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Disseminated Toxoplasmosis in a Captive Porcupine (*Coendou mexicanus*) from Costa Rica

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Source: *The Journal of Parasitology*, Vol. 82, No. 1 (Feb., 1996), pp. 185-186

Published by: [Allen Press](#) on behalf of [American Society of Parasitologists](#)

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*J. Parasitol.*, 82(1), 1996, p. 185–186  
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## Disseminated Toxoplasmosis in a Captive Porcupine (*Coendou mexicanus*) from Costa Rica

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**ABSTRACT:** An adult porcupine (*Coendou mexicanus*) from the National Zoo, San Jose, Costa Rica, died because of toxoplasmosis involving the heart, lungs, liver, and kidneys. *Toxoplasma gondii* was found in lesions and the diagnosis was confirmed by immunohistochemical staining with *T. gondii*-specific polyclonal rabbit antibodies. This is a new host record for *T. gondii*.

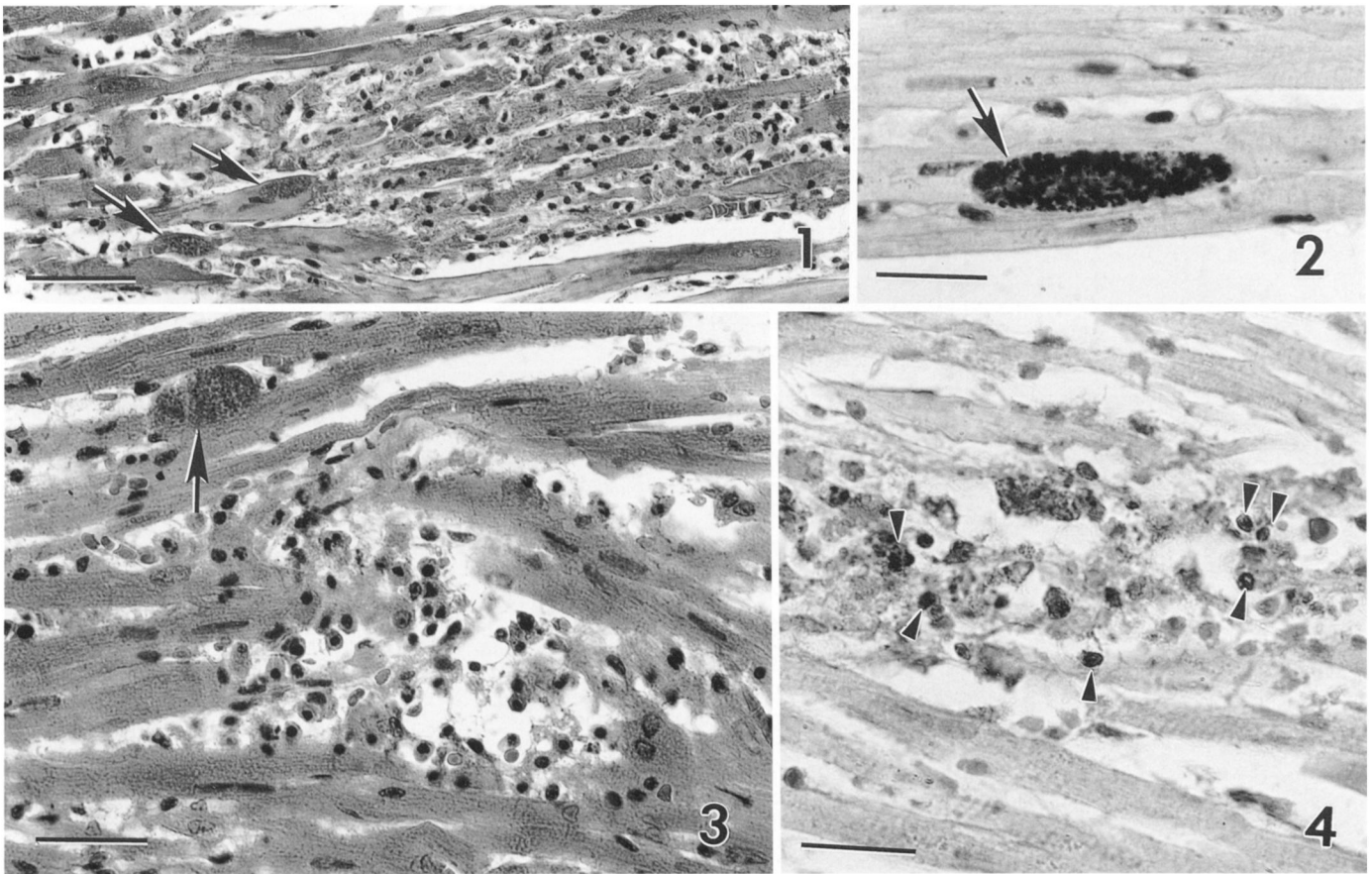
Infections by *Toxoplasma gondii* are widespread in many species of warm-blooded animals (Dubey and Beattie, 1988). We report disseminated toxoplasmosis in a porcupine (*Coendou mexicanus*) from Costa Rica. This is the first report of *T. gondii* infection in this host.

