Eosinophilic bronchitis caused by *Crenosoma vulpis* infection in dogs

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Eosinophilic bronchitis in dogs usually results from parasitic infection or allergic (hypersensitivity) reactions (1). Parasites that infect the lower respiratory tract of dogs include *Paragonimus kellicotti, Capillaria* (=*Eucoleus*) *aerophila, Oslerus* (=*Filaroides*) *osleri, Filaroides hirthi, Andersonstrongylus* (=*Filaroides*) *milksi,* and *Crenosoma vulpis* (1). Allergic causes of eosinophilic inflammation are usually attributed to an environmental antigen or, in some cases, heartworm infection. Parasitic lower respiratory infections in dogs are considered uncommon (1). Consequently, when eosinophilic airway inflammation is detected using transtracheal wash cytology, an allergic etiology is often considered most likely. In areas where heartworm or lungworm infection is considered rare, diagnostic investigation may cease and corticosteroids are prescribed to suppress the eosinophilic inflammation. This approach may overlook a potentially curable disorder, such as lungworm infection, and inflict unnecessary long-term corticosteroid therapy with its related side effects on affected dogs.

Crenosoma vulpis is a metastrongyloid lungworm that infects many wild canids, especially foxes (2). The parasite is prevalent in the fox population of the northeastern United States and the Maritime provinces (3–5). Infection with *Crenosoma vulpis* in domestic dogs has been reported rarely (6,7).

This paper describes the clinical, radiographic, and laboratory findings in 3 dogs diagnosed with *Crenosoma vulpis* infection that were presented to the Atlantic Veterinary College — Veterinary Teaching Hospital (AVCVTH) during a 13-month period (July 1993 to August 1994).

Dog 1

A 12-year-old, female spayed, collie was presented for a deep rasping, somewhat productive cough of 15-days duration. The dog lived in a small rural community. The dog's appetite was reduced. Treatment with amoxicillin had not resulted in an improvement in the clinical signs. A cough was easily induced on tracheal palpation. Body temperature, pulse, respiratory rate, and lung sounds were normal. Thoracic radiographs revealed a moderate bronchial pattern, with an alveolar pattern in the right cranial and middle lung lobes. Mild eosinophilia was present (1.712×10^9/L). Cytologic examination of samples collected by transtracheal wash were highly cellular. Eosinophils, neutrophils, and large mononuclear cells made up approximately 50%, 40%, and 10% of the cells present, respectively. About 4 larvated eggs or free larvae per slide were observed on direct smears of the transtracheal wash fluid. The eggs were oval, clear (unstained), and about 40 × 70 µm in size (Figure 1). Larvae were visible within eggs in a figure eight pattern, or free in the sample, and were approximately 300 µm in length with blunt conical anterior ends and finely tapered tails containing a slight deflection with no severe kinks or spines (Figure 2). Larval morphology was characteristic of *C. vulpis* (8).

A Baermann fecal examination was positive for larvae. The dog was treated with fenbendazole (33 mg/kg body weight (BW) PO, q24h for 5 d). Five days later, a Baermann fecal examination was negative, the alveolar pattern in the right cranial and middle lung lobes had resolved, and the frequency of coughing was significantly reduced.

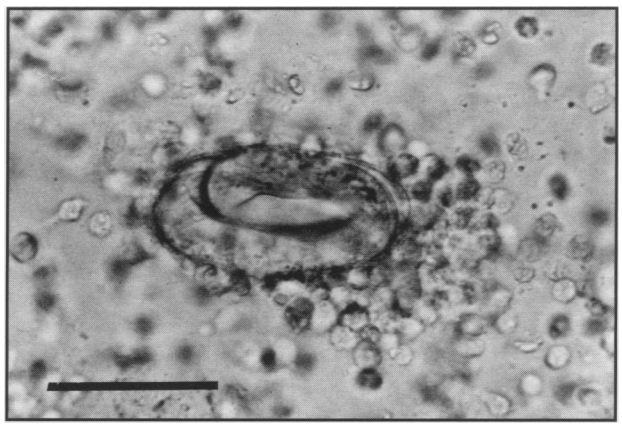

Figure 1. Egg of *Crenosoma vulpis* containing a first-stage larvae (unstained, wet mount preparation from a transtracheal wash sample). Bar = 50 µm.

