Cerebral cysticercosis by *Taenia crassiceps* in a domestic cat

Arno Wünschmann, Virginia Garlie, Gary Averbeck, Harold Kurtz, Eric P. Hoberg

Abstract. Cerebral cysticercosis by *Taenia crassiceps* was diagnosed in an adult female domestic shorthair cat. The animal was euthanized 6 weeks after the initial presentation with signs of vomiting, lethargy, and ataxia. The disease took an intermittent relapsing course with the neurological signs progressing eventually to recumbency and coma. At necropsy, numerous cysticerci were found in the dilated left lateral ventricle and the adjacent brain parenchyma. The cysticerci were identified as metacestodes of *T. crassiceps* larvae based on size and morphology of the cysts; shape, number, and size of the rostellar hooks; and mode of proliferation, including endogenous and exogenous budding. Cerebral cysticercosis by *T. crassiceps* is rare in atypical intermediate hosts and has not been described in cats.

A 7-year-old, spayed female, domestic shorthair cat was presented to the referring veterinarian in June 2001 for sudden onset of anorexia, ataxia and lethargy, and an increasing frequency of vomiting. The cat weighed approximately 4.0 kg. It was primarily kept indoors but was allowed outdoors in the garden where it occasionally caught mice. The animal had had intermittent vomiting in the past and was therefore fed a bland diet and had been dewormed with praziquantel and parantel pamoate (5 and 20 mg/kg body weight/12 hours), respectively, even though parasites had not been found on previous fecal flotation analyses. On physical examination, a marked periodontitis was noted. The cat was mildly hypothermic (37.7°C) and was slightly dehydrated (approximately 5%). The animal had a low leukocyte count (4,000 leukocytes/ml; reference range: 4,000–16,000) and a low serum potassium concentration (3.5 mmol/liter; reference range: 3.5–5.8 mmol/liter). The cat was treated with subcutaneous lactated Ringers solution supplemented with KCl (20 mEq KCl/liter) and clindamycin (10 mg/kg body weight/12 hours) twice a day. Two days later, the owner reported that the cat was circling and disoriented. Although the butorphanol treatment was discontinued, the neurological signs worsened with additional progressing ataxia and weakness within the next few days. The animal tolerated overcrossing of the front and hind limbs without correcting reaction. Lactated Ringers solution and vitamin B complex led to an improvement of the cat’s condition within a week. However, 10 days later, there was a dramatic decline of the cat’s condition with recurring vomiting, anorexia, lethargy, disorientation, and ataxia. The animal became comatose. Serum chemistry demonstrated a hypokalemia (2.96 mmol/liter; reference range: 3.5–5.8 mmol/liter). Repeated intravenous infusions of KCl-supplemented lactated Ringers solution led to a temporary improvement with normalization of its behavior. However, the cat deteriorated rapidly and became comatose a second time within a few days. The owners requested euthanasia approximately 6 weeks after the initial presentation with neurological signs. The cat was submitted for necropsy to the Veterinary Diagnostic Laboratory of the University of Minnesota.

At necropsy, pathological findings were restricted to the central nervous system of which the brain was examined but not the spinal cord. Approximately 20–25 thin-walled, ovoid to tear-shaped cystic structures, ranging in length from 1 to 10 mm, were found freely floating within or attached to the surface of the left lateral ventricle of the cerebrum (Fig. 1). These structures were interpreted as viable and potentially infective cysticerci because they were intact and did not show any evidence of calcification. The brain parenchyma adjacent to the left lateral ventricle had cavitations. The left lateral ventricle was moderately dilated. The periventricular white matter had areas of malacia. Samples of various tissues, including brain, heart, liver, kidney, lungs, thyroid gland, parathyroid gland, adrenal glands, stomach, and pancreas were fixed in 10% buffered formalin. The fixed tissues were trimmed, dehydrated, embedded in paraffin, and sectioned according to routine procedures. The 4–5 μm tissue sections were stained with hematoxylin and eosin (HE). Histologically, a moderate to marked lymphoplasmacytic and histiocytic encephalitis was present in the periventricular white matter and the sub-
cortical white matter neighboring the macroscopically visible cavitations. The left lateral and third ventricle were lined by a membranous layer of gemistocytic and fibrous astrocytes. This layer was multifocally markedly infiltrated by lymphocytes, plasma cells, and macrophages. The layer was approximately 200 μm thick and laid on the ependymal lining. The ependymal lining appeared to be multifocally fenestrated and had occasional rosette formation (Fig. 2). The intraventricular cysts and parenchymal cavitations represented discrete cysticerci that were not apparently continuous with a common membrane of parasite origin. The cysticerci were characterized by a wall, an internal cavity, and inverted scolexes (Fig. 3). The wall consisted of an outer dense narrow tegument layer with microtriches. A single layer of cuboidal to columnar cells was palisading the inner surface of the tegument. A loose meshwork of cells and their cell processes formed the inner lining of the internal cavity. Calcareous corpuscles were embedded into this meshwork, which became more dense at the base of the scoleces. Exogenous budding was occasionally present. Rostellar hooks were not detected in the histological sections. The cysticerci embedded in the brain parenchyma were surrounded by fibrous astrocytes similar to those found in the inner lining of the ventricles. Multiple formalin-fixed intraventricular cysticerci were collected, stained with Semichons acetic carmine, dehydrated in ethanol, cleared in xylene, and mounted entirely on Canadian balsam. Hooks from some specimens were mounted separately to allow detailed study of structure. The cysticerci ranged in length from 1 to 10 mm and in width from 1 to 4 mm, and each cysticercus contained clear colorless watery fluid with a single invaginated scolex, approximately 1 mm in diameter (Fig. 4). Endogenous and exogenous budding occurred at the polar end opposite the invaginated scolex. The scolices of a few larger cysts had rostellar hooks (Fig. 5) that were missing handles, whereas other cysts had fully developed hooks. One rostellum had 17 small hooks (117 ± 7.7 μm; range 104–127 μm) and 14 large hooks (141 ± 3.4 μm; range 138–146 μm). The hooks did not have handles. The cysticerci were identified as *Taenia crassiceps* based on morphology, mode of budding and structure, dimensions, and number of the rostellar hooks. Further, to confirm identity, cysticerci from this feline host were compared directly with those of *T. crassiceps* collected from an atypical canine host. Voucher specimens including intact cysticerci and mounts of rostellar hooks have been deposited in the US National Parasite Collection as USNPC No. 92801.

