

Helminth Parasites of the Canine and Feline Respiratory Tract

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KEYWORDS

- *Eucoleus boehmi* • *Eucoleus aerophilus* • *Crenosoma vulpis*
- *Angiostrongylus vasorum* • *Aelurostrongylus abstrusus*
- *Oslerus osleri* • *Filaroides hirthi* • *Filaroides milksi*
- *Paragonimus kellicotti*

The helminth parasites of the respiratory tract of dogs in North America consist of two capillarids (*Eucoleus aerophilus*, *Eucoleus boehmi*), five metastrongyloids (*Angiostrongylus vasorum*, *Crenosoma vulpis*, *Filaroides hirthi*, *Filaroides milksi*, *Oslerus osleri*), and a trematode (*Paragonimus kellicotti*). Those infecting the cat include a capillarid (*E aerophilus*), a metastrongyloid (*Aelurostrongylus abstrusus*), and a trematode (*P kellicotti*). Diagnosis of these parasitic infections is infrequent in most parts of North America. Necropsy data on infection prevalence in North America is lacking for most of the lungworms. The results of fecal flotation examination surveys, some involving thousands of samples, have indicated a low prevalence for the various lungworms.^{1–5} However, as acknowledged by the investigators of the studies, fecal flotation is not the technique of choice for most of the lungworm parasites and therefore these may have been underestimated. Fecal flotation is probably the most widely used diagnostic technique for the detection of parasitic infection in veterinary private practice, and appropriately so.^{6,7} However, veterinary clinicians should bear in mind the limitations of fecal flotation as a diagnostic tool in the detection of operculate eggs and nematode larvae, which complicate the detection of most of the helminth lungworm parasites of dogs and cats. Clinical impressions that helminth parasites play little or no role as causative pathogens in canine and feline respiratory disease in a practice area may be inaccurate if based on fecal flotation as the primary or sole screening technique. The potential for missing lungworm cases that occur, whether they represent a significant number of animals or very few, exists due to the diagnostic challenge involved in the detection of most of these parasites. Never having had a case involving lungworm infection and never having diagnosed one may be two different things.

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CANINE NASAL EUCOLEOSIS/CANINE AND FELINE TRACHEOBRONCHIAL EUCOLEOSIS

Eucoleus aerophilus (= *Capillaria aerophila*) occurs in the trachea, bronchi, and bronchioles, infects dogs, cats, and various wild carnivores, and has a worldwide distribution.⁸ At one time, *E aerophilus* was also thought to sometimes occur in the nasal passages and sinuses of dogs and wild canids, but it is now recognized that this was a second species, *Eucoleus boehmi*.⁹ Interpretation of study results from some of the older literature is complicated by the uncertainty of which of the two species the researchers may have been dealing with.

E boehmi occurs in the nasal passages and sinuses of wild and domestic canids in Europe, North America, and South America.⁹ The worms are long, thin (22–43 mm × 0.08–0.15 mm), and are found embedded in the epithelial lining of the nasal turbinates, frontal sinuses, and paranasal sinuses.^{9,10} The life cycle and routes of transmission are unknown. Earthworms may serve as intermediate hosts but further study is required to confirm this.⁹ Clinically affected dogs show signs of sneezing and mucopurulent nasal discharge that may contain blood.

Fecal flotation survey results usually do not differentiate between capillarid species, therefore little is known on prevalence and distribution of *E boehmi* infection in canids in North America. Only 0.4% of 6458 canine fecal samples tested in a national fecal flotation survey in the United States were positive for capillarid eggs, most of which were *E boehmi*. Positive samples were recorded from each of the regions sampled.⁴ A fecal flotation survey of greyhounds in Kansas detected eggs of *E boehmi* in 2% (4 of 230) of the samples.¹¹ Diagnosis is based on detection of eggs in feces by fecal flotation. Egg shedding may be cyclical, therefore multiple fecal examinations may be needed to detect infection.¹¹ Eggs may also be detected by microscopic examination of nasal discharge. The eggs are bipolar plugged, contain a multicelled embryo, and are 54 to 60 by 30 to 35 microns in size (Fig. 1).^{12,13} The eggs of *E boehmi* resemble those of *Trichuris vulpis* (Fig. 2) and the other capillarids that may be present in canine fecal samples (*Eucoleus aerophilus*, *Pearsonema plica*, and *Callodium hepaticum*).^{12,13} Eggs of *E boehmi* can be differentiated from those of *T vulpis* based on size and morphology. *Trichuris* eggs are 72 to 90 by 32 to 40 microns in size and the shell wall surface is smooth. The bipolar plugs tend to be more prominent, and have ridges that give the appearance that they are threaded into the shell wall. The bipolar plugs of



Fig. 1. *Eucoleus boehmi* egg detected on fecal flotation of a dog (original magnification ×400).



