
Keywords: *Acinonyx jubatus/captivity/cheetah/disease/Toxoplasma gondii/toxoplasmosis*

Abstract: A juvenile cheetah (*Acinonyx jubatus*) died with rapidly progressive pyrexia, tachypnea, abdominal effusion, and hepatomegaly. Postmortem examination revealed lesions consistent with acute disseminated infection with *Toxoplasma gondii*. The presence of this organism was confirmed in multiple organs by immunohistochemistry and polymerase chain reaction. To the best of our knowledge, we propose this to be the first reported case of primary acute disseminated toxoplasmosis in a cheetah.
RH: DISSEMINATED TOXOPLASMOsis in A CHEETAH

ACUTE DISSEMINATED TOXOPLASMOSIS IN A JUVENILE CHEETAH

(ACINONYX JUBATUS)

Christopher Lloyd BVSc., MSc., CertZooMed, MRCVS, and Mark F. Stidworthy, MA, VetMB, PhD, MRCPath., MRCVS

From the Nad Al Shiba Veterinary Hospital, PO Box 116345, Dubai, United Arab Emirates. (Lloyd);and the International Zoo Veterinary Group, Keighley Business Centre, South Street, Keighley, West Yorkshire, BD 21 1AG, United Kingdom (Stidworthy)

Address all correspondence to
Christopher Lloyd BVSc MSc CertZooMed MRCVS
Nad Al Shiba Veterinary Hospital, PO Box 116345, Dubai, United Arab Emirates
+971 4 3401060

chris@nadvethosp.com
**Abstract:** A juvenile cheetah (*Acinonyx jubatus*) died with rapidly progressive pyrexia, tachypnea, abdominal effusion and hepatomegaly. Post mortem examination revealed lesions consistent with acute disseminated infection with *Toxoplasma gondii*. The presence of this organism was confirmed in multiple organs by immunohistochemistry and PCR. The authors believe this to be the first reported case of primary acute disseminated toxoplasmosis in a cheetah.

**Key words:** *Acinonyx jubatus*, acute disseminated toxoplasmosis, cheetah, *Toxoplasma gondii*.
Brief Communication

A privately owned 16-wk-old cheetah cub (Acinonyx jubatus) was referred to Nad Al Shiba Veterinary Hospital (Dubai, United Arab Emirates) for examination. The cub had been in the owner’s possession for 3 wks and was suspected to have been wild caught. Diet consisted of unsupplemented beef fillet and quail. The cheetah was kept with three adult domesticated cats that had been vaccinated against feline herpes, calicivirus, and panleukopaenia.

The cheetah presented with a history of weakness and anorexia of 2 days duration. Body weight was 3.65 kg. The animal was pyrexic and tachypnic with a body temperature of 39.9 °C and respiratory rate of 70 breaths per minute. The mucus membranes were pale and mildly icteric, and the abdomen was swollen and tender with palpable hepatomegaly. Auscultation of the cardiovascular system and capillary refill time were unremarkable. The animal was estimated to be 5% dehydrated and the referring veterinarian had administered 500ml intravenous lactated Ringers solution and ceftriaxone. Prednisolone and metronidazole had been given by mouth. No clinical signs had been reported by the referring veterinarian 7 days earlier at vaccination when body weight was 4 kg. Blood taken at this time had revealed elevated alanine aminotransferase (ALT), aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) but the cause was not investigated.

Following admission to the hospital, the cheetah was premedicated with intravenous diazepam (Valium, Roche, 1mg/kg, i/v) and general anaesthesia induced with a 4% isoflurane (“Attane”, Minrad Inc) and oxygen mixture by facemask.
Radiology revealed a swollen abdomen, hepatomegaly, loss of definition of abdominal contents with a ground glass appearance and pulmonary edema. The main haematology and biochemistry abnormalities from blood drawn at this time were as follows: a mild hypoproteinaemia with an elevated AST of 4709 IU/L (normal range, 18-84 IU/L), ALT of 2157 IU/L (normal range 27-169 IU/L), LDH of 944 IU/L (normal range, 5-175 IU/L) and creatine kinase (CK) of 2154 IU/L (normal range, 15-532 IU/L). There was a leucocytosis (14500/µl; normal 6.85-13.84/µl) with a neutrophilia and lymphopenia. A mild regenerative anemia was present with a moderate thrombocytopenia (108/µl; normal 224-486/µl). Blood tested for FeLV/FIV antibodies (Snap Test kit – Idexx Laboratories Inc) was negative. A fecal sample examined by floatation was unremarkable.

