# Cerebral cysticercosis in a cat

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### **ABSTRACT**

The metacestode of *Taenia solium, Cysticercus cellulosae*, was recovered from the brain of a cat showing central nervous clinical signs *ante mortem*. This is the first record of cerebral cysticercosis in a cat in South Africa.

**Key words**: cat, cerebral cysticercosis, *Cysticercus cellulosae*, neurocysticercosis, praziquantel, South Africa, *Taenia solium*.

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## INTRODUCTION

Cerebral cysticercosis, also known as neurocysticercosis, caused by the larval stage (metacestode) Cysticercus cellulosae of Taenia solium (Cyclophyllida: Taeniidae), has been identified as a major public health problem in African populations<sup>13</sup>. T. solium has an indirect life-cycle. With the extremely rare exception (experimental infection only) of the white-handed gibbon (Hylobates (Hylobates) lar), the only final host of *T. solium* is man who contracts the infection by ingesting pork containing cysticerci<sup>5</sup>. Heteroinfection or autoinfection of man by ingesting eggs of T. solium will lead to the development of cysticerci in nearly every organ and tissue of the body, most commonly in the subcutaneous tissues and muscles of the tongue, neck or ribs, next in the eye and then in the brain<sup>11</sup>. Besides its normal intermediate host, the domestic pig, the metacestode of T. solium has been reported from a wide range of terrestrial<sup>1</sup> as well as a few marine mammals<sup>7</sup>. Apart from man, cases of cerebral cysticercosis have been reported in both domestic dogs14,15 and to a lesser extent in cats6,12. Indiscriminate defaecation by humans, observed in both urban and rural areas of South Africa, exposes wandering dogs and cats to infection with C. cellulosae. This report describes the first case of *C*. cellulosae infection in a cat in South Africa.

# **CASE HISTORY**

A 10-month-old, neutered male domestic short-hair cat was presented to Radiokop Animal Clinic in Johannesburg, South Africa, for acute onset of ataxia which resolved after flunixin (Finadyne, Schering-Plough Animal Health) at 1 mg/kg intramuscularly and enrofloxacin (Baytril 5 % injectable solution, Bayer Animal Health Division) at 5 mg/kg subcutaneously were given.

Three months later the cat had a relapse starting with nausea, salivation and vomition, 6 hours after vaccination against feline rhinotracheitis, panleukopaenia, calici virus infection (Felocell CVR, Pfizer Animal Health) and rabies (Quantum R, Schering-Plough Animal Health) as well as simultaneous deworming with a combination of praziquantel and pyrantel pamoate (Drontal cat tablets, Bayer Animal Health Division) at the dose recommended by the manufacturer. The cat was treated with 1 mg atropine (Atropine 0.5 injection, Centaur) given intravenously and a combination of procaine penicillin and benzathine benzylpenicillin (Duplocillin, Intervet SA) at 26.5 mg/kg intramuscularly. The following day, fluids were administered subcutaneously as well as enrofloxacin (Baytril 5 % injectable solution, Bayer Animal Health Division) at 5 mg/kg subcutaneously as well as 0.4 mg dexamethasone (Dexa 0.2 % Phenix, Logos Agvet) subcutaneously. Convulsions started that were treated with 5 mg diazepam (Valium, Roche). The convulsions progressed to nystagmus. Routine biochemistry revealed raised alanine aminotransferase (116 U/l), mild hypercalaemia (5.4 mmol/ $\ell$ ), low urea (5.4 mmol/ $\ell$ ), hyperalbuminaemia (41 g/l) and hyperglycaemia (9 mmol/l) (Table 1). The haematology results showed a slight increase in haemoglobin (14.6 g/dl), low platelet count (151  $000 \times 10^3 \ell$ ) and eosinopaenia (0/l) (Table 2). Faecal flotation as well as serological tests for feline immunodeficiency virus and feline leukaemia virus were negative.

On the following day the cat was referred to Bryanston Veterinary Hospital, Johannesburg. Clinical presentation consisted of diffuse central nervous signs including collapse, convulsions, falling to the left, anisocora with unresponsive pupils, blindness, left eye more mydriatic than right, lateral and verticle nystagmus (varied with position), rigid forelimbs and hyper-reflexic hind limbs. Temperature, pulse and respiration were normal. Urine analysis revealed a specific gravity of 1.030, pH 8 and a glucose trace. The urine sediment showed some debris. A basal ammonia reading was zero. Hepatic ultrasound and fine-needle aspirate revealed mild hepatic lipidosis. Cisternal puncture did not yield any cerebrospinal fluid (CSF). Initial treatment included

Table 1: Serum biochemistry results for the cat.

Parameter	Results	Reference range
ALT (U/ℓ)	116	5–30
ALP (U/ℓ)	58	100–150
Urea (mmol/ℓ)	5.4	7.1–10.7
Creatinine (µmol/ℓ)	93	80–140
Total protein (g/l)	75	54–72
Albumin (g/ℓ)	41	20–30
Globulin (g/ℓ)	34	25–38
Lipase (U/ℓ)	228	138–625
Glucose (mmol/ℓ)	9	3.8-6.5

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Table 2: Haematology results for the cat.

