first recognized in Norway (BJERKÅS et al. 1984), the disease has been found to cause paralysis in dogs in several continents including Europe, Africa, Australasia, North America and Asia (DUBEY 1992). The objective of this paper is to document fatal neosporosis and toxoplasmosis in dogs from Costa Rica.

Material and methods

Dog 1: A 2 year-old female cocker spaniel was submitted for diagnosis in April 1990 to the Department of Veterinary Pathology, School Veterinary Medicine, National University, Heredia, Costa Rica. The dog had a 6 week history of head-tilt. Head and neck movements were reported as irregular; circling, incoordination of the forelimbs, and occasional seizures were also noted. The dog was initially diagnosed as having otitis media and was euthanatized because treatment with antibiotics was not effective. A completely necropsy was performed.
Dog 2: A 6 year-old mixed breed male was initially presented to the veterinary clinic because of hindlimb weakness, incoordination, and pain in the lumbar region. Initial diagnosis was nephritis but the dog showed no improvement with antibiotics and corticosteroid therapy. The dog became paralyzed in the hindlimbs and was euthanatized 3 weeks from the time the hindlimb problem was detected. A complete necropsy was performed.

Tissues were fixed in 10% buffered formalin, and paraffin – embedded sections were stained with hematoxylin and eosin. Retrospectively, paraffin sections were reacted with antibodies to *T. gondii* and *N. caninum* using reagents and techniques described by LINDSAY & DUBEY (1989), and COLE et al. (1993). Samples of formalin-fixed brain from dog 1 were post-fixed in osmium tetroxide and routinely processed for transmission electron microscopy.

Results

- Dog 1

An approximately 1 cm mass with a smooth surface projected through the meninges on the right side of the cerebellum of dog 1 (Fig. 1). On cut section this mass consisted of a yellowish-tan area, approximately 1.5 cm in diameter (Fig. 2). The lungs showed a few small pale foci. No lesion was identified in the ears.
On histologic examination of the nervous system there was a nonsuppurative meningoencephalitis involving the cerebral cortex, hippocampus, thalamus, and pons, characterized by multifocal gliosis and perivascular infiltration by mononuclear cells. The most severe lesion was located in the cerebellar cortex and adjacent white matter and consisted of granulomatous, eosinophilic meningoencephalitis with extensive areas of necrosis, perivascular cuffing, and neovascularization (Fig. 3). Inflammatory cells formed variably thick perivascular cuffs and extended into the necrotic parenchyma where macrophages, eosinophils and reactive astroglia were abundant.
Numerous protozoal tachyzoites were present, particularly in necrotic areas and in the walls of blood vessels (Fig. 4). A few tissue cysts with 1 to 2 µm thick walls were seen, particularly towards the periphery of the necrotic area (Fig. 5). Nume-