A peritoneal tap taken under anesthesia failed to draw significant amounts of fluid from the abdomen. A nasoesophageal (NO) tube was placed while the animal was under anesthesia for continued supportive treatment. The cheetah was placed on enrofloxacin (Baytril 2.5% injection: Bayer, UK; 5mg/Kg, s/c, q 12hr), ceftriaxone (Rocephin, Roche, Switzerland 1.8 ml, i/v, q 12hr) metronidazole (Flagyl-S paediatric suspension, Rhone – Poulenc Rorer; UK, 30mg, p.o., q 12hr), lactulose (Duphalac, Solvay Pharmaceuticals, Netherlands, 670mg, p.o, q 12hr), and vitamin K (Konakion, Roche, UK, 5mg, s/c, q 24hr). The animal was fed up to 80ml of liquid food (Royal Canin Convalescence Support, Austria) by NO tube every 4 hours and placed on maintenance intravenous fluids. Despite treatment, the PCV had dropped to 17% after 48 hours of treatment. The cheetah subsequently developed hemoptysis and died from suspected pulmonary hemorrhage later that day.
Gross lesions found during a postmortem examination revealed hepatomegaly, splenomegaly and pronounced edema and hemorrhagic congestion of all lung lobes. Bacteriological culture of the liver grew *Escherichia coli*. A range of tissues were collected in buffered 4% formalin and submitted for histopathology. Histological examination of tissues revealed acute multifocal necrotising myocarditis with acute multifocal necrosis also present in the pancreas and liver. In the lung, severe diffuse interstitial pneumonia was present with multifocal intra-alveolar hemorrhage; accumulations of protein-rich alveolar edema fluid (Figure 1a); pneumocyte hyperplasia; and interstitial infiltrates of macrophages a small numbers of lymphocytes. In the sections of myocardium, a small number of structures consistent with protozoal tachyzoites and small *Toxoplasma gondii* tissue cysts could be visualized in routinely-stained sections, but organisms were not readily visualized in the other tissues. Immunohistochemistry (performed by routine methods at the Moredun Research Institute, Edinburgh) using *T. gondii* specific antibodies revealed widespread positive labeling in multiple tissues including the heart, lung (Figure 1b), pancreas, liver and spleen. Single organisms were also identified in enterocytes in the intestinal mucosa, and in the epithelium of the urinary bladder. Sections of cerebellum and kidney did not reveal and organisms. In addition, a sample of frozen lung tissue submitted for PCR (MDS labs, Westville, South Africa) confirmed the presence of *T. gondii* nucleic acid. A frozen serum sample submitted to IDEXX Laboratories, Wetherby, UK for serological testing revealed a positive IgM titer of 20 and an IgG titer of 100. Titers greater than 50 are considered positive. Samples of lung, liver, pancreas and brain submitted for virus isolation (Central Veterinary Research Laboratory, Dubai, UAE) proved negative.
There are a number of reports of *T. gondii* exposure to cheetahs in both zoological \textsuperscript{14,15} and free ranging populations \textsuperscript{3}. While infection is common in both non-domestic and domestic felids, clinical disease is very rare \textsuperscript{7,15} There are few reported cases of acute disseminated toxoplasmosis in non-domestic felids with reports existing of two juvenile lions, *Panthera leo*, \textsuperscript{12} and one juvenile Siberian tiger, *Panthera tigris altaica*. \textsuperscript{4} The disease however, appears to be common in Pallas cats (*Otocolobus manul*) causing high mortality in neonates and kittens \textsuperscript{8,16}. There is one previous report of toxoplasmosis occurring in a juvenile cheetah with concurrent feline infectious peritonitis (FIP) \textsuperscript{17}.

