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Canine Angiostrongylosis (French Heartworm) (30-May-2000)

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Introduction

Angiostrongylus vasorum, the French Heartworm, is a metastrongyloid nematode parasite that infects the pulmonary arteries and right ventricle of wild and domestic canids. It was first recovered from a dog at necropsy in 1853 in Toulouse, France by Serres but was not described until 1866 by Baillet [1]. Infections in dogs tend to be chronic (months to years) and range from subclinical to fatal in their effects upon the host.

Morphology

Adult worms can be detected at necropsy by careful gross examination of the lumen of the branches of the pulmonary artery and sometimes the right ventricle. The small slender worms are pinkish colored and approximately 14.0-20.5 x 0.170-0.306 mm in size [2]. Males are bursate (Fig. 1); females have a "barber pole" appearance (similar to *Haemonchus*) due to the red intestine intertwined with the white reproductive tract (Fig. 2) [3]. Females produce and lay undifferentiated eggs which develop and hatch first-stage larvae which are passed to the outside in feces. First-stage larvae are 310-399 x 14-16 microns in size [1, 4]. They have an anterior cephalic button and the tail terminates in a sinus wave curve ("severe kink") with a dorsal spine (Fig. 3, Fig. 4).



Figure 1. Adult male *A. vasorum* in lactophenol clearing solution. The bursa, spicules and bursal rays are visible. - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 2. Adult female *A. vasorum* in lactophenol clearing solution. Note the loops of uterus wrapped around the intestine giving the "barber pole" appearance. - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 3. First-stage larvae of *A. vasorum* (killed and stained with a drop of iodine) recovered from the feces of a dog using the Baermann technique. Note the dorsal spine and kink in the terminal portion of the tail. - To view this image in full size go to the IVIS website at www.ivis.org . -



Figure 4. Tail of *A. vasorum* first-stage larvae (iodine stained) showing the distinctive tail morphology (severe kink and a small dorsal spine). - To view this image in full size go to the IVIS website at www.ivis.org . -

Host Range/Geographic Distribution

The natural definitive hosts are various species of wild foxes. Natural infections have been reported in red fox (*Vulpes vulpes*), African desert fox (*Fennecus zerda*), crab-eating zorros (*Cerdocyon thous*), hoary fox (*Pseudalopex vetulus*) and domestic dogs [1, 4, 5]. Patent experimental infections were established in the jackal (*Canis aureus*) and the Nile rat (*Arvicanthus niloticus*). Worms reached maturity, but no larvae were produced, in experimentally infected cats [4]. *Angiostrongylus vasorum* occurs in discrete endemic pockets in various parts of Europe (France, Denmark, England, Germany, Ireland, Italy, Spain, Switzerland), Africa (Uganda), Turkey, the countries of the former USSR, South America (Brazil, Columbia), and Canada (Newfoundland) [1,4-12]. Diagnoses based solely on identification of first-stage larvae on fecal examination have been reported in Argentina, Australia, and Greece [13-15].

Life Cycle

Wild foxes serve as infection reservoirs for domestic dogs. Canids acquire infections by the ingestion of gastropod intermediate hosts. Natural infections of the slug *Arion ater* with *A. vasorum* have been reported in France [16]. A wide variety of molluscs including slugs (*Arion lusitanicus*, *A. hortensis*, *Deroceras reticulatum*, *Limax flavus*, *Laevicaulus altes*), terrestrial snails (*Achatina fulica*, *Arianta arbustorum*, *Bradybaena similaris*, *Cepaea nemoralis*, *Cochlodina laminata*, *Eceparrypha physana*, *Helix pomatia*, *H. aspersa*, *Prosoppeas javanicum*, *Subulina octona*, *Succinea putris*) and aquatic snails (*Biomphalaria glabrata*, *B. pfeifferi*, *Physa* sp.) have been experimentally infected with *A. vasorum* [1, 2, 16-18]. Gastropods are exposed to first-stage larvae of *A. vasorum* through feeding on the feces of infected canids. The canid definitive host may also acquire infections through ingestion of frog paratenic hosts containing infective third-stage larvae [19]. Larval shedding in feces from infected dogs in France has been reported to temporarily cease from November to January [4].

Third-stage larvae are digested free of intermediate/paratenic host tissue, penetrate the gut wall, develop in abdominal lymph nodes before travelling via portal circulation to the liver and then to the pulmonary arteries and right ventricle. Worms mature, mate and produce eggs which lodge and develop in lung capillaries. Larvae hatch from the eggs, breakout into airspace, are coughed-up and swallowed to be passed in the feces of infected animals [1,17]. The prepatent period is about 28-108 days [20]. Adult worm lifespan is approximately equal to that of the canid host; animals shed larvae intermittently in the feces for the rest of their lives. Dogs have been reported to shed as many as 280,000 larvae/g feces [21].

