Rhinoscopic Diagnosis of *Eucoleus boehmi* Infection in a Dog

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ABSTRACT

A dog presenting for chronic purulent nasal discharge was diagnosed with an *Eucoleus boehmi* infection based upon rhinoscopic appearance of the nasal worms in situ, identification of the adult parasites in rhinoscopic nasal biopsies, and ova in the feces. The dog was successfully treated with a 2 wk course of fenbendazole and measures preventing reinfection through coprophagia. Patients with chronic nasal discharge should have a fecal examination performed to rule out infection with *E. boehmi*. (*J Am Anim Hosp Assoc* 2011; 47:60–63. DOI 10.5326/JAAHA-MS-5707)

A 3.5 yr old castrated male American foxhound dog was presented for evaluation of chronic bilateral nasal discharge of 18 mo duration. The dog had been adopted from a research colony at 2 yr of age and was neutered without complication. Approximately 1 mo after adoption, the dog developed a bilateral nasal discharge that progressed from serosanguineous to mucopurulent over the ensuing 6 mo. A rhinoscopic evaluation at that time revealed bilateral mildly edematous and erythematous nasal mucosae. Biopsies of the nasal mucosa showed a chronic plasmacytic and eosinophilic rhinitis with submucosal edema and marked infiltration of plasma cells, eosinophils, neutrophils, and lymphocytes. Initial treatment following the rhinoscopy included amoxicillin trihydrate/clavulanate potassium[a] (16.7 mg/kg per os [PO] *q* 12 hr for 5 days) and prednisone (0.65 mg/kg PO *q* 12 hr for 7 days). The nasal discharge temporarily improved, but did not resolve.

The owner, a veterinarian, empirically treated the dog throughout the next 1.5 yr with several courses of antibiotics (amoxicillin trihydrate/clavulanate potassium, azithromycin, and cephalexin) and with various other drugs including antihistamines (diphenhydramine), nonsteroidal anti-inflammatory drugs (i.e., carprofen, tepoxalin, piroxicam), corticosteroids (i.e., prednisone, dexamethasone, methylprednisolone acetate), azathioprine, chlorambucil, and cyclosporine. At the time of presentation, the dog was receiving a pulsed dosing schedule of dexamethasone (0.4 mg/kg PO *q* 7 days) which resulted in the best control of clinical signs. Despite improvement, the nasal discharge persisted with this treatment and the right zygomatic lymph node had become enlarged.

Prior to its adoption, the dog had been part of a research study. Its origin and travel history were unknown. The dog was vaccinated against canine distemper, adenovirus type 2, parvovirus, parainfluenza[b], leptospirosis[c], *Bordetella bronchiseptica*[d], and rabies[e], and was receiving a monthly heartworm preventative (ivermectin[f]). He had been treated 1 yr prior to presentation for a presumptive nasal mite infection with two doses of ivermectin[g] (0.2 mg/kg PO 2 wk apart) which had resulted in a temporary improvement in clinical signs.

Other medical history included a diagnosis of a yeast pododermatitis and paronychia 4 mo prior to presentation. Intradermal allergy testing revealed a mild positive response to sheep wool and storage mite antigens. The dog was administered antigen-specific immunotherapy injections, but these were discontinued due to a perceived worsening of the nasal discharge.

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CBC *complete blood count;* CT *computed tomography;* HPF *high-power field;* PO *per os*

On presentation, the dog was bright, alert and responsive, weighed 37 kg, and had a body condition score of 4 out of 5. Rectal temperature, pulse rate, and respiration rate were normal. Bilateral serous nasal discharge was present and the dog sneezed occasionally during the examination. The right zygomatic lymph node was palpable. The remainder of the physical examination was unremarkable. A CBC and serum chemistry profile revealed a hyperproteinemia (7.5 g/dL; reference range, 5.7–7.2 g/dL), lymphocytopenia 0.8 $\times$ 10^9/L (reference range, 1.0–4.6 $\times$ 10^9/L), basophilia (0.4 $\times$ 10^9/L; reference range, 0), hypokalemia (3.4 mEq/L; range, 4.2–5.4 mEq/L), and hyperglobulinemia (3.3 g/dL; reference range, 2.2–2.9 g/dL). Urinalysis revealed a specific gravity of 1.039, negative dipstick reactions, 0–1 WBC/high-power field (HPF), 0–1 epithelial squamous cells/HPF, and 0–2 erythrocytes/HPF.

After an overnight fast, the dog was premedicated intramuscularly with acepromazine[h] (0.08 mg/kg) and hydromorphone[i] (0.05 mg/kg). General anesthesia was induced with thiopental[j] (6.3 mg/kg IV), and maintained with sevoflurane[k] in oxygen.