Fig. 2. *Trichuris vulpis* egg detected on fecal flotation of a dog (original magnification $\times 400$).

capillarid eggs lack ridges, and the shell wall surface has a pattern unique for each of the species. The shell surface pattern of *E boehmi* consists of fine pitting (**Fig. 3**).¹³

Treatment using an oral dose of ivermectin at 0.2 mg/kg (this dose is not safe for use in collie-type breeds) appeared to be effective in a naturally infected dog.¹⁴ Similar results were reported using milbemycin oxime (2.0 mg/kg, oral).¹⁵ Failure to control an *E boehmi* infection in 2 dogs has been reported using ivermectin (0.2–0.3 mg/kg, oral) and fenbendazole (50 mg/kg, oral, once a day for 10 days).^{16,17}

Eucoleus aerophilus are long and thin measuring 16 to 40 by 0.06 to 0.18 mm.¹⁰ Reports of prevalence in North America have ranged from 0% to 5% in dogs and 0.2% to 9% in cats.^{1–4} The life cycle is considered direct; however, there is some speculation that earthworms serve as a paratenic or intermediate host.^{9,13} Eggs are long-lived in the environment and the prepatent period has been reported as 40 days.⁸ Infection in dogs and cats is usually well tolerated; however, chronic cough can develop that may also lead to loss of weight and body condition, and rarely ends in death.^{9,13} Definitive diagnosis is by detection of eggs on fecal flotation. The eggs are bipolar plugged and 58 to 79 by 29 to 40 microns in size (**Fig. 4**).¹² The shell wall surface has a series of anastomosing ridges forming a netlike pattern (**Fig. 5**). Eggs may also be detected in bronchoalveolar lavage samples.^{18,19} Other diagnostic



Fig. 3. *Eucoleus boehmi* egg detected on fecal flotation of a dog. The shell wall surface is in focus, showing the finely pitted surface (original magnification $\times 450$).



Fig. 4. *Eucoleus aerophilus* egg detected on fecal flotation of a dog (original magnification $\times 400$).

tests, suggestive of but nonspecific for *E aerophila* infection, include radiographs indicating a diffuse interstitial lung pattern and transtracheal wash cytology showing an eosinophilic inflammatory response.²⁰

Fenbendazole (30 mg/kg, oral, once a day for 2 days repeated every 2 weeks for a total of four treatments) was reported to be safe and effective in the treatment of clinically affected arctic foxes.²¹ Use of fenbendazole (50 mg/kg, oral, once a day for 14 days) in a dog and abamectin (0.3 mg/kg, subcutaneous, repeated in 2 weeks) in a cat were also reported to be effective treatments for *E aerophilus* infection.^{18,19} Anthelmintics with apparent efficacy against *E boehmi* (ivermectin, milbemyxin oxime) may also be useful in cases of *E aerophilus* infection.

CANINE CRENOSOMOSIS (*CRENOSOMA VULPIS*)

Crenosoma vulpis, the fox lungworm, occurs in the trachea, bronchi, and bronchioles of wild and domestic canids in the temperate regions of North America and Europe.²² Crenosomosis has recently been recognized as an important cause of chronic respiratory disease in dogs in parts of Canada and Europe.^{23–25} Adult worms are 5 to 10 mm in length, and the anterior end is marked by a characteristic series of 18 to 26 ringlike cuticular folds.²² In North America, the geographic distribution of *C vulpis*



Fig. 5. *Eucoleus aerophilus* egg detected on fecal flotation of a dog. The shell wall surface is in focus, showing the network of anastomosing ridges (original magnification $\times 400$).