Parameter	Results	Reference range
RBC (×10 <sup>12</sup> /ℓ)	8.96	5.5–10
Haemoglobin (g/dl)	14.6	8–14
Haematocrit (%)	42	30–45
MCV (fl)	47	39-55
MCHC (%)	35	30–36
Platelets (×10 <sup>9</sup> /ℓ)	151	300-600
WBC (×10 <sup>9</sup> /ℓ)	7.4	7–20
Neutrophils (mat abs) (×10 <sup>9</sup> /ℓ)	5.25	2.5-12.5
Neutrophils (immat abs) (×109/ℓ)	0.07	0-0.3
Lymphocytes (abs) (×10 <sup>9</sup> /ℓ)	1.7	1.5–7
Monocytes (abs) (×10 <sup>9</sup> /ℓ)	0.37	0-0.8
Eosinophils (abs) (×10 <sup>9</sup> /ℓ)	0	0.1-1.5
Basophils (abs) (×10 <sup>9</sup> /ℓ)	0	0–1

RBC, red blood cell count; MCV, mean corpuscular volume; MCHC, mean corpuscular haemoglobin concentration; WBC, white blood cell count.

enrofloxacin at 5 mg/kg (Baytril 5 % injectable solution, Bayer Animal Health Division) subcutaneously, prednisolone (Prednisolone 1 % KELA, Centaur) at 2 mg/kg subcutaneously and vitamin B1 at a total dose of 50 mg/kg intramuscularly. Euthanasia was elected as the nervous signs were progressive.

Necropsy revealed a portion of the cerebellum prolapsing through the foramen magnum, thus accounting for the absence of CSF. On opening the meninges, CSF welled up, indicating increased intracranial pressure, possibly contributing to the cerebral prolapse. The foramen magnum did not appear abnormally shaped, nor the basal bone shortened. Multiple, spherical, 0.5 cm in diameter, thin-walled, semi-transparent, fluid-containing cysts with a single invaginated scolex were seen throughout the cerebral hemispheres (Fig. 1). The cysts were identified as cysticerci. The number of hooks on scoleces (n = 4) was 27. The large hooks (n = 40) varied in

length from 166 to 192  $\mu$ m whereas the small hooks (n = 40) varied in length from 114 to 137  $\mu$ m. The number and size of the hooks fall in the range of the figures given by Verster<sup>19</sup> for *Cysticercus cellulosae*. The owner found the cat as a stray. Other cats in the household were asymptomatic.

## **DISCUSSION**

Apart from a six-year old dog<sup>4</sup>, cerebral cysticercosis (neurocysticercosis) caused by *Taenia solium* has never before been recorded from a domestic cat in South Africa. Cysticercosis is the most common parasitosis affecting the central nervous system in man<sup>16</sup>. The disorder produces a complex neurological picture with many degrees of severity depending on topography and the number of lesions. In the brain, cysts may be parenchymal, meningeal or ventricular<sup>8</sup>. Secondary to the parasitic infestation is an inflammatory reaction that produces additional complications and in many instances causes

Fig. 1: Invaginated scolex of Cysticercus cellulosae.

more neurological disturbances than does the parasite itself<sup>16</sup>. Dying cysts provoke acute inflammation and tissue damage. The most common manifestation of cerebral cysticercosis is epilepsy caused by parenchymatous cysts<sup>3</sup>. Meningeal cysts in the basal meninges excite an intense inflammatory response, and may cause obstructive hydrocephalus. Ventricular cysts may float about blocking the aqueduct of Silvius, causing intermittent hydrocephalus with severe headache and vomiting<sup>11</sup>.

Clinical signs described from affected cats range from hypersensitivity, progressive ataxia, ambulatory incoordination to paralysis <sup>6,12</sup>.

The latest episode of nervous clinical signs in the present case appears to have started 6 hours following vaccination and deworming with Drontal cat tablets (Bayer Animal Health Division). Praziquantel, one of the active ingredients in Drontal cat tablets, is a cestocidal drug, active in a single oral, subcutaneous or intramuscular dose against a broad range of juvenile and adult tapeworms in the gut and bile ducts as well as flukes of animals and humans 2,20. In addition, praziquantel is effective against the cysticerci of several Taenia species, including Cysticercus cellulosae of T. solium, and is therefore widely used as the drug of choice in the treatment of human neurocysticercosis. However, the reaction caused around the dying cyst can lead to severe complications such as acute obstructive hydrocephalus<sup>3</sup>. For this reason, praziquantel must only be used in hospital inpatients under careful expert neurological supervision. In the present case the onset and aggravation of the neurological clinical signs were most probably caused by the administration of praziquantel. Although the recommended dose of 5 mg/kg praziquantel for the treatment of intestinal tapeworm infection in animals and man is only a tenth of the one usually recommended for human neurocysticercosis, adverse reactions have been recorded in a human population in Mexico shortly after receiving mass treatment for intestinal taeniasis; neurocysticercosis was confirmed in all cases9.

*T. solium* is endemic in South Africa. Although some isolated studies were conducted in South Africa, the prevalence and incidence of *T. solium* infection and neurocysticercosis in humans are unknown<sup>10,13,18</sup>. As a typical anthropozoonosis, involving humans as the only final host, control has to focus on humans by identifying and treating human carriers, disposing properly of human faeces and preventing human infection<sup>17</sup>.

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