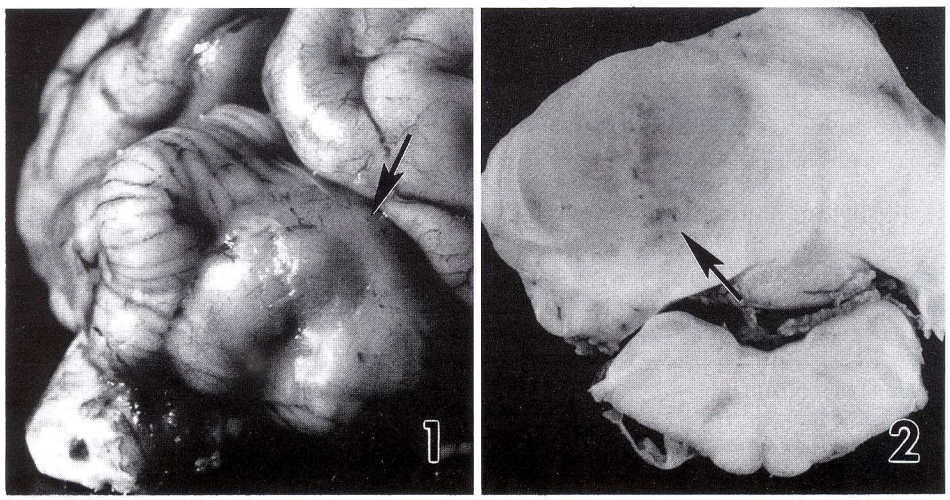

Figure 1. A grossly visible mass (arrow) on the right side of the cerebellum of dog 1.
Figure 2. Cut section of the cerebellar mass shown in Fig. 1. Note dark area of malacia (arrow).

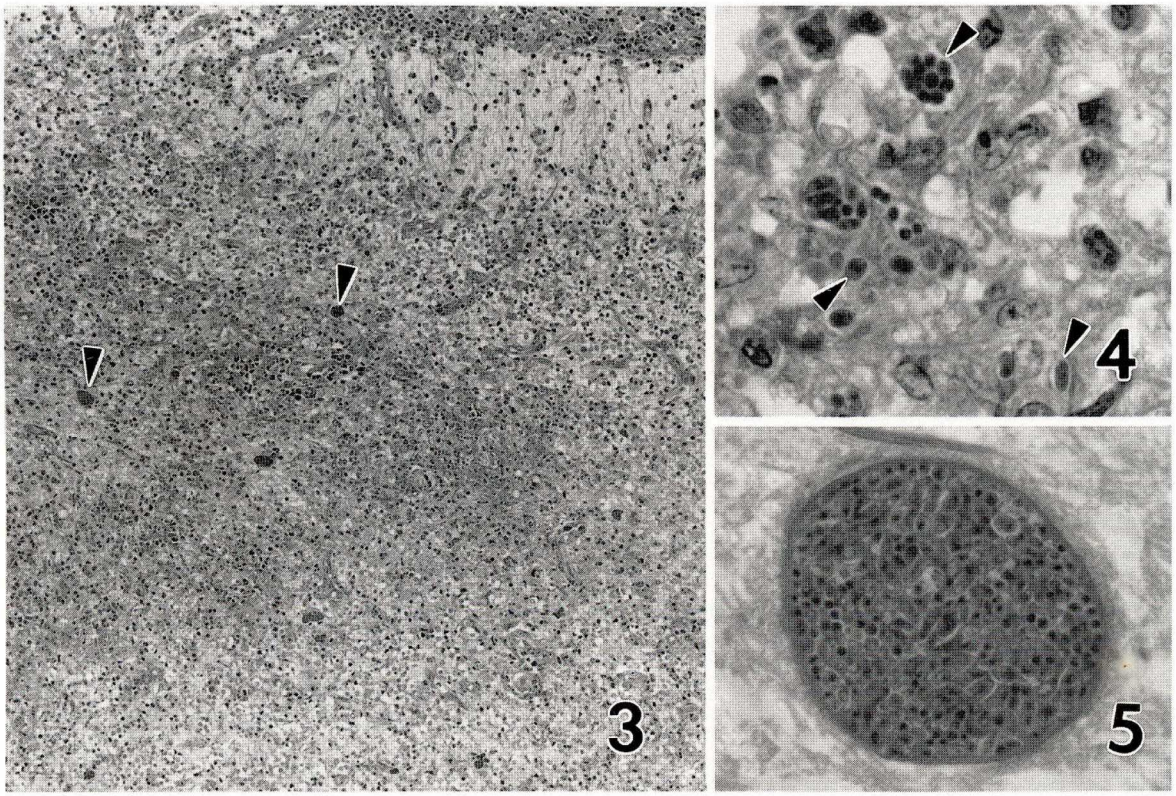

Figure 3. Necrosis, and meningitis in the cerebellum of dog 1. Arrowheads point to large collections of *N. caninum* tachyzoites. Hematoxylin and eosin stain. ×75.
Figure 4. Higher magnification of the lesion in Fig. 3. Note individual and groups of *N. caninum* tachyzoites (arrowheads). Hematoxylin and eosin stain. ×750.
Figure 5. A *N. caninum* tissue cyst in cerebellum of dog 1. Hematoxylin and eosin stain. ×750.

