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Biliary cryptosporidiosis in two corn snakes (Elaphe guttata)

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Cryptosporidia are 5-7- m apicomplexid coccidian parasites that infect humans and many other species of mammals, birds, reptiles, and fish. In humans and cattle, infection by Cryptosporidium parvum usually affects the intestinal tract, resulting in self-limiting diarrhea.[4] Respiratory and intestinal cryptosporidiosis occur in birds infected with C. bayleyi and C. meleagridis.[5] Sneezing, coughing, or dyspnea are associated with infection of the nasal cavity and sinuses, trachea, or air sacs, whereas intestinal infection is manifested by diarrhea. Other cryptosporidia, such as C. muris in mice[7] and C. serpentis in snakes,[3] affect the gastric mucosa. In this study, we report the occurrence of concurrent gastric and biliary cryptosporidiosis in snakes.

A total of 13 snakes raised by a commercial breeder were evaluated for the presence of cryptosporidia. Two corn snakes (Elaphe guttata) purchased for inclusion in a reptile exhibit developed clinical signs typical of cryptosporidiosis, including chronic postprandial regurgitation, weight loss, midbody swelling, and the presence of large numbers of oocysts in both regurgitated material and feces. Neither snake, exhibited signs that could be attributed specifically to biliary disease. Subsequently, 11 snakes (9 corn snakes, 1 king snake [Lampropeltis getula], 1 rat snake [Elaphe obsoleta]), culled for poor reproductive performance, were obtained from the commercial breeder. All 13 snakes were euthanized by deep anesthesia with halothane[a] followed by decapitation. Fresh gastric content and fecal material from the distal intestinal tract were collected for parasitologic examination. Tissue from the heart, lung, stomach, liver, gall bladder, pancreas, intestine, and kidney were fixed in 10% buffered formalin, embedded in paraffin, sectioned at 6 m, and stained with hematoxylin and eosin (HE) for light microscopic examination.

Light microscopic examination revealed the presence of cryptosporidian oocysts in the gastric content and feces of 6 of the 13 snakes; all 6 were corn snakes. Additionally, feces from 1 of these 6 snakes contained moderate numbers of Eimeria oocysts, and another snake had small numbers of Ophiostrongylus ova. Fecal smears from 2 of the 7 cryptosporidia-negative snakes had Ophisascaris and Ophiostrongylus ova and Eimeria oocysts.

Five of 6 Cryptosporidium-infected corn snakes had moderate to severe hypertrophic gastritis characterized by thickening of the gastric mucosa, accentuation of longitudinal gastric ridges, and narrowing of the gastric lumen. The stomach of the 6th infected snake was thin walled and markedly distended by a partially digested mouse. Microscopically, the gastric mucosae of the 6 infected snakes were thickened, gastric glands were dilated, and numerous Cryptosporidia were present on the luminal and glandular epithelial surfaces. The lamina propria and submucosa were edematous and infiltrated by moderate numbers of heterophils and fewer numbers of lymphocytes. In the 2 clinically affected snakes, there was mild to moderate lymphocytic cholecystitis and cholangitis. Small to moderate numbers of Cryptosporidia were associated with the microvillous borders of the gall bladder and intra- and extrahepatic bile ducts (Figs. 1-3). The lamina propria of the gall bladder was diffusely infiltrated by moderate numbers of lymphocytes. Similarly, cuffs of small to moderate numbers of lymphocytes were present around intra- and extrahepatic bile ducts.

Cryptosporidiosis in snakes is an insidious and slowly progressive disease characterized by postprandial regurgitation and chronic weight loss. The most common lesion of the disease is hypertrophic gastritis with thickening of the gastric mucosa, accentuation of longitudinal ridges, and narrowing of the gastric 1umen.[3] Concurrent infection of the biliary system has not been previously documented in snakes. However, cryptosporidiosis of hepatic bile ducts and pancreatic ducts has been reported in primates and mice.[1,2,6] In these species, biliary infection usually occurs in C. parvum-infected immunodeficient animals, particularly AIDS patients, simian immunodeficiency virus-infected monkeys, and severe combined mice and is associated with prolonged, severe, life-threatening intestinal cryptosporidiosis. Similarly, the 2 snakes in this study with biliary cryptosporidiosis also had chronic debilitating gastric cryptosporidiosis. The role that immunodeficiency may play in predisposing snakes to biliary cryptosporidiosis is unknown. Adenoviruses in snakes may cause immunodeficiency, predisposing them to more severe infections.[8] Extension of infection into the biliary system may also