seems to be mainly in the northeastern portion of the continent including parts of the United States and Canada.^{23,25} The North American natural definitive hosts are species of wild canids including foxes and coyotes.^{22,26} Excepting the Atlantic Canadian provinces (New Brunswick, Newfoundland-Labrador, Nova Scotia, Prince Edward Island), infection in dogs seems to be infrequent in North America. There are several case reports involving *C vulpis* infection in dogs in New York, Quebec, and Ontario.²⁷⁻²⁹ In Atlantic Canada, crenosomosis has been found to be a frequent cause of chronic respiratory disease in dogs with *C vulpis* infection, occurring in 21% of dogs showing signs of chronic cough.²⁵ Canids acquire infection by the ingestion of terrestrial snail and slug gastropod intermediate hosts.²³ The prepatent period is 19 to 21 days, and the adult worm life-span is about 10 months.²⁵ Infection induces chronic bronchitis-bronchiolitis, which results in clinical signs consisting primarily of chronic cough sometimes accompanied by gagging.³⁰ Definitive diagnosis is by detection of first-stage larvae in feces or transtracheal wash samples. Larvae are detected in feces by Baermann examination, ZnSO₄ centrifugal flotation (CF), or FLOTAC device. The Baermann technique seems to be the most sensitive method for diagnosis.^{24,25} Crenosomosis was diagnosed in a dog in Italy using the FLOTAC device, recently developed and available in Europe.³¹ This method was considered superior in larval recovery when compared with the Baermann technique; however, this was based on the examination of fecal samples from a single dog. As with other metastrongylids, fecal larval shedding may be intermittent and appears to become more so on reinfection.³² Therefore, examination of multiple fecal samples (three collected over 7 days) may increase detection sensitivity.²³ Larvae are 264 to 340 by 16 to 22 microns in size (**Fig. 6**).¹² There is a narrowing at the anterior-end of the larva (= cephalic button) and the tail lacks a kink or dorsal spine, but has a slight deflection that is best seen in a lateral view of a larva that has been killed with iodine, heat, or formalin.¹² Febantel (14 mg/kg, oral, once a day for 7 days), fenbendazole (25 to 50 mg/kg, oral, once a day for 3 to 14 days), and milbemycin oxime (0.5 mg/kg, oral, single dose) have all been used to treat dogs naturally infected with *C vulpis*, with a clinical cure occurring within 7 to 10 days of treatment.^{23-25,27-30,33,34} A treatment efficacy of 98% to 99% was reported for milbemycin oxime (0.5 mg/kg, oral) used in the treatment of dogs experimentally infected with *C vulpis*.³⁴ Crenosomosis may be misdiagnosed as allergic respiratory disease, and dogs will show a positive clinical response due to the symptomatic relief of corticosteroid therapy.³⁰



Fig. 6. First-stage larvae of *Crenosoma vulpis* recovered on Baermann examination of a canine fecal sample (original magnification $\times 200$).

CANINE VERMINOUS NODULAR BRONCHITIS (*OSLERUS OSLERI*)

Oslerus osleri is a parasite found in the trachea and bronchi of dogs, dingoes, coyotes, and wolves,²² and has a worldwide distribution. In North America, infection is fairly common and widespread in wild canids, particularly coyotes.²² However, wild canids do not seem to serve as an infection reservoir for dogs; dogs exposed to infective larvae derived from coyotes failed to develop *O osleri* infections.³⁵ Infection in dogs is infrequent, but isolated cases have been reported throughout the United States and Canada.³⁶

Adult worms are 6.5 to 13.5 mm long, and reside coiled inside wartlike nodules that are attached to the mucosal epithelium in the lumen of the trachea and bronchi.³⁷ The nodules are clustered at the bifurcation of the trachea. Individual nodules range in size from 1 to 20 mm and can become confluent when present in large numbers.^{38,39} Nodules from naturally infected coyotes contained 1 to 105 worms per nodule.⁴⁰

