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**Objective**—To determine clinical signs and clinicopathologic abnormalities in dogs with naturally occurring clinical spirocercosis.

**Design**—Retrospective case series.

**Animals**—39 dogs with spirocercosis.

**Procedures**—Medical records were reviewed, and information on signalment, residence (rural vs urban), owner complaints, physical examination findings, clinicopathologic abnormalities, radiographic and endoscopic findings, and concurrent systemic diseases was recorded.

**Results**—Hellenic hounds and mixed-breed dogs were overrepresented, compared with a group of 117 control dogs without spirocercosis that were examined because of gastrointestinal tract disease, and mean body weight of dogs with spirocercosis was significantly higher than mean body weight of control dogs. Odynophagia (34 [87%]), regurgitation (24 [62%]), and excessive salivation (14 [36%]) were the most common clinical findings. The most common radiographic abnormalities were a mass in the caudodorsal aspect of the mediastinum (15/35 [43%]) and spondylitis of the caudal thoracic vertebrae (10 [29%]). Parasitic nodules were seen during esophagoscopy in all 39 dogs. Normocytic, normochromic, nonregenerative anemia; neutrophilic leukocytosis; hyperproteinemia, and high alkaline phosphatase activity were more common in dogs with spirocercosis than in a control group of 56 healthy dogs. Concurrent systemic diseases, mainly leishmaniosis, dirofilariosis, and monocytic ehrlichiosis, were documented in 14 (36%) dogs.

**Conclusions and Clinical Relevance**—Results suggest that clinical spirocercosis occurs more often in young-adult, large-breed dogs. Nonregenerative anemia, neutrophilic leukocytosis, hyperproteinemia, and high alkaline phosphatase activity may be useful clinicopathologic indicators of this disease. (J Am Vet Med Assoc 2006; 228:1063–1067)

**S** pirocerc**a** l**u**pi is recognized worldwide as an endoparasite of dogs and other carnivores, which become infected after the ingestion of larvae-laden intermediate or transport hosts.1 Esophageal granuloma and occasionally sarcoma, aortic aneurysm and thrombosis, thoracic diskospondylitis and spondylitis, hypertrophic osteopathy, and salivary gland necrosis have all been associated with migration of larvae and persistence of adult worms in tissue granulomas.2-7 Although *S* *lupi* infection is often subclinical, esophageal dysphagia is a common manifestation in infected dogs.8,9 A diagnosis of spirocercosis is made on the basis of results of fecal tests, thoracic radiography, and esophagoscope.9 The epidemiologic, clinical, and clinicopathologic features of spirocercosis may vary widely among areas in which the organism is endemic.1,9-11 In Greece, for instance, a high infection rate has been documented in dogs used for tracking (mainly Hellenic hounds),11 but the clinical form of spirocercosis is apparently uncommon.12

The purposes of the study reported here were to determine clinical signs and clinicopathologic abnormalities in dogs with naturally occurring clinical spirocercosis, determine the nature and prevalence of concurrent diseases in affected dogs, identify potential risk factors for the development of clinical spirocercosis, and identify clinicopathologic abnormalities indicative of the disease.

**Criteria for Selection of Cases**

Medical records of all dogs admitted to the Clinic of Companion Animal Medicine, Aristotle University of Thessaloniki between September 1996 and June 2004 were searched, and cases in which a definitive diagnosis of clinical spirocercosis had been established were included in the study. For purposes of the present study, a diagnosis of clinical spirocercosis was made if *S* *lupi* ova were recovered from feces or typical parasitic nodules were seen during endoscopic examination of the esophagus and histology and clinical findings were consistent with the disease. Cases were excluded if the medical record was incomplete.