The life cycle of taeniid tapeworms involves a de-
finitive host and 1 intermediate host, both of which are always mammals. Adult tapeworms live in the intestine of the definitive hosts, usually carnivores including canids and felids, which pass gravid proglottids in their feces (taeniasis). The intermediate host, usually a herbivore such as a rodent, lagomorph, or ruminant, becomes infected by ingestion of the eggs. The first-stage larvae (oncospheres) migrate hematogenously to the organ of predilection. The metacestodes of taenid tapeworms develop in extraintestinal organ systems and are infective for the definitive host. They are classified according to structure; a bladder, containing a single scolex is called a cysticercus, whereas a bladder with multiple scolices is referred to as a coenurus. The definitive host becomes infected by the ingestion of musculature or visceral organs containing these infectious metacestodes; transmission is basically linked to predator–prey relationships. In rare cases, metacestodes may develop in carnivores that hereby become aberrant intermediate hosts. Adults and metacestodes are generally identified according to morphological or molecular criteria. Tapeworm cysts in the brain of domestic animals nowadays represent fairly rare findings in the USA and Western Europe because of improved sewage water processing, improved hygiene measures, and anthelmintic treatment of the definitive hosts. However, neurocysticercosis in humans, attributable to the host-specific Taenia solium, remains a considerable public health problem and represents the most common metazoan disease of the brain in humans worldwide. Infection of the brain with metacestodes has been rarely reported in dogs and cats. In these instances coenuri likely of Taenia serialis have been reported in the cats. In contrast, metacestodes in dogs have been reported as either of T. solium or Taenia pisiformis. Cases of neurocysticercosis attributable to other taeniid tapeworm species have not been described in carnivorous domestic or wild animals.

Strobilate adults of T. crassiceps, widely distributed in the Holarctic, reside usually in the intestine of canid definitive hosts. Only rarely are adults of T. crassiceps found in the intestine of felids. The cysticerci of T. crassiceps are usually found in the musculature, subcutaneous tissue, as well as peritoneal and pleural cavities of a wide range of rodent intermediate hosts. Cys-
cysticerci of *T. crassiceps* may be encountered in the brain of mice and may occasionally cause circling disease.\textsuperscript{7,23} Cysticerci of *T. crassiceps* were found in the lungs, the peritoneal cavity, and the pleural cavity of one dog.\textsuperscript{14} In another case of canine *T. crassiceps* cysticercosis, the cysticerci were restricted to the subcutis.\textsuperscript{6} Cysticerci of *T. crassiceps* have not been described in cats.

It is unknown how long the cat had been infected. The cat may have become infected by ingesting eggs that were excreted by infected definitive hosts such as foxes or dogs. Alternatively, the cat may have eaten an infected intermediate host. The detection of cysticerci with endogenous and exogenous budding and different stages of hook development suggests that at least 2 generations of cysticerci were present in the cat brain, indicating a longer lasting infection. *Taenia crassiceps* is unique among the taeniids in that it has proliferative cysticerci that develop asexually by endogenous and exogenous budding.\textsuperscript{9} Theoretically, the cysticerci present in the brain of this cat may have been derived from only one oncosphere. Experimental infection of mice by ingestion of eggs results in circling disease caused by intracranial cysticerci at 31 days postinfection.\textsuperscript{23}