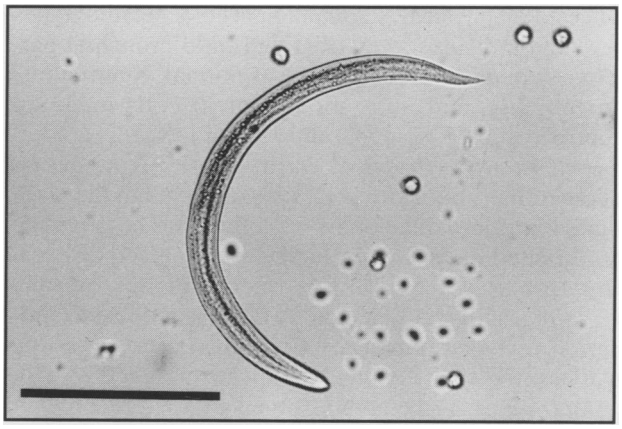

Figure 2. First-stage larvae of *Crenosoma vulpis* recovered from the feces of a dog using the Baermann technique (wet mount, iodine stained). Identification is based on morphology (bluntly conical anterior end, tail finely tapered with a slight deflection but lacking any severe kinks or spines) and size (246 to 308 µm in length). Bar = 100 µm.

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Dog 2

A 6-month-old, female miniature dachshund was presented for a productive cough of 5-days duration. The dog's attitude, activity level, and appetite were normal. Physical examination was normal. A diffuse interstitial and mild bronchial pattern was observed on thoracic radiographs. The transtracheal wash fluid was highly cellular and made up of neutrophils (10%), eosinophils (75%), and large mononuclear cells (15%). Bacterial culture of the wash fluid was negative. Numerous free larvae or larvated eggs were observed (approximately 15/slide) on a direct smear of the wash fluid. The morphology of the eggs and larvae was similar to that described in dog 1. A complete blood count was not performed. Fecal flotation was negative. A Baermann fecal examination was positive for *C. vulpis* larvae. The dog was treated with fenbendazole (50 mg/kg BW, PO, q24h for 7 d). The cough resolved within 2 to 3 d.

Dog 3

A 6-month-old, female miniature dachshund was presented because of a moist productive cough of 2-days duration. The dog lived on an acreage with dog 2 and was presented 2 d after dog 2. The dog's attitude, activity level, and appetite were normal. No abnormalities were detected on physical examination. Thoracic radiographs revealed a diffuse interstitial and mild bronchial pattern. A complete blood count was normal. Neutrophils, eosinophils, and large mononuclear cells made up approximately 70%, 15%, and 15% of the cells, respectively, in a transtracheal wash sample. A moderate number of erythrocytes were also observed in the wash fluid. Bacterial culture of the wash fluid was negative and no larvated eggs or free larvae were detected. Fecal flotation was negative for other parasites but one *C. vulpis* larva was detected. A Baermann fecal examination revealed numerous *C. vulpis* larvae. The dog was treated with fenbendazole (50 mg/kg BW, PO, q24h for 7 d). The cough resolved within 2 to 3 d.

During the period when these 3 dogs were diagnosed, 3 other dogs with almost identical clinical (including transtracheal wash findings) and radiographic signs were presented to the AVCVTH. One of the dogs lived with dogs 2 and 3. However, no *C. vulpis* larvae (or any other lungworm) could be identified on either fecal flotation or Baermann fecal examination. Treatment with fenbendazole resulted in prompt resolution of the cough and most radiographic signs. We consider it quite likely that these dogs were also infected with *C. vulpis*.

There was a wide age range in affected dogs (6 mo to 12 y). The ages of 2 previously reported cases were 8 and 10 mo (6,7). A common historical feature was that all dogs had liberal access to the outdoors and resided in rural areas. In a rural environment, the intermediate hosts (slugs and snails) would be infected with *C. vulpis* more often, due to the proximity of the definitive wildlife host, the fox. The duration of illness varied from 2 to 15 d. All dogs had a productive cough, and systemic signs of illness were either not present or mild.

The radiographic findings in these dogs were similar to those reported in 2 other cases of dogs naturally infected with *C. vulpis* (6,7). These nonspecific abnormalities are also seen in dogs with nonobstructive chronic bronchitis due to a variety of etiologies, such as, viruses or inhalation of irritants or dust. A bronchial pattern results from bronchial wall thickening, which can be visible as "tram lines" and "doughnuts" in peripheral lung fields. A reticular interstitial pattern is seen as a diffuse, unstructured "lacy" increase in density of the pulmonary interstitium, which often obscures vessels and airways (9). The calcification of airways observed in all of the affected dogs has been reported as a normal aging change in older dogs. However, 2 dogs in this series were young, indicating that the calcification was more likely related to chronic airway inflammation (9).