**Procedures**

Medical records of cases included in the study were reviewed, and information on signalment, residence (rural vs urban), season of onset of clinical signs, owner complaints, physical examination findings, clinicopathologic abnormalities, radiographic and endoscopic findings, and concurrent systemic diseases was recorded. Fecal samples collected directly from the rectum were examined by means of the...
Teleman sedimentation method, and feces collected daily for up to 3 consecutive days were tested before a dog was declared to be free from *S. lupi* ova. Esophagoscopy and gastroscopy were performed with the aid of a flexible, 7.9-mm-diameter endoscope; the location, number, and gross appearance of parasitic nodules were recorded. Thoracic radiographs obtained at the time of admission were reviewed for lesions compatible with a diagnosis of spirocercosis.

To identify potential risk factors for development of clinical spirocercosis, age, sex, residence, breed, and body weight data for dogs with clinical spirocercosis were compared with data for a control group of 117 dogs determined to be free from *S. lupi* infection on the basis of results of fecal and endoscopic examinations that were examined at the Clinic of Companion Animal Medicine during the same period because of gastrointestinal tract disease and were randomly selected from the medical records of the clinic. To identify potential clinicopathologic indicators of clinical spirocercosis, results of hematologic and serum biochemical testing for dogs with clinical spirocercosis were compared with results for 56 healthy dogs examined at the clinic during the same period for routine vaccination for which results of a fecal test were negative for *S. lupi* ova.

### Statistical analysis

Pearson $\chi^2$ tests were used to compare sex, breed, and residence distributions for dogs with clinical spirocercosis with distributions for control animals examined because of gastrointestinal tract disease, and $t$ tests were used to compare mean age and body weight between groups. For dogs with clinical spirocercosis, the Fisher exact test was used to compare prevalence of clinical spirocercosis between dogs < 1 year old and $\chi^2$ tests were used to analyze data for onset of clinical signs (warm months [April through October] vs cold months [November through March]) and annual incidence of disease. The Fisher exact test was used to compare frequencies of hematologic and serum biochemical abnormalities between dogs with clinical spirocercosis and healthy control dogs, after excluding dogs with clinical spirocercosis that had other systemic diseases. Statistical analyses were performed with standard software; values of $P < 0.05$ were considered significant.

### Results

Thirty-nine cases of clinical spirocercosis met the criteria for inclusion in the study. They included 23 (59%) purebred dogs, 14 (36%) mixed-breed dogs, and 2 (5%) crossbred dogs. There were 23 (59%) sexually intact males and 18 (46%) sexually intact females. Median age was 3.5 years (range, 0.8 to 10.5 years), and median body weight was 20.3 kg (44.7 lb; range, 8.4 to 44 kg [18.5 to 96.8 lb]).

Sex distribution for dogs with clinical spirocercosis was not significantly different from sex distribution for control dogs examined because of gastrointestinal tract disease; however, mixed-breed dogs ($P = 0.008$) and Hellenic hounds ($P = 0.01$) were significantly over-represented. Dogs with clinical spirocercosis were significantly ($P = 0.017$) younger (mean, 4.0 years) than control dogs examined because of gastrointestinal tract disease (5.4 years). Dogs > 1 year old were significantly ($P < 0.001$) more likely than dogs < 1 year old to develop clinical spirocercosis. Mean body weight of dogs with clinical spirocercosis was significantly ($P < 0.001$) higher than mean body weight of control dogs examined because of gastrointestinal tract disease, and large-breed dogs (body weight > 20 kg) were significantly ($P = 0.008$) overrepresented. Residence distribution (urban vs rural) for dogs with clinical spirocercosis was not significantly different from distribution for control dogs examined because of gastrointestinal tract disease. Distribution of cases of clinical spirocercosis did not vary significantly with season of onset of clinical signs or year of the study (median, 6 cases/y; range, 1 to 10 cases/y).