The clinical and post mortem findings in this case were very similar to those from a retrospective case study of 100 domestic felids with clinical toxoplasmosis \textsuperscript{7} and also reports from Pallas cats\textsuperscript{13} Dyspnea, abdominal discomfort and hepatomegaly were common while interstitial pneumonia, myocarditis and hepatocyte necrosis were also frequent. In addition, the majority of domestic felids with fatal toxoplasmosis in this study were under 1 year old while this case and the majority of cases reported in non-domestic felids, especially Pallas cats, affected juvenile animals. \textsuperscript{4,12,16}

The ante mortem diagnosis of this disease appears to be challenging. Fecal shedding of oocysts in cats suffering from acute clinical toxoplasmosis is very rare\textsuperscript{7} and no oocysts were found in this case. Since interstitial pneumonia is a common feature of this disease, cytological\textsuperscript{7} examination together with PCR testing of lung or tracheal washes may be useful. Serological tests are validated for use in domestic cats but not the cheetah. There appears to be little information available on the optimal assay for serological use in non-domestic felids.\textsuperscript{11} However, in this case,
there was an elevated level of IgM which is only detected in the serum of clinically sick domestic cats during a period of active infection and does not last longer than 3 months post-infection\textsuperscript{7,11}. The authors believe that this is the first report of an elevated IgM titer in a non-domestic felid actively infected with \textit{Toxoplasma gondii}. This case indicates a need for further research into the use of this assay as an aid to diagnosing toxoplasmosis in actively infected non-domestic felids using a single blood sample. However, it is worth noting that IgM in domestic cats was only detectable within a post-inoculation period of 5 weeks so it is possible animals may die of acute infection before mounting a detectable immune response\textsuperscript{11}. Although IgG titers are commonly raised in exposed non-domestic felids, single samples do not aid in the diagnosis of active infection as IgG levels may be raised for months to years following exposure\textsuperscript{11}. The demonstration of a rising IgG titer (at least 4 fold) over a 2-3 week period may indicate recent or active infection in domestic cats\textsuperscript{11}.

Treatment of acute toxoplasmosis has proved largely unsuccessful\textsuperscript{7,16}. However, some zoological institutions have claimed success using clindamycin prophylactically for asymptomatic Pallas cat kittens considered to be at risk\textsuperscript{1}.

Domestic and exotic felids are the only known definitive hosts of \textit{T. gondii} with transmission via three routes: ingestion of feline fecal matter containing oocysts; transplacental infection; and ingestion of bradyzoites in infected meat\textsuperscript{6}. The latter route appears the most efficient in domestic cats\textsuperscript{5}. In addition to the route of infection, the host age, presence of concurrent infections, immunodeficiencies and immunological naïveté\textsuperscript{2} are all known to affect the clinical outcome of toxoplasmosis in domestic cats. The age of this animal at presentation suggests that infection
was probably acquired from contaminated meat consumed in captivity. It is possible, but less likely that the disease was caused by delayed development of *T. gondii* acquired in utero. Unfortunately this cheetah was highly stressed having been captured from the wild, transported illegally and likely housed with other domestic and non domestic animals before being sold as a household pet. It seems probable that this also contributed to the clinical outcome of this case.

**Acknowledgments:** The author would like to thank Dr David Buxton of the Moredun Research Institute for performing the immunohistochemistry, providing the photomicrographs used in Figures 3 and 4, and for review of the manuscript. The authors would also like to thank Ellen Kruyning of Al Basha Veterinary Hospital, Dubai for referring this case, Mia Jessen for her nursing care, and Dr Renata Padrtova of Nad Al Shiba Veterinary Hospital for her laboratory support. Thanks to An Pas for her review of this manuscript.

**LITERATURE CITED**


Received for publication 26 February 2007
**Figures**

**Figure 1**

Figure 1a. Edema and hemorrhage adjacent to a bronchiole within the lung. Interstitial inflammation in adjacent alveoli. H & E. Figure 1b. Immunohistochemistry using *T. gondii* specific antibodies demonstrating positive labeling of tachyzoites in cells within the alveolar septum of the lung. Images are derived from serial sections of the same piece of tissue.