CT scan images of the nasal cavity and sinuses revealed a thickening of the nasal turbinates. Contrast enhancing, increased soft-tissue attenuation material resulted in the loss of definition of the right and left nasal turbinates. These findings were consistent with chronic inflammation. The cribriform plate was within normal limits.

Rhinoscopy was performed with the dog in ventral recumbency. An endotracheal tube (14 mm) with cuff inflated was in place throughout the procedure. Retrograde rhinoscopic examination of the choanae was performed using a flexible videoendoscope[l]. Antegrade rhinoscopic examination was performed using a rigid arthroscope[m] (5.7 mm in diameter and 16.5 cm in length). Sterile 0.9% saline solution was infused continuously through the scope channel during the rigid rhinoscopic procedure. Biopsies were taken through the channel of the rigid scope using 2.0 mm arthroscopic biopsy forceps[n]. Additional antegrade biopsies were taken blindly without the scope using rigid cup biopsy forceps[o] (4.5 mm in diameter and 30 cm in length).

Rhinoscopy revealed diffuse bilateral erythematous mucosae without visible erosions or bleeding. Upon antegrade examination, several adult *E. boehmi* nematodes were identified as white, linear, serpentine-shaped worms on the surface of the turbinate mucosae of both the left and right nasal passages (**Figure 1**). Multiple attempts were made to retrieve some of the worms using the endoscopic biopsy forceps, but they were too friable and broke apart upon efforts to pick them off the mucosa. Nematodes were not seen on the retrograde examination of the choanae. Histopathologic evaluation of biopsies of the nasal mucosa revealed a marked plasmacytic rhinitis with low numbers of eosinophils. Several *E. boehmi* parasites were present on the surface of the epithelium (**Figure 2**).

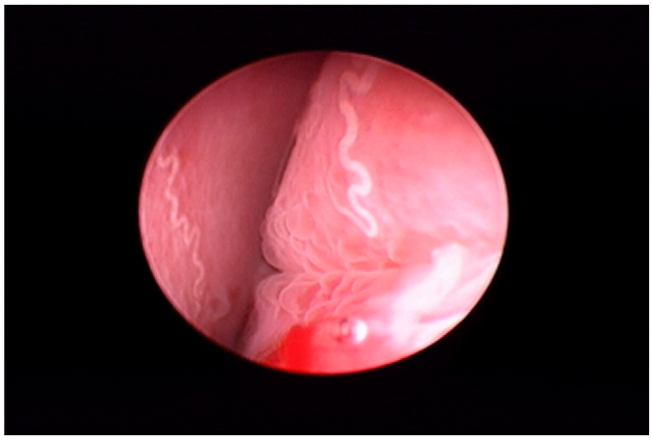

FIGURE 1 *Rhinoscopic view of* Eucoleus boehmi *adult worms on the mucosa of the nasal turbinates of a dog.*

Subsequent to the CT and rhinoscopy, a fecal sample was evaluated by sugar centrifugation flotation. Numerous bioperculate capillarid ova characteristic of *E. boehmi* were identified (**Figure 3**).

The *E. boehmi* infection was treated with fenbendazole at 50 mg/kg PO *q* 24 hr for 2 wk. This treatment completely resolved the patient's nasal discharge; however, the clinical signs recurred a few weeks later, concurrently with the owner's discovery that the dog had been eating its own feces in the back yard, presumably causing reinfection. A second course of fenbendazole at the same

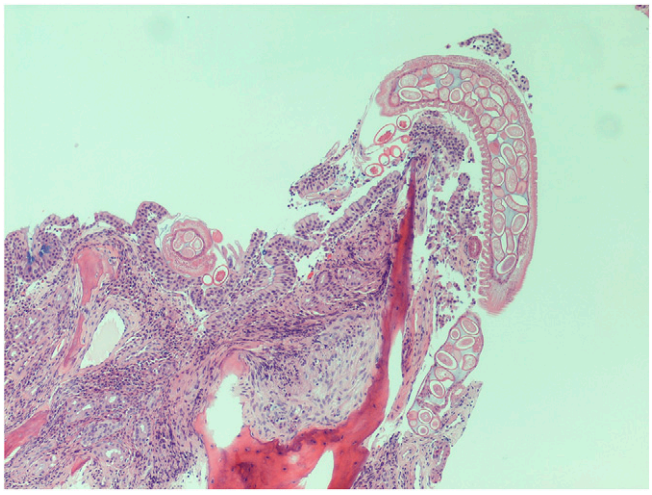

FIGURE 2 *Biopsy from the nasal mucosa stained with hematoxylin and eosin. Adult worms are present on the mucosa, which shows a marked plasmacytic inflammatory response with occasional eosinophils.*