### Table 1—Hematologic and serum biochemical abnormalities in 39 dogs with clinical spirocercosis.

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>No. with abnormality/No. examined (%)</th>
<th>Range (mean)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia (PCV &lt; 37%)</td>
<td>20/39 (51)*</td>
<td>16.2–36.5 (26.3)</td>
</tr>
<tr>
<td>Leukocytosis (&gt; 17,000 WBCs/µL)</td>
<td>9/37 (24)*</td>
<td>17,500–44,100 (26,950)</td>
</tr>
<tr>
<td>Leukopenia (&lt; 4,000 WBCs/µL)</td>
<td>3/37 (8)</td>
<td>4,900–5,940 (5,310)</td>
</tr>
<tr>
<td>Thrombocytopenia (&lt; 200,000 platelets/µL)</td>
<td>6/37 (16)</td>
<td>35,000–178,000 (59,610)</td>
</tr>
<tr>
<td>Thrombocytosis (&gt; 500,000 platelets/µL)</td>
<td>4/37 (11)</td>
<td>521,000–715,000 (609,500)</td>
</tr>
<tr>
<td>Neutrophilia (&gt; 11,500 neutrophils/µL)</td>
<td>11/37 (30)</td>
<td>11,850–37,040 (19,520)</td>
</tr>
<tr>
<td>Neutropenia (&lt; 3,000 neutrophils/µL)</td>
<td>1/37 (3)</td>
<td>2,290</td>
</tr>
<tr>
<td>Lymphocytosis (&gt; 4,000 lymphocytes/µL)</td>
<td>3/37 (8)</td>
<td>5,360–8,460 (6,390)</td>
</tr>
<tr>
<td>Lymphopenia (&lt; 1,000 lymphocytes/µL)</td>
<td>3/37 (8)</td>
<td>220–680 (350)</td>
</tr>
<tr>
<td>Monocytosis (&gt; 1,350 monocytes/µL)</td>
<td>10/37 (27)</td>
<td>1,370–3,490 (2,290)</td>
</tr>
<tr>
<td>Eosinopenia (&lt; 100 eosinophils/µL)</td>
<td>6/37 (16)</td>
<td>0</td>
</tr>
<tr>
<td>Eosinophilia (&gt; 1,250 eosinophils/µL)</td>
<td>2/37 (5)</td>
<td>1,480–2,110 (1,800)</td>
</tr>
</tbody>
</table>

*ALP = Alkaline phosphatase. ALT = Alanine aminotransferase. CK = Creatine kinase. *Significantly ($P < 0.05$) higher than prevalence among a control group of 56 healthy dogs.
The most common owner complaints for the 39 dogs with clinical spirocercosis included regurgitation (27 dogs [69%]), odynophagia (23 [59%]), excessive salivation (13 [33%]), anorexia or poor appetite (12 [31%]), progressive weight loss (5 [13%]), coughing (3 [8%]), weakness (3 [8%]), and vomiting (1 [3%]). Duration of clinical signs prior to examination ranged from 3 to 700 days (median, 20 days).

Clinical signs in dogs with spirocercosis included odynophagia (34 [87%]), regurgitation of solid (22 [56%]) or semisolid (2 [5%]) food, excessive salivation (14 [36%]), progressive weight loss (12 [31%]), lymphadenomegaly (5 [13%]), splenomegaly (5 [13%]), melena (3 [8%]), exfoliative dermatitis (3 [8%]), anorexia (3 [8%]), pyothorax (2 [5%]), cough (2 [5%]), masti- catory muscle atrophy (1 [3%]), hepatomegaly (1 [3%]), and cranial vena cava syndrome (1 [3%]). Thirty-eight of the 39 (97%) dogs had esophageal dysphagia, defined as odynophagia, regurgitation of food, excessive salivation, or some combination of these. The remaining dog was examined because of melena.

Normocytic, normochromic, nonregenerative anemia ($P < 0.001$); neutrophilic leukocytosis ($P = 0.047$); hyperproteinemia ($P = 0.037$); and high serum alkaline phosphatase activity ($P < 0.001$) were significantly more common in dogs with clinical spirocercosis than in healthy control dogs (Table 1).