An approximately 4-yr-old male porcupine was submitted for diagnosis in September 1990 to the Department of Pathology, School of Veterinary Medicine, the National University, Heredia, Costa Rica. The porcupine was from the National Zoo in San José (Zoológico Nacional, Simón Bolívar) and was found dead without any obvious sign of illness. A necropsy was performed. Portions of heart, lungs, liver, kidneys, stomach, and intestines were fixed in 10% formalin, and paraffin-embedded

sections were stained with hematoxylin and eosin or periodic acid Schiff (PAS) reaction. Retrospectively, paraffin sections were reacted with polyclonal antibodies to *T. gondii* and *Neospora caninum* raised in rabbits using reagents and techniques described by Lindsay and Dubey (1989).

Grossly, the lungs were congested and edematous. Microscopically, multifocal myocarditis and diffuse granulomatous pneumonia were the most predominant lesions. Myocardial changes consisted of necrosis of small groups of myocytes and multifocal, interstitial myocarditis with infiltration of plasma cells, lymphocytes, macrophages, and a few neutrophils (Figs. 1–3). Tachyzoites were seen in cardiac myocytes bordering the lesions and among inflammatory cells. Tissue cysts were present in myocytes often without inflammatory response. Tissue cysts were thin-walled (<0.5  $\mu$ m thick), elongated, and contained hundreds of PAS-positive bradyzoites (Fig. 4).

The lungs were congested and had diffuse interstitial pneumonia. Alveolar septa were thickened by infiltration of macrophages, lymphocytes, plasma cells, neutrophils, and fibrin.



FIGURES 1–4. *Toxoplasma gondii* in lesions in the heart of the naturally infected porcupine from Costa Rica. 1. A focus of myocardial necrosis with tissue cysts (arrows) at the periphery of the lesion. Hematoxylin and eosin. Bar = 100  $\mu\text{m}$ . 2. A tissue cyst (arrow) with thin cyst wall and enclosing PAS-positive bradyzoites. PAS reaction and hematoxylin. Bar = 20  $\mu\text{m}$ . 3. Infiltration of mononuclear cells among myocytes. Arrow points to a tissue cyst. Hematoxylin and eosin. Bar = 50  $\mu\text{m}$ . 4. A necrotic focus with several *T. gondii* tachyzoites (arrowheads). Avidin–biotin immunohistochemical stain with anti-*T. gondii* serum. Bar = 20  $\mu\text{m}$ .

Foci of necrosis were noted in the septal wall and bronchiolar epithelium. Tachyzoites were present in pulmonic lesions. Proteinaceous fluid, fibrin, and macrophages with microvacuolated cytoplasm were present in alveolar spaces.

Renal lesions consisted of scattered areas of tubular epithelial cell necrosis and interstitial aggregates of lymphocytes, plasma cells, and macrophages. Tachyzoites were seen in glomeruli and in tubular epithelium.

In the liver, the hepatocytes had an accumulation of biliary pigment in the cytoplasm and there were multiple foci of hepatocellular necrosis and mononuclear cell infiltrates. Additionally, a small number of lymphocytes, plasma cells, and macrophages were present in the portal area. Few tachyzoites were seen in hepatocytes.

Protozoan organisms reacted strongly with *T. gondii* antibodies but not with *N. caninum* antibodies. Many more tachyzoites were seen in sections stained with anti-*T. gondii* antibodies than in hematoxylin/eosin-stained sections.

The porcupine probably became infected with *T. gondii* by ingesting food or water contaminated with oocysts. The porcupine was caged and was fed corn and vegetables but not meat. Feral cats were known to be present in the zoo, especially during the night.

Medway et al. (1989) reported *T. gondii*-like organisms in

sections of the brains of 2 American porcupines (*Erethizon dorsatum*) from Pennsylvania that had neurological signs; 1 of these porcupines also was concurrently infected with the raccoon ascarid *Baylisascaris* sp. Marchiondo et al. (1976) found antibodies to *T. gondii* in 2 of 10 apparently healthy *E. dorsatum* trapped from New Mexico, Arizona, and Colorado.

In the present report from Costa Rica, the porcupine was of a different species than reported from North America and the diagnosis was confirmed by immunohistochemical staining with *T. gondii*-specific antibodies.

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