Atypical for metastrongyloid parasites, the life cycle for *O oslerus* is direct and the first-stage larva is the infective stage. Adult females lay thin-shelled larvated eggs (80 × 50 microns) that hatch, and the first-stage larvae migrate up the bronchial system to pass either in saliva or in the feces. Larvae recovered from the feces tend to be sluggish, and are often found to be dead and degenerating. Transmission in wild canids is thought to occur mainly by exposure of weanling pups by the dam through regurgitative feeding.⁴¹ Transmission in dogs is thought to be mainly through saliva from the dam cleaning her pups through licking. Exposure can also be through ingestion of larvae from fecal contamination, but this is of lesser importance.^{37,41} Immature worms arrive in the trachea about 70 days after exposure, and nodules are visible soon after. The prepatent period is about 92 to 126 days.^{41,42}

Diagnosis of infections tends to occur in young dogs, 6 months to 2 years old,^{39,43,44} which is consistent with exposure at an early age. Clinical signs consist of chronic cough, which may be worse with exercise. In some cases wheezing and dyspnea occur.⁴³ Weight loss, emaciation, and collapse may be observed in the most severely affected dogs.⁴⁵ Pneumothorax was reported in one case of *O osleri* infection.⁴⁶ Infections may be subclinical in some dogs.

Definitive diagnosis is by visualization of the nodules at the bifurcation of the trachea with bronchoscopy followed by recovery of first-stage larvae in bronchial mucus, or less commonly, in feces. Larvae recovered from bronchial mucus are 233 to 267 microns in length, and the tail ends in a distinctive sinus wave-shaped kink.³⁷ Transtracheal wash samples or bronchial mucus collected during bronchoscopy are superior for larval recovery to fecal detection. Zinc sulfate centrifugal fecal flotation (ZnSO₄ CF) has greater detection sensitivity than Baermann examination; however, false-negative results are a problem with both methods.⁴⁴ Larvae when recovered from feces are 326 to 378 microns in size (Fig. 7).³⁷ Evidence of tracheobronchial nodules may also be detected by radiographs in some cases.⁴³

Fenbendazole and ivermectin have been used in naturally infected dogs, with variable results.^{43,44,47} Fenbendazole (50 mg/kg, oral, once a day for 7 to 14 days) was reported to be effective in the treatment of 20 dogs with clinical *O osleri* infections.⁴³ One severely affected dog required two 14-day courses of the fenbendazole treatment. Ivermectin (0.4 mg/kg, subcutaneous, repeated every 3 weeks for four treatments) was reported to be effective in the treatment of four dogs, resulting in a clinical cure and resolution of tracheobronchial nodules.⁴⁷

CANINE VERMINOUS PNEUMONIA (*FILAROIDES* SPP)

There are two closely related species, *Filaroides hirthei* and *Filaroides milksi*, occurring in the lung parenchyma of dogs.²² *Filaroides milksi* (= *Andersonstrongylus milksi*) was



Fig. 7. First-stage larvae of *Oslerus osleri* detected on $ZnSO_4$ centrifugal flotation of a fecal sample from a coyote (original magnification $\times 400$).

first reported as an incidental finding from the necropsy of a 10-year-old Boston terrier.⁴⁸ Adult worms (3.4 to 10.9 mm in length) were found in bronchioles and coiled in nests in the lung parenchyma. *Filaroides hirthi* was first reported, also as an incidental finding at necropsy, in the bronchioles and lung parenchyma of purpose bred research beagles.^{49,50} Adult worms are 2.3 to 13 mm in length. The two species are differentiated from each other based on subtle differences in adult worm size, and male spicule morphology and length.⁵¹ The validity of *F milksi* and *F hirthi* as two separate species has been questioned, resulting in some debate.^{51–53} Prevalence of *F hirthi* infection as high as 78% in individual research dog colonies has been reported.⁵⁴ Diagnosis is rare in nonresearch colony dogs. Infections in client-owned dogs in the United States have been reported in Alabama, Georgia, New York, Pennsylvania, Texas, and Washington.^{55–60}

There are fewer reports of *F milksi* infection. Diagnosis in dogs based on histopathology has been reported in Australia, Canada, and the United States; however, differentiation between *F hirthi* and *F milksi* is not possible based on histopathology and therefore, these may have been *F hirthi*.^{49,53} In addition to the original species description, there is only one other report of a diagnosis in a dog based on identification of adult male worms.⁶¹ Reports of *F milksi* infection in the skunk and in a dog from Belgium have been disputed.⁵³

The life cycle is unknown for *F milksi*. Transmission of *F hirthi* occurs by ingestion of infective L1 larvae, usually through coprophagia of fresh fecal material. In research beagle colonies this is thought to occur in puppies by 4 to 5 weeks of age through exposure to feces from infected dams.⁶² The prepatent period is 35 days.⁶³ Infections appear to be long-lived, and this is probably due to reexposure to infective first-stage larvae through autoinfection.