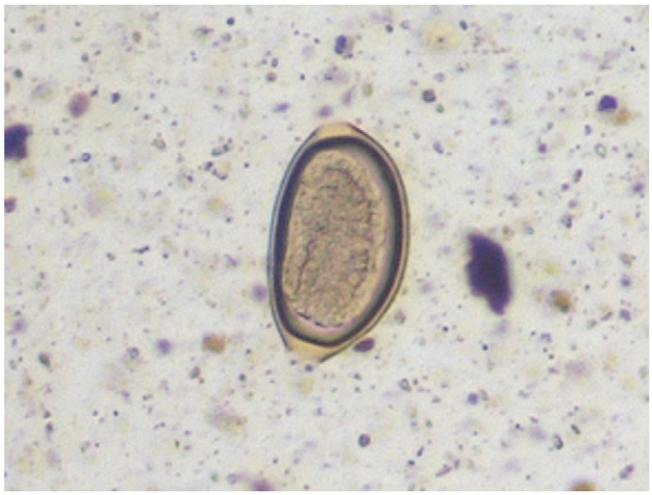

FIGURE 3 Eucoleus boehmi *ova in the feces of the dog performed via the sugar centrifugation flotation technique.*

dose again resolved the clinical signs. In addition, the owner prevented further coprophagia and follow-up fecal examinations remained negative. Clinical signs of nasal disease did not recur in the 24 mo posttreatment period.

Discussion

E. boehmi is an uncommonly diagnosed parasitic infection. Diagnosis is achieved by detection of adult worms or ova during cytologic evaluation of nasal flushes, detection of ova in the feces, or on post mortem evaluation of the nasal cavity.[1–5] Prevalence of infection based on a fecal flotation survey of 6,458 canines in the United States revealed 0.4% to be positive for *E. boehmi* ova.[3] A survey of 230 greyhound dogs in Kansas revealed 2% positive fecal samples.[5] Little is known about the distribution of *E. boehmi* infection in canids in North America but positive samples were recorded in each of the regions sampled (i.e., Northeast, Southeasth, West, and Midwest regions).[3] Egg shedding is thought to be cyclical and multiple fecal examinations may be required to detect infection.[5]

The life cycle of *E. boehmi* is not known. Other capillarids and whipworms have a direct life cycle. The presumed recurrence of infection following coprophagia in the case reported here would support a direct life cycle for *E. boehmi* as well.

To the authors' knowledge, this is the first report that describes the appearance of adult *E. boehmi* worms in situ in the live animal. Campbell and Little (1991) described *E. boehmi* infection in two asymptomatic random-source research dogs at necropsy.[2] Sixteen to 88 adult nematode worms were detected within the epithelial lining of the nasal turbinates, the frontal sinuses, and the paranasal sinuses.[2] The gross description of these worms matches the ones found rhinoscopically in the case reported here (i.e., white nematodes measuring 1.5 cm to 4.0 cm in length). In retrospect, two prior case reports describing nasal capillariasis in dogs likely represented *E. boehmi* as well.[1,4]

It can be challenging to differentiate between ova of *E. boehmi* and those of the lung worm *E. aerophilus* (formerly *Capillaria aerophila*) and the intestinal whipworm *Trichuris vulpis*. *E. boehmi* eggs are clear to golden in color, barrel-shaped, and measure 45 μm to 60 μm × 30 μm to 35 μm (Figure 3).[2] Asymmetrically placed at each pole, a small, clear, blister-like prominence extends outward from a 5 μm gap in the shell. The egg surface is marked by delicate pits giving it a porous appearance. This latter feature distinguishes *E. boehmi* from the other capillarid eggs passed in the feces of dogs which have shells with striations rather than pits.[2] In addition, ova from *E. boehmi* have already undergone partial embryonation of the developing larvae and this development causes the enclosed embryo to retract from the shell. By comparison, ova from *Trichuris vulpis* are larger (70 μm to 80 μm × 30 μm to 42 μm) and have a smooth shell surface. In addition, the ova are symmetric with the polar plugs aligned on opposite sides of the egg.