rous tachyzoites were visible in sections stained with *N. caninum* polyclonal and monoclonal antibodies (Fig. 6). Parasites did not react with *T. gondii* antibodies.
Ultrastructurally, tachyzoites contained electron dense rhoptries and micronomes arranged perpendicular to the plasmalemma and were structurally identical with *N. caninum* tachyzoites (Fig. 7).
The spinal cord appeared normal. A minute focus of necrosis and mononuclear cell infiltration was present in the lungs, but parasites were not found.

• Dog 2

Except for a few *Dipylidium caninum* in the small intestine, there were no gross lesions. Microscopically, severe lesions were present in the cerebrum, cerebellum and the spinal cord. In the brain, multiple foci of gliosis and perivascular cuffing were present in the cortex, thalamus and the pons.
Lesions in the cerebellum consisted of nonsuppurative meningitis, necrosis of the grey matter and adjacent white matter, gliosis, perivascular cuffs and infiltration of lymphocytes, plasma cells and macrophages into the neuropil. Lesions in the spinal cord were more severe than the brain and were most pronounced in the thoracic and lumbar areas. Changes consisted of nonsuppurative meningitis with massive histiocytosis. In the gray matter there were perivascular cuffs, gliosis,

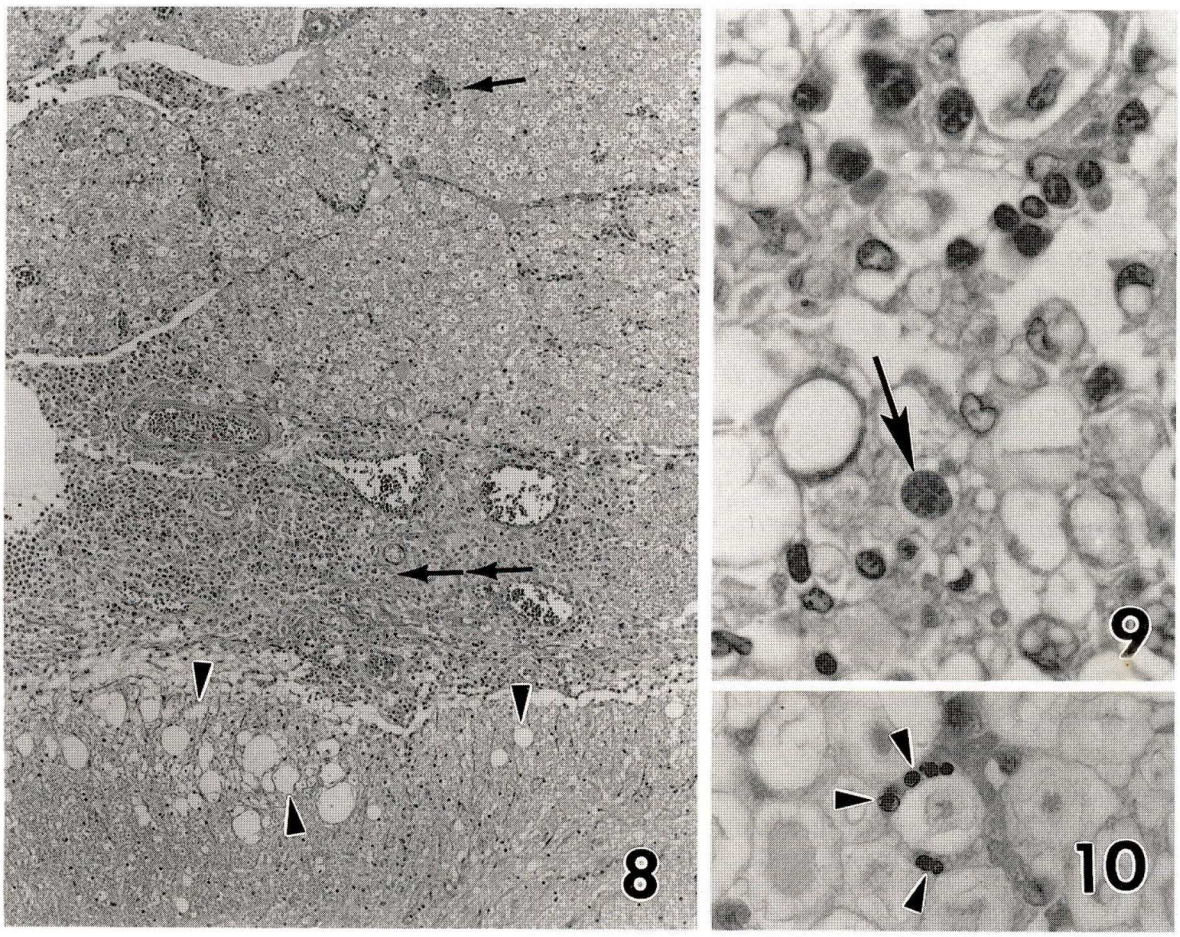

Figure 8. Section of spinal cord of dog 2 showing radiculitis (upper half, arrow), meningitis (middle, double arrow) and necrosis and vacuolization of dorsal funiculi (lower third, arrowheads). Hematoxylin and eosin stain. ×150.

Figure 9. Higher magnification of Fig. 8 showing necrosis of a spinal nerve, infiltration of mononuclear cells, and a small *T. gondii* tissue cyst (arrow). Hematoxylin and eosin stain. ×750.

Figure 10. Section of spinal cord from dog 2. Note a ring of 6 *T. gondii* tachyzoites (arrowheads) around a degenerating neuron. Immunohistochemical reaction with polyclonal *T. gondii* serum. ×750.