Most infections appear to be subclinical. *F hirthi* infection in research dogs can compromise or invalidate study results depending on the nature of the project.⁴⁹ Studies involving immunosuppression may induce a fatal hyperinfection.⁶⁴ Fatal hyperinfection secondary to immunosuppression or some predisposing state of stress has also been reported in client-owned dogs. Long-term corticosteroid therapy, neoplasia, severe trauma, and distemper infection have been cited as predisposing factors.^{55,56,59,60,65} Most reports involve young (<3 years), small toy breeds such as Chihuahua, West Highland terrier, toy poodle, and Yorkshire terrier. Fatal infections have also occurred in dogs up to 10 years old and in such breeds as the King Charles spaniel and Dalmatian.^{58,59} Clinically affected dogs show signs of dyspnea, cough, and cyanosis, and may be depressed. Diagnosis is by detection of first-stage larvae

in bronchial mucus or feces. The larvae of *F hirthi* and *F milksi* cannot be differentiated from each other or those of *O osleri*. Larvae are 240 to 290 microns in length and have a kinked tail.⁶⁶ Also in common with *O osleri*, detection sensitivity of *Filaroides* larvae by Baermann examination is poor.⁶³ Larvae are best detected by examination of bronchial mucus. Fecal detection is best achieved by ZnSO₄ CF; however, false-negative results are common.⁶³ Additional diagnostics might include radiographs, showing interstitial linear and focal nodular pulmonary infiltrates.⁶⁷

Albendazole, fenbendazole, and ivermectin have been used to treat dogs infected with *F hirthi*. Control in research dog colonies by treating breeding animals using albendazole (25 mg/kg, oral, twice a day for 5 days; repeated in 2 to 4 weeks) and ivermectin (1 mg/kg, subcutaneous, repeated in 1 week) has been reported.^{54,68} Fenbendazole (50 mg/kg, oral, once a day for 14 to 21 days or 100 mg/kg, oral, once a day for 7 days) appeared to be an effective treatment in three dogs.^{57,69,70} Corticosteroids were used in one of the dogs as an adjunct therapy due to severe posttreatment dyspnea that was attributed to an inflammatory response to dead worms.⁵⁷ Ivermectin (0.034 mg/kg, oral, single dose) followed by fenbendazole (50 mg/kg, oral, once a day for 14 days) appeared to be an effective treatment in one dog.⁶⁰

FELINE AELUROSTRONGYLOSIS (*AELUROSTRONGYLUS ABSTRUSUS*)

Aelurostrongylus abstrusus occurs in the terminal respiratory bronchioles and alveolar ducts in the lung parenchyma of domestic cats, and has a worldwide distribution.²² In North America it has been reported in the United States in the eastern (Connecticut, New York, Maryland, New Jersey, Pennsylvania), southeastern (Alabama, Georgia, North Carolina, South Carolina, Tennessee, Virginia), southwestern (Texas), and west coast (California, Oregon, Washington) states, and Hawaii.^{1,2,5,36,71} In Canada, it has been diagnosed in cats in Ontario, Newfoundland-Labrador, and Nova Scotia⁷² (Gary Conboy, DVM, unpublished data, 2003). Fecal flotation surveys have indicated *A abstrusus* infection rates in cats of 0.1% to 1.1%.^{1,2,5} A Baermann fecal examination survey found 18.5% prevalence in cats in Alabama.⁷³ Results of experimental infections have indicated that dogs are not a susceptible host.⁷⁴ Coprophagia rather than patent infection may explain the occasional finding of *A abstrusus* larvae in canine fecal surveys.

