OPOSSUM NEMATODIASIS: DIAGNOSIS AND TREATMENT OF STOMACH, INTESTINE, AND LUNG NEMATODES IN THE VIRGINIA OPOSSUM (*DIDELPHIS VIRGINIANA*)

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Abstract

Internal parasitic infections are common in the Virginia opossum (*Didelphis virginiana*). More than 50 different species of nematodes have been identified from the genus *Didelphis*. Of them, 3 can cause significant illness to native opossums in Southern California, 2 gastrointestinal nematodes and 1 pulmonary nematode. This article provides an overview of the life cycles, methods for identification, pathologic findings, diagnostic tests, clinical signs, and options for treating these common nematode species. Copyright 2013 Elsevier Inc. All rights reserved.

Key words: *Cruzia americana*; *Didelphostrongylus hayesi*; *Didelphis virginiana*; nematodes; opossum; *Turgida turgida*

Virginia opossums (*Didelphis virginiana*) acquire endoparasites because of their foraging and scavenging behavior. The type of parasite and source of exposure depend on the maturity of the opossum and various environmental factors, including weather, seasonal temperature, habitat, and food availability. In Southern California, opossums are often infected with 3 clinically important nematodes: a stomach roundworm (*Turgida turgida*) from the family Physalopteroidea, an intestinal roundworm (*Cruzia americana*) from the family Cruzidae, and a lung nematode (*Didelphostrongylus hayesi*) from the family Metastrongyloidea.[1]

In the author's experience, every opossum captured from the wild has endoparasites. Nematodes cause pathology to their host by direct physical damage to tissues, leading to easier access for opportunistic bacterial organisms to invade and disseminate. The cumulative effect of endoparasitism contributes to chronic debilitation of the host.[2] Nematode infections can lead to sepsis, blood loss, anemia, stomach ulceration, respiratory problems, peritonitis, and organ damage. Chronic, prolonged infection combined with trauma, stress, and malnutrition results in systemic compromise, delayed disease recovery, and often death. Levamisole was previously used for the treatment of internal parasites in Virginia opossums. According to the World Health Organization, levamisole has been restricted in its use and availability since 2001. Veterinarians should be aware of available alternative drug choices to treat endoparasites in parasitized animals, including the Virginia opossum.

STOMACH NEMATODES

Opossums become infected with *T. turgida* by ingesting coprophagic insects such as beetles, crickets, cockroaches, and earwigs. Adult *T. turgida* consume ingesta within the stomach of the

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opossum and attach to the mucosal lining of this organ when not feeding. The worm burden found in the stomach can be overwhelming (Fig. 1A). *Turgida turgida* attaches to the mucosal lining of the greater curvature of the stomach wall (Fig. 1B), with larvae scattered throughout the remainder of the stomach. Adult *T. turgida* can live for 360 days in the opossum host.[3]

Ulceration occurs at the site of attachment and can vary in diameter from 1 to >24 mm (Fig. 1C and D). Gastric ulcers associated with the attachment sites of *T. turgida* can result in chronic blood loss and anemia. The ulcer size and bleeding that occurs with the nematode infection is directly correlated with the number of parasites present.[2] The parasites compromise the mucosal and submucosal stomach wall layers, leaving only a thin layer of granulation tissue separating the parasites from the peritoneal cavity. In severe cases, perforation of the stomach wall occurs. Ulceration allows opportunistic enteric bacteria to enter the host's circulatory system.

Fibrinous exudate may be present on the serosal surface as the muscularis externa is replaced with granulation tissue. The tissue becomes infiltrated with eosinophils, neutrophils, fibroblasts, collagen fiber and nerotic debris.

Microscopic examination of affected tissue would often reveal eosinophils on the surface of the ulcer (Fig. 1E and F).[3]

Clinical signs associated with *T. turgida* infection include weight loss, anorexia, profuse black diarrhea (melena), anemia, poor hair coat, and death.[4] A definitive diagnosis is achieved with fecal centrifugation floatation analysis. The eggs of this parasite are 40 to 45 μm in length and are ovoid shaped with a thick clear colorless shell that contains a fully formed larval worm.[5] Parasitized animals can shed a large number of eggs in their feces.

INTESTINAL NEMATODES

Cruzia americana parasitizes the cecum and large intestine of the opossum. These intestinal parasites have also been identified in armadillos, raccoons, and pigs. It is believed that reptiles may be the natural host of *C. americana*.[6] The exact mode of transmission for *C. americana* is unknown; however, it is believed to be direct. First-stage larvae are formed in 7 to 9 days, and the first molt takes place within 10 days of the eggs being shed in the feces. In the opossum, ingested eggs with second-stage larvae are hatched in the duodenum or upper ileum. After hatching, the larvae are transported through the intestines via peristalsis to the cecum. In the cecum, the larvae undergo 3 molts to reach the adult stage (Fig. 2A and B); the adults go on to infect the colon (Fig. 2C). *Cruzia americana* eggs are typically shed 46 to 48 days after worm maturation within the intestinal tract.[7]

Cruzia americana attaches to the wall of the cecum and lower intestine. This parasite feeds on the intestinal mucosa, ingesting blood and absorbing nutrients. Adult *C. americana* reside in the ileum and colon. The pathology associated with this parasite can be correlated with the parasite burden. Gross pathologic findings in an infected animal reveal evidence of localized hemorrhage and tissue damage (Fig. 2D). Histopathologic examination of affected tissues often reveals localized inflammation and hyperplastic crypts (Fig. 2E and F).

Diarrhea is the only overt clinical sign reported in animals infected with *C. americana*.[1] The clinical disease associated with this parasite is routinely overshadowed by the pathology caused by *T. turgida*. *Cruzia americana* appears to interfere with host nutrition and causes blood loss and anemia.[2]

Clear 48- to 52-μm × 84- to 100-μm eggs with fine irregular transverse striations in a morula or

FIGURE 1. The nematode *Turgida turgida* in the stomach of an opossum. (A) Large numbers of nematodes in the stomach of an 8-month-old opossum. Larvae and adults feed on stomach contents and attach to the mucosal lining of the organ when not feeding. Larvae scatter throughout the stomach, but adults often congregate in 1 to 3 groups in the cranial areas. (B) Adult *T. turgida* attach to the greater curvature of the stomach. Most worms are found in this location immediately caudal to the fundus. (C) Ulceration caused by *T. turgida*. Ulcers always occur at the site of the attachment. The size of the ulcer is related to the number of adult worms present and the length of time the animals have been infected. Small ulcers, 2 to 3 mm in diameter, are associated with the attachment of only a few adults. Small ulcers extend superficially into the submucosa. (D) Severe ulceration with mucosal compromise and perforation. Note large ulcers 6 to 15 mm in diameter. The mucosa and submucosa are destroyed, but some of the muscularis externa remains intact in the region of the ulcer. Large ulcers extend deep into the submucosa. A thin layer of granulation tissue separates nematodes from the peritoneal cavity. Most of the muscularis externa is replaced by granulation tissue. (E) Eosinophils, neutrophils, fibroblasts, and collagen fiber material are present at the surface of the ulcer and are associated with attachment areas (arrows). (F) Multifocal to coalescing, mild to marked, nodular to polypoid gastric mucosal hyperplasia with nodular to confluent mural lymphocytic, plasmacytic, pyogranulomatous, and eosinophilic and ulcerative gastritis (arrows).

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