toxoplasmosis were reported in the 1950's and 1960's (DUBEY 1985). Most cases of clinical canine toxoplasmosis occur in young dogs and often in association with distemper virus infection and often involve lung, liver and the brain (DUBEY & BEATTIE 1988). A syndrome of protozoan polyradiculoneuritis resulting in hindlimb paralysis was thought to be due to *T. gondii* until the discovery of *N. caninum.* Unlike toxoplasmosis, neosporosis is considered to be a primary pathogen of dogs and the predominant feature of neosporosis in dogs is hindlimb paralysis.

Although there are numerous reports of neosporosis and toxoplasmosis in dogs from many countries, there are special features of the cases from Costa Rica presented here. This is the first documented report of toxoplasmosis polyradiculoneuritis in a dog after the discovery of *N. caninum* in 1988. Therefore, the diagnosis

of canine neosporosis should not be made until toxoplasmosis has been excluded. Because *T. gondii* and *N. caninum* tachyzoites are structurally similar and have some common antigens, caution is needed in interpreting testing serologic results on serum and immunohistochemistry. Although an indirect fluorescent antibody test titer of 1:200 is indicative of *N. caninum* infection, dual infection with *N. caninum* and *T. gondii* does occur (LINDSAY et al. 1990). Occasionally there is cross reaction between *T. gondii* and *N. caninum* in tissues using polyclonal sera (DUBEY et al. 1990). However, one monoclonal antibody against *N. caninum* does not react with *T. gondii* (COLE et al. 1993). In the present report, protozoa in the spinal cord of dog 2 reacted with polyclonal *T. gondii* but not with monoclonal antibody to *N. caninum*.

The dogs in both cases of neosporosis and toxoplasmosis in the present report were adults. As stated earlier, most cases of neosporosis and toxoplasmosis have been seen in young dogs.

This is the first report of neosporosis in dogs from Central America and the first report of toxoplasmosis in dogs from Costa Rica.

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