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Fatal cytauxzoonosis in a Kentucky cat (*Felis domesticus*)

Carney B. Jackson *, Tina Fisher

Livestock Disease Diagnostic Center, University of Kentucky, 1490 Bull Lea Road, Box 14125, Lexington, KY 40512-4125, United States

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Abstract

A male domestic shorthair cat was presented with lethargy, anorexia, pyrexia and respiratory harshness. Diagnostic clinical chemistries, viral identification and tissue fluorescent antibody testing were negative or revealed no underlying system failure. Histopathology examination revealed multifocal, variable sized and shaped protozoal life forms in the brain, heart, lung, intestine, spleen, lymph node and kidney consistent with the intraleukocytic schizonts of *Cytauxzoon felis*.

Keywords: *Cytauxzoon*; *Dermacentor variabilis*; Intravascular schizonts; Feline; Protozoa

Cytauxzoon felis is in the order Piroplasmida and family Theileriidae (Allsopp et al., 1994). This family has erythrocytic and leukocytic or tissue phases in its life cycle. The leukocytic or tissue phase consists of large schizonts/meronts that develop in macrophages or monocytes (Cowell et al., 1988a,b) whereas *Theileria* schizonts are found in lymphocytes (MacWilliams, 1987). Classically schizonts are demonstrated in the pulmonary vessels, however, they may be observed in the vessels of any systemic tissue which containing cells of the mononuclear phagocyte system (Butt et al., 1990; Cowell et al., 1988a,b; Glenn and Stair, 1984; Hauck, 1982; Hoover et al., 1994). The only known tick vector of *C. felis* is *Dermacentor variabilis* (Cowell et al., 1988a,b; Hoover et al., 1994; Verdon, 2002).

After a tick blood meal, *C. felis* sporozoites enter intravascular macrophages or monocytes forming schizonts. Schizonts then bud forming merozoites resulting in cell enlargement. Schizont ladened leukocytes occlude vessels causing circulatory impairment resulting in tissue hypoxia (Kier et al., 1977, 1987; Meier and Moore, 2000; Verdon, 2002). The enlarged leukocytes subsequently rupture releasing merozoites which infect erythrocytes forming piroplasm ring stages. Piroplasms are detectable with parasitemias ranging from less than 1% to a high of 4% in blood smears but are difficult to detect at the beginning of the febrile response. After the fever has subsided and the cats are moribund, up to 25% of erythrocytes may contain piroplasms (MacWilliams, 1987). The erythrocytic phase causes signs of

* Corresponding author. Tel.: +1 859 253 0571x166; fax: +1 859 255 1624.
E-mail address: cjackson@uky.edu (C.B. Jackson).

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hemolytic anemia with moderate regenerative anemia and a mild increase in metarubricytes. Splenomegaly and hepatomegaly may also be observed (MacWilliams, 1987; Meier and Moore, 2000).

In late stages, cats become dyspneic, moribund and cry out with progression to death with the clinical course usually less than 1 week (MacWilliams, 1987). Mortality in cytauxzoonosis in treated and untreated cases approaches 100% in naturally infected cases, however, the majority of cases that survive have been treated with supportive care, antibiotics for secondary bacterial infection or antiprotozoal drugs such as parvaquone, buparvaquone, diminazene aceturate or imidocarb diproprionate (Hoover et al., 1994; Meier and Moore, 2000).

In the current study, a 1.5-year-old male neutered domestic shorthair cat was presented to the veterinary clinic. The cat was born and raised in eastern Pulaski County near Somerset in south central Kentucky approximately 35 miles from the Kentucky Tennessee border. The cat was 1 of 18 outdoor cats and exhibited clinical signs consisting of lethargy, anorexia, 105 °F temperature, respiratory harshness, signs consistent with cytauxzoonosis (MacWilliams, 1987). A sibling had previously died exhibiting the same clinical signs. Tests for feline leukemia virus and feline immunodeficiency virus were negative. Complete blood count (CBC), blood urea nitrogen (BUN), creatinine, alkaline phosphatase (ALP) and alanine aminotransferase (ALT) were unremarkable and within normal reference ranges. Blood smears were not examined for the presence of erythrocytic piroplasms. Despite antibiotic treatment with enrofloxacin and ampicillin and isotonic fluid therapy, the cat died. During the 12 h preceding death, the cat became very agitated and aggressive.

At necropsy, the cat presented in fair flesh and poor postmortem preservation. The only gross finding was dilation of the descending colon with a dark red serosa and dark red content. Nonpathogenic bacteria were isolated from cultures of the lung and intestines with no growth obtained from the liver. Tissues were negative for feline herpes virus, feline infectious peritonitis virus and panleukopenia virus by fluorescent antibody testing. No parasites were detected on a fecal flotation. Histopathology examination revealed multifocal, variable sized and shaped (up to 50–60 μm in diameter) protozoal schizonts in the brain, heart, lung, intestine, spleen, lymph node and kidney (Figs. 1–5). These intraleukocytic schizonts were present within blood vessels and were consistent with the asexual reproduction of *C. felis* within circulating monocytes or macrophages. The only other microscopic change observed was the presence of adult cestodes in the duodenum.

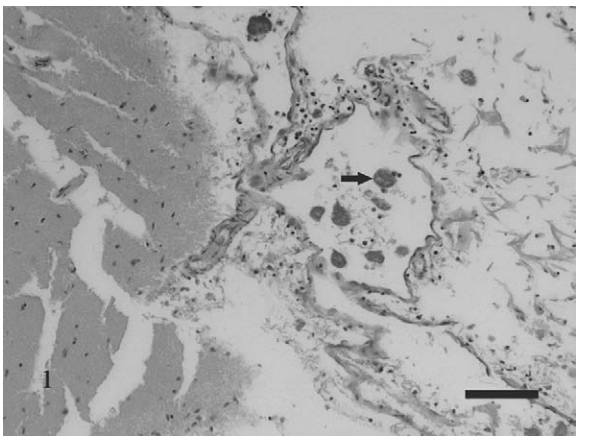

Fig. 1. Brain; intravascular schizonts (arrow) in the meningeal vasculature. HE stain. Bar = 100 μm.

Cytauxzoonosis was first described in Missouri in the 1970's (Jakob and Wesemeier, 1996; Meinkoth et al., 2000; Verdon, 2002) with naturally occurring cases having been reported in Oklahoma, Missouri, Texas, Georgia, Arkansas, Mississippi, Florida, Louisiana and Kansas (Glenn and Stair, 1984; Hauck, 1982; Hoover et al., 1994; Kier et al., 1982a,b;

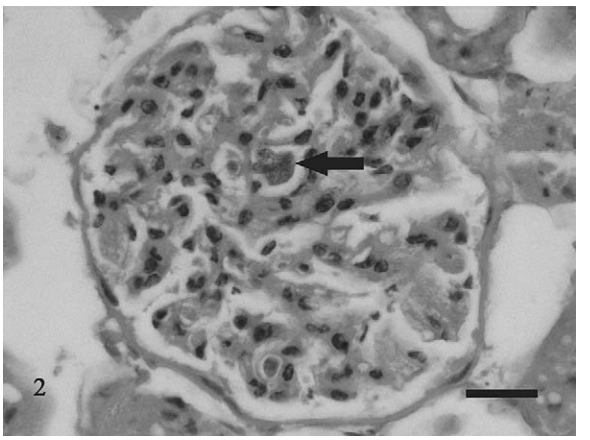

Fig. 2. Kidney; intravascular schizont (arrow) in a glomerular tuft. HE stain. Bar = 60 μm.