Transtracheal aspirates from these dogs showed a range of cellularity. Eosinophils were identified in significant numbers in all samples. However, neutrophils predominated in 1 dog. In many cases, the eosinophils showed evidence of degranulation, and it is possible that some eosinophils were mistakenly included in the neutrophil count. Erythrocytes with no evidence of erythrocytophagia or intracellular hemosiderin were seen in the transtracheal aspirates from 1 dog, suggesting mucosal trauma during sampling. Parasite eggs and/or free larvae were identified in transtracheal wash samples from 2 dogs.

Fenbendazole is commonly recommended for the treatment of lungworm infections in dogs and cats (1). Other drugs reported to be efficacious in *C. vulpis* infections include febantel (6), diethylcarbamazine, and levamisole (10). Ivermectin (200 µg/kg BW, SC) has been used successfully to treat 2 silver foxes in a zoo environment (Conboy, unpublished observations). A single course of treatment appeared to cure the infected dogs, as no relapses occurred.

Clinically apparent infection with *C. vulpis* has rarely been reported in domestic dogs. However, infection is very common in the red fox (*Vulpes vulpes*) in eastern Canada. It has been reported in Newfoundland, New Brunswick, Nova Scotia, and Ontario (3,5,8). Surveys have indicated infection rates in the red fox of 54.1% in Nova Scotia and New Brunswick (3) and as high as 80% in Prince Edward Island (Conboy, unpublished observations). The role of *C. vulpis* as a pathogen in the fox has not been thoroughly studied, but bronchopneumonia due to infection with it has been reported in foxes, and in several cases death was attributed to the infection (4).

Canids are exposed to infective third-stage larvae of *C. vulpis* by ingesting molluscan intermediate hosts (slugs, terrestrial snails). Larvae are digested free of the mollusc and penetrate through the stomach wall to reach the liver and then lungs via the circulatory system. Larvae reach the lungs as early as 20 h postinfection (11). Adult worms mainly inhabit the distal portions of the bronchial tree. The prepatent period is 19 to 21 d (11). Larvated eggs are produced, which hatch first-stage larvae as they are coughed-up, swallowed, and passed in the feces. The larvae infect terrestrial gastropods, reaching the infective third-stage in about 17 d (12). The role of paratenic host(s) in the transmission of *C. vulpis* is unknown. Experimentally infected dogs and foxes passed larvae in the feces for 240 to 290 d (12); this time period may represent the life span of the parasite after a single infection.

Definitive diagnosis of *C. vulpis* infection involves either the detection of first-stage larvae (Figure 2) in the feces with the Baermann technique or the finding of larvated eggs (Figure 1) or larvae by transtracheal wash. Since the Baermann technique relies on larval motility for recovery, fresh feces must be used (formalin preserved fecal samples are unsuitable). Alternatively, larvae can be detected on centrifugal flotation examination of feces using zinc sulfate ($ZnSO_4$); however, they may be damaged to a degree that prevents proper evaluation of morphological features. First-stage larvae of *C. vulpis* are identified based on morphology and size (246–308 μm in length) (8). Evaluation of larval morphology is aided by immobilizing (killing) and staining the larvae by the addition of a drop of dilute iodine at the edge of the coverslip. *Crenosoma vulpis* eggs recovered from the transtracheal wash are thin-walled, 72 × 43 μm in size, and contain fully developed first-stage larvae (Figure 2). Fecal flotation examinations should be done to rule-out infection with *Oslerus* (=*Filaroides*) *osleri* and *Capillaria aerophila*. A flotation method using $ZnSO_4$ flotation medium (specific gravity = 1.18) is recommended for *O. osleri*.

Crenosoma vulpis infection should be considered in dogs with eosinophilic airway inflammation or bronchitis, especially in the Atlantic provinces. The prevalence of infection in domestic dogs may be much higher than previously thought. This may also be true in other geographic areas that have large fox populations and the necessary intermediate hosts (slugs, terrestrial snails). Since larvae or larvated eggs may not be found on fecal or transtracheal wash examinations, trial therapy with fenbendazole should be considered in dogs with eosinophilic airway cytology and an interstitial and/or bronchial pattern on thoracic radiographs, even when Baermann fecal examinations are negative. A prompt (within days) response to fenbendazole would support a diagnosis of parasitic lower respiratory disease.

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