Cats acquire infection by the ingestion of infective third-stage larvae contained in terrestrial gastropod intermediate hosts (slugs, land snails) or a wide range of paratenic hosts (amphibians, reptiles, birds, small rodents).⁷¹ Adult worms are 4 to 10 mm in length.⁷⁴ Mature females produce undifferentiated eggs, which develop and hatch first-stage larvae. The larvae are coughed up, swallowed, and passed in the feces. The prepatent period is about 5 to 6 weeks, and infected cats shed L1 larvae in the feces for a period that usually lasts 2 to 7 months with a peak in shedding 10 to 17 weeks after infection.^{71,75,76} In some cats, the period of larval shedding may last 1 to 2 years.^{75,76} There is a delayed onset of patency, less larval shedding, and a more erratic shedding pattern after reexposure in cats that have been infected previously.^{75,76}

Infections are usually subclinical.^{71,77} Heavy infections can result in severe, potentially fatal, respiratory disease. Severe clinical disease was reproduced experimentally in kittens given 800 L3 larvae, with cough developing 6 weeks after exposure.⁷⁸ Clinically affected cats often show signs of cough, dyspnea, and fever, and may suffer anorexia and emaciation. As with *C vulpis* infection in dogs, *A abstrusus* infection may be misdiagnosed as allergic respiratory disease, and show a positive response to administration of corticosteroids and bronchodilators.⁷⁹ Infection occurs more

often in younger cats (3 months to 3 years) and outdoor cats.^{77,80} Pneumothorax and pyothorax secondary to *A abstrusus* infection has been reported in a kitten. It was speculated that third-stage larvae became contaminated with *Salmonella typhimurium* in the lumen of the intestine and carried it to the lungs.⁸¹

Diagnosis is by detection of L1 larvae in feces, bronchial mucus, or pleural fluid. False-negative results in larval detection can occur due to sporadic shedding patterns.⁷¹ Fecal detection occurs by Baermann examination, fecal flotation, direct smear, and FLOTAC device. The Baermann is considered the most sensitive method for larval detection.⁷³ The FLOTAC device was considered more effective in larval recovery than the Baermann technique when compared on samples collected from a single *A abstrusus*-infected cat.⁸² The larvae are 360 to 400 microns in length, and the tail ends in a distinctive sinus wave-shaped kink with a dorsal spine (Fig. 8).¹² A nested polymerase chain reaction assay for *A abstrusus* infection used on Baermann sediment, feces, and pharyngeal swabs has recently been developed in Europe, and shows great promise; it had a reported specificity of 100% and sensitivity of 96.6%.⁸³ Additional diagnostic testing options would involve radiography, transtracheal wash, and bronchoalveolar lavage. Radiographic changes tends to show a mixed pattern, with an alveolar pattern predominating during the period of heaviest larval shedding (5 to 15 weeks post-infection) followed by bronchial and interstitial patterns.⁸³ Computed tomography images may also be useful in assessing lesions in *A abstrusus*-infected cats.⁸⁴

Options currently available for treating cats infected with *A abstrusus* include abamectin, fenbendazole, ivermectin, moxidectin, and selamectin. One to two applications of selamectin (6 mg/kg, topical) were reported to be effective in the treatment of 1 of 3 cats.⁷⁹ Ivermectin (0.4 mg/kg, subcutaneous, repeated in 2 weeks) has been reported to be effective in the treatment of several cats.^{85–87} Fenbendazole (20 mg/kg, oral, once a day for 5 days or 50 mg/kg, oral, once a day for 15 days) was reported to be effective in the treatment of *A abstrusus* infection in cats.^{79,88} One to three topical applications of 1 mg/kg moxidectin (in combination with imidacloprid) appeared to be effective in the treatment of eight cats infected with *A abstrusus*.⁸⁹ Abamectin (0.3 mg/kg, subcutaneous, repeated in 2 weeks) appeared to be effective in the treatment of one cat.⁹⁰



Fig. 8. First-stage larvae of *Aelurostrongylus abstrusus* recovered on Baermann examination of a feline fecal sample (original magnification $\times 200$).