Unfortunately, the immune status of the cat is unknown. The cat was vaccinated for feline leukemia virus but was not tested for feline leukemia virus or feline immunodeficiency virus antigen. The therapy-resistant, long-standing periodontitis may indicate some degree of immunosuppression. The mouse model of *T. crassiceps* cysticercosis indicates that CD4+ T cell receptor (TCR) alpha/beta T cells may be critically involved in the control of the disease.\textsuperscript{19} Further evidence that immunosuppression caused by decreased counts of CD4+ T cells predisposes aberrant hosts to infection comes from the observation that *T. crassiceps* cysticercosis is occasionally seen in AIDS patients. In humans, cysticerci of *T. crassiceps* are occasionally found within the eye globe, but the cysticerci also may be found in intramuscular sites.\textsuperscript{1,3,8,10,17}

The neuropathological changes accompanying the intraventricular and parenchymal manifestation of the cysticerci were characterized in the present case by an inflammatory reaction, fibrillary and gemistocytic astrocitosis, and the formation of an astrocytic lining of the ventricles that was superimposed onto the ependyma. The inflammatory reaction was fairly severe, considering that the cysticerci appeared to be viable. Necrotic or calcified cysticerci are more likely to elicit a significant inflammatory reaction than are viable cysticerci, which may be undetected by the host immune system for a long time.\textsuperscript{22} The formation of an astrocytic lining of the ventricle is referred to as granular ependymitis in the human literature and may enhance the adherence of the parasite to the ventricular surface.\textsuperscript{22}

Neurocysticercosis is a rare disease in domestic companion animals but may be considered as a differential diagnosis in patients with a protracted disease characterized by intermittent, relapsing neurological signs. *Taenia crassiceps* constitutes a zoonotic risk especially for immunocompromised humans. Intravitalm diagnosis of neurocysticercosis requires neuroimaging and cytological and immunological analysis of the cerebrospinal fluid.\textsuperscript{22} Immunological tests for human neurocysticercosis by *T. solium* have been performed with antigen derived from *T. crassiceps* cysticerci because *T. solium* cysticerci and *T. crassiceps* cysticerci share antigens.\textsuperscript{29} Increasingly, morphological and molecular criteria should be combined in establishing the identity of metacestodes in cases of cysticercosis.\textsuperscript{13}

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Sources and manufacturers

a. Hills\textsuperscript{8} prescription diet I/D, Topeka, KS.

b. Drontal\textsuperscript{8}, Bayer AG, Mohnheim, Germany.

c. Antirobe Aquadrops\textsuperscript{8}, Pharmacia Upjohn, Kalamazoo, MI.

d. Torbugesic\textsuperscript{6}, Fort Dodge, Animal Health, Ames, IA.

References


Nested multiplex RT-PCR for detection and differentiation of West Nile virus and eastern equine encephalomyelitis virus in brain tissues

Donna J. Johnson, Eileen N. Oslund, Beverly J. Schmitt

Abstract. A traditional nested reverse transcription-polymerase chain reaction (RT-PCR) assay specific for eastern equine encephalomyelitis (EEE) virus was designed to multiplex with a previously described West Nile (WN) virus nested RT-PCR assay. Differentiation of EEE and WN was based on base pair size of the amplified product. One hundred fifty-seven mammalian and avian brain tissues were tested by EEE/WN nested multiplex RT-PCR, EEE nested RT-PCR, and WN nested RT-PCR, and results were compared with other diagnostic test results from the same animals. Serological and virus isolation testing confirmed the results of the multiplex PCR assay. When compared with cell culture virus isolation, the multiplex assay was shown to be more sensitive in detecting the presence of EEE or WN virus in brain tissues. The multiplex assay was shown to be sensitive and specific for North American EEE and WN and provided a rapid means of identifying both viruses in brain tissues. No apparent sacrifice in sensitivity was observed in the multiplex procedure compared with the individual EEE and WN nested RT-PCR assays. Data collected from an additional 485 multiplex RT-PCR tests conducted during the summer and fall of 2002 further support the validity of the procedure.

The number of horses and birds with clinical signs of encephalitis has increased in the past 4 years because of the emergence of the West Nile (WN) virus in the Western hemisphere. By the end of year 2001, the WN virus was identified in most states in the eastern half of the United States. The eastern equine encephalomyelitis (EEE) virus is endemic in the same region. Nearly 800 equine clinical cases of either EEE or WN were identified by testing conducted at the National Veterinary Services Laboratories (NVSL), Ames, Iowa, in 2001. In 2002, nearly 15,000 equine clinical cases of EEE or WN were diagnosed nationally. Animal exposure to either disease can be determined by serological testing performed on acute-phase serum or cerebrospinal fluid (CSF). An equine immunoglobulin M–capture enzyme-linked immunosorbent assay (MAC-ELISA) specific for either EEE or WN supports recent exposure of equids to either disease. Viral infection can be confirmed by isolation of virus from animal tissues; virus isolation testing can require 3 weeks. Alternatively, the presence of viral nucleic acid can be determined by reverse transcription-polymerase chain reaction (RT-PCR) testing in one day. Because EEE and WN are zoonotic infections