Clinical signs of an *E. boehmi* infection are variable and can depend on the number of adult worms present within the nasal mucosa, which can vary between 3 to 88.[2,5] The dogs described by Campbell and Little (1991) were asymptomatic; however, they were only observed for a few days before necropsy.[2] Four of 230 greyhounds in a racing kennel testing positive for *E. boehmi* ova on fecal examination also did not show any clinical signs.[5] In contrast, another dog diagnosed with *E. boehmi* had a 2 mo history of reddish-brown tenacious unilateral nasal discharge and severe epistaxis requiring a blood transfusion.[4] An additional dog had serous nasal discharge with occasional sneezing initially which, over a period of about 4 mo, progressed to a copious purulent nasal discharge and severe sneezing fits multiple times per day.[1] Like the patient described herein, this latter dog had been treated with antibiotics, antihistamines, and steroids prior to diagnosis of the nasal worm infection.

The nasal mucosal inflammatory response to *E. boehmi* infection has been reported to be predominantly eosinophilic.[1] The case described in our report had only a mild eosinophilic component, and the rhinitis was predominantly plasmacytic. A basophilia was noted on the CBC, which could have been related to the dog's nasal parasite infection or its unrelated skin disease. The histopathologic appearance of nematodes from the *Capillaria* family in tissue sections has been described previously.[4,6] The worms identified in the nasal mucosal biopsy specimens from the patient presented here had a similar appearance.

Infections caused by capillarid nematodes such as *E. boehmi* are not successfully eliminated by regular anthelmintic dosages. A previous report described initial rapid resolution of clinical signs and a negative fecal examination after treatment with fenbendazole for 10 days (50 mg/kg/day).[4] However, the clinical signs and a positive fecal exam returned 4 mo later, at which time the patient was treated with ivermectin (0.2 mg/kg PO). Ivermectin seemed to resolve clinical signs, but the feces remained positive for the next 3 mo despite three additional doses of ivermectin. Potential reinfection by coprophagia after each of these treatments was not considered and the patient was lost to follow-up. This is in contrast to the report by Evinger et al. (1985) in which a single dose of ivermectin (0.2 mg/kg PO) eliminated clinical signs within 7 days and fecal shedding of ova within 14 days.[1] The dog in this latter report remained clinically normal for 8 mo after ivermectin treatment and monthly fecal examinations were negative for ova for up to 4 mo posttreatment. Similar treatment success has been reported using milbemycin oxime (2.0 mg/kg PO).[7]

In the case described in this report, prior treatment with ivermectin (0.2 mg/kg PO) did not eliminate clinical signs. An extended course of fenbendazole for 2 wk relieved clinical signs initially, but the clinical signs returned a few weeks later concomitantly with the owner's discovery that the dog had been eating its own feces in the back yard. A second 2 wk course of fenbendazole was successful when coprophagia was also prevented. A fecal examination following the second treatment was negative for parasite ova. Since then, the dog has remained clinically normal for 24 mo. This finding suggests that *E. boehmi* infections can be successfully treated with a 2 wk course of fenbendazole when combined with removing feces from the dog's environment to prevent reinfection through coprophagia. In addition, periodic fecal rechecks following treatment are advisable to ensure that the parasite has been eliminated. Multiple fecal examinations may be needed to diagnose persistence of infection as shedding of *E. boehmi* ova may be cyclical.[5]

Conclusion

Infection with nasal worms should be considered in the differential diagnosis for chronic serous to mucopurulent nasal discharge. Patients with chronic nasal discharge should have at least one fecal examination performed. This case report illustrates the characteristic rhinoscopic appearance of the nematodes within the nasal mucosa, which can also be diagnostic of *E. boehmi* infection.

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FOOTNOTES

[a] Clavamox; Pfizer Animal Health, Exton, PA
[b] Recombitek C4; Merial, Duluth, GA; and Vanguard Plus 5 L4, Pfizer Animal Health, Exton, PA
[c] Vanguard L4; Pfizer Animal Health, Exton, PA
[d] Vanguard B; Pfizer Animal Health, Exton, PA
[e] IMRAB 3; Merial, Duluth, GA
[f] Heartgard Plus; Merial, Duluth, GA
[g] Ivomec; Merial, Duluth, GA
[h] Acepromazine maleate; Vedco Inc., St. Joseph, MO
[i] Hydromorphone HCl; Elkins-Sinn, Cherry Hill, NJ
[j] Sodium thiopental; Abbott Animal Health, IL
[k] Sevoflo; Abbott Animal Health, IL
[l] Video Gastroscope System GIF-P140; Olympus America Inc., PA
[m] Karl Storz Veterinary Endoscopy, CA
[n] Karl Storz Veterinary Endoscopy, CA
[o] Jarit, Integra Surgical Instruments, NY

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