CANINE ANGIOSTRONGYLOSIS (*ANGIOSTRONGYLUS VASORUM*)

Angiostrongylus vasorum, the French Heartworm, is a metastrongyloid that occurs in the pulmonary arteries and right heart of wild and domestic canids in Europe, Africa, and South America, and in a single focus in North America in Canada (Newfoundland-Labrador).^{22,25,91} The natural definitive hosts are various species of foxes. The risk of infection to dogs in North America is currently restricted to those animals living within this small endemic range. However, recent studies have indicated an alarming trend toward expansion in the geographic distribution of *A. vasorum*, and an increased exposure risk of infection to dogs within the various endemic ranges.⁹² Given the ease and frequency of travel within North America coupled with the presence of a large red fox population and the abundance of gastropod intermediate hosts, it seems highly likely that the endemic range of *A. vasorum* will spread from Newfoundland to other parts of North America. Canids acquire infection by the ingestion of L3 larvae contained in intermediate hosts (terrestrial gastropods, frogs). The prepatent period is 28 to 108 days.⁹³ Adult worms are 14 to 20.5 mm in length (about one-tenth the size of *Dirofilaria immitis*) and males are bursate.⁹¹ Infections result in potentially fatal cardiopulmonary disease with clinical signs consisting of chronic cough, dyspnea, exercise intolerance, anorexia, gagging, and weight loss.^{91,93} Secondary coagulopathies (disseminated intravascular coagulation, immune-mediated thrombocytopenia) can also occur, resulting in subcutaneous hematomas or occasionally in fatal cerebral, spinal, or abdominal hemorrhage. Ascites, syncope, vomiting, and signs of central nervous system disease may also occur. On rare occasions sudden death after an acute onset of clinical disease can occur, usually in younger dogs.^{91,93} Definitive diagnosis is by the detection of L1 larvae in feces or bronchial mucus.^{91,94} Larvae are 310 to 399 microns in length and have a cephalic button at the anterior end, and the tail terminates in a sinus wave-shaped kink with a dorsal spine (Figs. 9 and 10).¹² The method of choice for fecal detection of L1 larvae is the Baermann technique.^{25,93} Although not yet commercially available, a sandwich enzyme-linked immunosorbent assay detecting circulating antigens of *A. vasorum* has recently been developed, and shows promise as a diagnostic test. A test specificity of 100% and sensitivity of 92% was reported.⁹⁵ The presence of radiographic changes, reduced serum levels of fructosamine, or calcemia may also aid in diagnosis.⁹³



Fig. 9. First-stage larvae of *Angiostrongylus vasorum* recovered on Baermann examination of a canine fecal sample (original magnification $\times 200$).



Fig. 10. First-stage larvae of *Angiostrongylus vasorum* recovered on Baermann examination of a canine fecal sample. Close-up view of the tail morphology showing the kink and dorsal spine (original magnification $\times 450$).

Fenbendazole, ivermectin, milbemycin oxime, and moxidectin have all been used to treat angiostrongylosis in dogs, with apparent success. Irrespective of the choice of anthelmintic, posttreatment complications that may involve severe dyspnea or ascites can occur.⁹¹ Administration of bronchodilators and diuretics are indicated in these cases. Fenbendazole (20 to 25 mg/kg, oral, once a day for 20 to 21 days or 50 mg/kg, oral, once a day for 5 to 21 days) has been widely used in naturally infected dogs.⁹³ Milbemycin oxime (0.5 mg/kg, oral) given once a week for 4 weeks has also been used in naturally infected dogs.²⁵ The same therapeutic protocol used to treat dogs experimentally infected with *A. vasorum* had an efficacy of 85%. This study also reported an efficacy of 85% when experimentally infected dogs were given two doses of milbemycin oxime (0.5 mg/kg) at 1 month and 2 months after exposure (ie, as used in *Dirofilaria immitis* prevention).⁹⁶ A single topical application of moxidectin (2.5 mg/kg) was used to treat naturally infected dogs and an efficacy of 85% was reported.⁹⁷

LUNG FLUKE (*PARAGONIMUS KELLICOTTI*)

Paragonimus kellycotti is a trematode that occurs in the lung parenchyma infecting dogs, cats, pigs, goats, and various wildlife species in an endemic area that includes much of the eastern half of North America.^{36,98} Infections are most common in the north-central and southeastern states of the United States.⁹⁹ Fecal examination surveys have indicated a low prevalence of infection (<1%) however, these results are likely an underestimate due to suboptimal detection sensitivity of the flotation technique for fluke eggs.²⁻⁴ Infection with *P. kellycotti* was found to be the cause of disease in 8% (3 of 37) of cats showing signs of chronic respiratory disease in Louisiana.¹⁰⁰