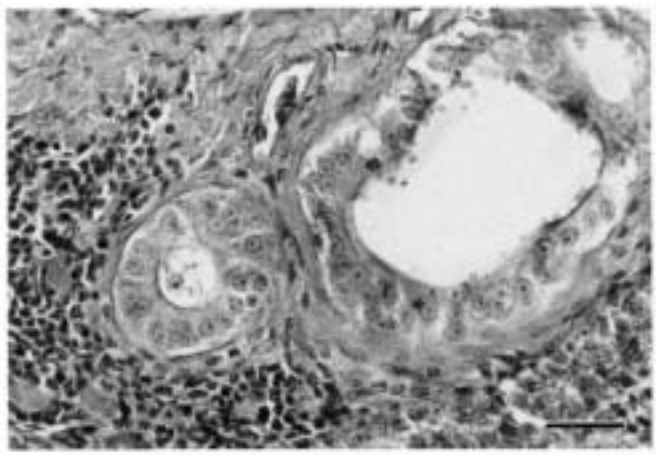

Figure 1. Liver, bile duct; E. guttata. There are cryptosporidia on the mucosal epithelium and moderate lymphocytic pericholangitis. HE. Bar = 70 m.

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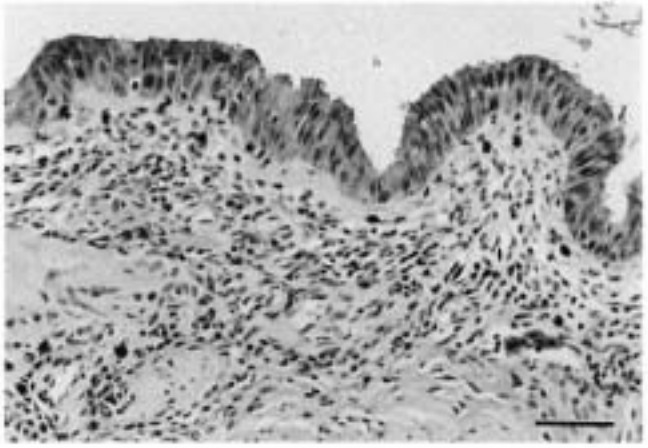

Figure 2. Extrahepatic bile duct; E. guttata. Lymphocytes infiltrate the periductular space. HE. Bar = 150 m.

occur simply as a result of persistent and prolonged gastric disease, regardless of the factors responsible for the initial infection. The absence of biliary infection in 4 Cryptosporidium-infected snakes, none of which exhibited clinical signs of disease, suggests that biliary infection may be an important factor in the development of clinical disease. However, it is unlikely that biliary infection occurs independently of persistent gastric disease. Additional observations of the biliary system from both clinical and subclinical cases of cryptosporidiosis in snakes are necessary to clarify the relationships between gastric and biliary components of this disease.

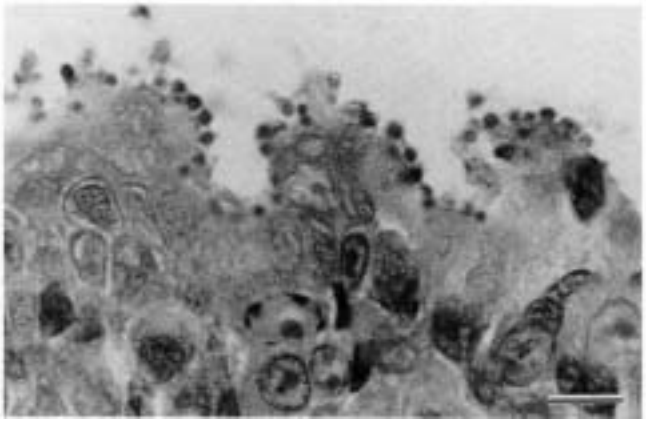

Figure 3. Extrahepatic bile duct; E. guttata. Large numbers of cryptosporidia are present on the mucosal epithelium. HE. Bar = 12.5 m.

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Sources and manufacturers

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