Lateral and dorsoventral radiographic projections of the thorax obtained at the time of admission were available for 35 of the 39 dogs with clinical spirocercosis. Radiographic abnormalities were observed in 27 of these 35 (77%) dogs, with the most common radiographic abnormality being a mass in the caudodorsal aspect of the mediastinum (15 [43%] dogs; Figure 1). Masses were variable in size and extended from the region of T6 to T10. Other radiographic abnormalities that were seen included segmental accumulation of intraluminal air in the esophagus (13 [37%]), spondylitis between T5 and T11 (10 [29%]), ventral displacement of the trachea or mainstem bronchi (8 [23%]), an alveolar pattern in the cranial lung lobes (4 [11%]), a mass in the cranidorsal aspect of the mediastinum (2 [6%]), and pleural effusion (pyothorax; 1 [3%]).

Parasitic nodules were seen during esophagoscopy in all 39 dogs. Parasitic nodules were seen exclusively in the distal aspect of the thoracic part of the esophagus, between 2 and 17 cm (mean, 5 cm) from the lower esophageal sphincter. A single nodule was seen in 23 (59%) dogs, 2 nodules were seen in 8 (21%) dogs, 3 nodules were seen in 7 (18%) dogs, and 4 nodules were seen in 1 (3%) dog. Ulcerative nodules were observed in 4 (10%) dogs, and 3 of the 4 had melena. In 5 (13%) dogs, adult *S. lupi* worms were seen to protrude from the nodule. Results of gastroscopy were unremarkable in all dogs.

Results of the Telemann fecal sedimentation test were positive for 36 of the 39 (92%) dogs. *Spirocerca lupi* ova were found in the first fecal sample collected from 33 (92%) dogs and in the second fecal sample collected from 3 (8%).

Concurrent systemic diseases were identified in 14 (36%) dogs at the time of admission. Three dogs had leishmaniosis, 3 had dirofilariasis, 3 had monocytic ehrlichiosis secondary to *Ehrlichia canis* infection, 2 had leishmaniosis and dirofilariasis, 1 had hepatozoosarosis, 1 had pyothorax, and 1 had pyometra.

**Discussion**

Our finding that clinical spirocercosis occurred primarily in young-adult (median age, 3.5 years), large-breed dogs is generally in agreement with results of previous studies.1-10 Mixed-breed dogs and Hellenic hounds were overrepresented, compared with the control group, presumably because their lifestyle (eg, outdoor activity) and use (eg, guarding and tracking) gave
them more opportunities to acquire the infection. Notably, in studies conducted in Greece, stray dogs were found to have an infection rate as high as 24.2%, whereas household pets (ie, dogs that spent at least 80% of their time indoors) and scent-hunting dogs had significantly lower infection rates (0% and 5%, respectively) than did track-hunting dogs. Labrador Retrievers and German Shepherd Dogs were found to be overrepresented in similar studies conducted in South Africa and Israel.

In the present study, year, season of onset of clinical signs, and residence (urban vs rural) were not associated with development of clinical spirocercosis. It is possible, therefore, that environmental factors (eg, soil type and pH, temperature, rainfall, and solar radiation) involved in sustaining the availability of intermediate hosts do not substantially affect the epidemiology of the disease, at least in the area of the present study. The high number of stray dogs in many urban areas in Greece probably makes it difficult to detect any differences in infection rate between urban and rural areas. Similarly, the long prepatent period (4 to 6 months) and the unpredictable time required for clinical manifestations to appear may explain the lack of seasonality. In contrast, an urban distribution of spirocercosis was recently documented in Israel, whereas a rural distribution has been reported in South Africa and Israel.

In the present study and has been considered an uncommon abnormality in dogs with clinical spirocercosis. In one of these dogs, it was not clear whether pyothorax was a result of esophageal perforation secondary to an ethanol-induced chemical burn or deep ulceration caused by parasitic granulomas. Because there was no endoscopic evidence of esophageal wall perforation or granuloma ulceration in the other dog, it is difficult to establish a strong cause-and-effect relationship between clinical spirocercosis and pyothorax. However, even in the absence of visible esophageal lesions, pyothorax could be caused by adult *S lupi* worms if they move towards the esophageal adventitia instead of the mucosa.