Pathobiology

Immature worms reach the pulmonary artery by 10 days post-infection [1]. The resulting inflammatory response with the subsequent involvement of the surrounding lung parenchymal tissue leads to foci of interstitial pneumonia. Pulmonary hemorrhage occurs with the onset of patency as larvae migrate into airspaces. Granulomas develop in response to eggs and larvae. Fibrosis occurs in pulmonary lesions with chronic infection [17]. The development of emphysema has been reported in some cases [4]. As with *Dirofilaria immitis*, pulmonary vascular lesions include thromboarteritis and intimal proliferation. The consequent pulmonary hypertension can lead to right congestive heart failure [4, 17].

Coagulopathies have been reported in both experimental and natural infections [7, 21-26]. Disseminated intravascular coagulation and immune-mediated thrombocytopenia have been reported [26, 27]. Anemia, melena, haemoptysis, and subcutaneous hematomas have all been associated with *A. vasorum* infection in dogs [4]. Prolonged clotting times and bleeding disorders may occur singly or be accompanied by signs of respiratory and/or cardiac disease. Subdural hemorrhage was posed as a possible etiology for the neurological signs observed in one naturally infected dog [23].

Aberrant migrations of adult worms (eye, left ventricle, femoral artery) and first-stage larvae (brain, spinal chord, eye, kidney, liver, skeletal muscle, intestine, stomach, pancreas, spleen, adrenal gland, thyroid gland) have been reported [4, 28-31]. Presumably, the worms gain entry to the left-side of the heart and are transported to various tissues via the systemic circulation.

Clinical Signs

Clinical signs can be variable. Infections are usually characterized by a gradual onset of progressively worsening signs of respiratory and/or cardiac disease. Chronic cough, dyspnea, exercise intolerance, anorexia, gagging and weight loss are the most common clinical signs of infection. Subcutaneous swellings

(hematomas), ascites, syncope, vomiting and signs of central nervous system disease may also occur. In rare cases, usually in younger dogs, an acute onset of illness followed by sudden death can occur. Occlusion of the pulmonary artery, acute onset of right-sided congestive heart failure and rupture of the femoral artery have been cited as the cause of death in these animals [21, 30, 32, 33].

Diagnosis

Diagnosis is based on history, clinical signs and the detection of first-stage larvae in fecal or transtracheal wash samples. Larvae are detected in feces using the Baermann technique or fecal flotation. Larvae are less reliably detected on fecal flotation and may be difficult to identify due to osmotic damage from exposure to the high specific gravity of the flotation media. The larvae are 310-399 x 14-16 microns in size. They have an anterior cephalic button and the tail terminates in a sinus wave curve ("severe kink") with a dorsal spine (Fig. 3, Fig. 4). Immobilizing larvae with mild heat or the addition of a drop of iodine allows the diagnostician to perform a detailed morphological evaluation and obtain accurate size measurements. Multiple fecal samples may need to be examined to detect larvae due to intermittent shedding.

Radiographs and hematological changes are nonspecific but may be supportive of a diagnosis of angiostrongylosis in cases where fecal examinations are inconclusive. Radiographic changes include a diffuse increase in peribronchial, interstitial and alveolar densities. Multiple nodular like densities may occur in the peripheral regions of the caudal lung lobes. Right-heart and pulmonary artery enlargement may also be visible [7, 21, 23, 34-35]. Hematological changes are inconsistent but may indicate anemia and monocytosis.

Treatment/Control

There are no anthelmintics approved for use in the treatment of angiostrongylosis in dogs. Levamisole, fenbendazole and ivermectin have been used successfully to treat dogs infected with *A. vasorum* [7, 21, 23]. The use of levamisole in dogs for the treatment of angiostrongylosis should be discontinued due to a low therapeutic index, potential side-effects and a presumed increased risk of post-treatment reactions due to its rapid killing rate of the worms. Release of worm antigen causing an anaphylactic reaction was thought to be the cause of hypovolemic shock in one *A. vasorum* infected dog after levamisole treatment [36]. Two doses of ivermectin (0.2 mg/kg) given subcutaneously one week apart has been reported to be effective for treatment of *A. vasorum*. Fenbendazole has been used at a dosage regimen of 20 mg/kg, per os, given 1-2 times daily for 2-3 weeks. Severe dyspnea and ascites may occur as post-treatment complications. Broncho-dilators, expectorants and diuretics may need to be administered due to these post-treatment reactions [4]. Currently, premise application of molluscicides or fencing designed to exclude wild canids are not available as practical control options. It is unknown whether any of the preventative anthelmintics designed for the prevention of *Dirofilaria immitis* infection in dogs would also offer protection against *A. vasorum*. Sanitation to limit exposure of gastropods to feces of infected animals may prevent the spread of infection within a kennel or multi-dog household. However, wild canids probably represent an infection reservoir beyond the effects of human control measures.

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