Adult flukes are 10 to 13 by 4 to 6 mm in size, occur inside capsules situated in the lung parenchyma, and rarely occur in other tissues.^{99,101} These flukes are easily differentiated from the nematode lungworms of dogs and cats by the body shape and presence of oral and ventral suckers. Capsules are 2 to 5 cm in diameter with walls 1 to 4 mm thick, usually contain two or more flukes, and are connected to the bronchioles.¹⁰² Capsules occur most often in the caudal lung lobes (right > left). Eggs passed in feces that are deposited into water develop and hatch ciliated miracidium, which



Fig. 11. *Paragonimus kellicotti* egg detected on fecal sedimentation of a feline fecal sample (original magnification $\times 400$).

infect the first intermediate host, aquatic snails (*Pomatiopsis lapidaria*; *Pomatiopsis cincinnatiensis*).^{99,103} Animals acquire infection by the ingestion of metacercariae contained in the tissues of the second intermediate host, crayfish (*Cambarus* spp, *Orconectes* spp). Prevalence of infection in crayfish can be as high as 94% in a stream in the late summer peak period.¹⁰⁴ In addition, rodents preying on infected crayfish can serve as paratenic hosts.¹⁰⁵ The prepatent period is 5 to 7 weeks. Infections have been reported to last as long as 4 years.¹⁰⁶

The most common clinical sign of infection is cough that is sometimes accompanied by sneezing, exercise intolerance, hemoptysis, and dyspnea.^{99,105} Infections can be subclinical to fatal. Subclinical and clinical pneumothorax may develop due to the rupture of the fluke capsule through the pleura, allowing air to pass from the bronchial system to the pleural space. Infected animals may suffer chronic cough for prolonged periods or die acutely, with no history of clinical disease.^{99,107}

Definitive diagnosis is by detection of the distinctive operculate eggs of *P. kellicotti* in feces or bronchial mucus. Fecal detection is best achieved through sedimentation.^{12,108} Eggs may be found by fecal flotation; however, detection sensitivity in samples with low levels of eggs is poor. The eggs are 75 to 118 by 42 to 67 microns in size, yellow-brown in color, and have an operculum at one end (**Fig. 11**).¹² The eggs can be differentiated from those of other trematode or pseudophyllidean tapeworms by the thickened ridge in the shell wall highlighting the opercular line.¹² In addition, fluke capsules can be visualized radiographically as multiloculated cystic structures 2 to 5 cm in size in dogs.^{102,109} Lesions in cats are smaller and have a greater density.^{102,108}

Current treatment options include extra-label usage of albendazole, fenbendazole, or praziquantel. Albendazole (25 mg/kg, oral, twice a day for 14 days), fenbendazole (50 mg/kg, oral, once a day for 10 to 14 days), and praziquantel (23 mg/kg, oral, three times a day for 3 days) are recommended as effective in the treatment of *P. kellicotti* infected dogs and cats.⁹⁸

SUMMARY

The helminth parasite infection of the canine and feline respiratory tract, excepting aelurostrongylosis in cats in the southeastern United States, crenosomosis in dogs in Atlantic Canada and eucoleosis in dogs and cats throughout North America, is

uncommon. As such, a veterinary clinician may be hesitant to include several fecal examination methods (fecal flotation: *E boehmi*, *E aerophilus*; ZnSO₄ CF: *O osleri*, *F hirthi*, *F milksi*; Baermann examination: *C vulpis*, *A vasorum*, *A abstrusus*; sedimentation: *P kellicotti*) when conducting a diagnostic investigation in cases involving animals with respiratory disease. However, these techniques are inexpensive, noninvasive, and if positive they indicate a clear course of action. An argument could be made, even in areas where prevalence seems to be low, for the inclusion of at least one of the aforementioned tests (ZnSO₄ CF) to be included as part of the baseline data collection in the diagnostic workup of all cases involving respiratory disease.

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