One dog in the present study had, in addition to esophageal dysphagia, nonpainful, pitting edema that extended over the head and neck and was indicative of cranial vena cava syndrome. Thoracic radiography confirmed the presence of a cranial mediastinal mass in this dog, but the nature of the mass could not be elucidated. *Spirocerca lupi* is occasionally responsible for formation of granulomas, abscesses, or hematomas in the cranial portion of the mediastinum, which could explain both induction of cranial vena cava syndrome and esophageal dysphagia in this dog.

Other manifestations of spirocercosis described in previous reports, such as vomiting, fever, lameness, paraparesis, dyspnea, salivary gland necrosis, and hypertrophic osteopathy, were not identified in the present study.

In agreement with results of a recent report, normocytic, normochromic, nonregenerative anemia and neutrophilic leukocytosis were more common among dogs with clinical spirocercosis in the present study than among healthy dogs, even after accounting for the confounding effects of concurrent systemic diseases. These hematologic abnormalities mainly reflect the underlying chronic disease process. Intermittent or continuous blood loss resulting from ulceration of granulomatous or neoplastic esophageal nodules may lead to microcytic, hypochromic anemia, but this was not observed among the 3 dogs with melena in the present study, probably because of the short duration of clinical signs. Hyperproteinemia and high serum alkaline phosphatase activity were the only serum biochemical abnormalities found more commonly among case dogs than healthy control dogs. Hyperproteinemia was most likely a result of the chronic granulomatous tissue reaction induced by the parasite. Interestingly, serum alkaline phosphatase activity was also found to be significantly increased in a previous case series of dogs with *S lupi*-associated esophageal sarcomas.

Caudodorsal mediastinal masses and thoracic spondylitis were the most prominent radiographic abnormalities detected in dogs in the present study. These findings, especially when seen in combination, are strongly indicative of spirocercosis in dogs living in areas where the disease is endemic. In dogs with clinical spirocercosis, thoracic masses are typically localized to the caudal portion of the mediastinum, reflecting the location of granulomas, sarcomas, abscesses, and hematomas induced by *S lupi*. Accumulation of intraluminal air in the esophagus may reflect changes in esophageal motility secondary to intramural granulomatous inflammation.
Spondylitis has been attributed to periosteal irritation or obstruction of intervertebral arteries caused by migrating juvenile *S lupi* worms. Ventral displacement of the trachea or mainstem bronchi was a result of the mechanical effects of the parasitic granulomas, whereas the alveolar pattern seen radiographically was most likely a result of mild aspiration pneumonia secondary to esophageal dysphagia, as it was localized to the cranial lobes. Diagnostically, thoracic radiography is less sensitive and specific than endoscopy but does allow detection of many pathologic consequences of *S lupi* infection.

Interestingly, the long-standing assumption that clinical spiroceriosis results from mechanical obstruction of the esophageal lumen by single or multiple *S lupi* granulomas could not be confirmed in dogs in the present study. In these dogs, either no obstruction was seen during esophagoscopy or the obstruction that was seen was not considered sufficient to result in esophageal dysphagia. This is in agreement with our previous clinical experience that no substantial differences could be seen during esophagoscopy in dogs with clinical versus subclinical spiroceriosis.

In the present study, the Telemann fecal sedimentation technique was associated with a high diagnostic yield, similar to that obtained with the sugar flotation technique. Flotation techniques that use zinc sulfate or sodium nitrate solution apparently have lower diagnostic sensitivity. Regardless of the method used, false-negative results have been ascribed to prepatent infection, intermittent egg passage, aberrant parasite migration, and neoplastic transformation of granulomas. Therefore, examination of multiple fecal samples collected on consecutive days is always recommended to increase the diagnostic yield.

In dogs with clinical spiroceriosis, esophagoscopy has a higher diagnostic value than does fecal testing because many pitfalls of the latter method can be easily overcome. However, unless there is visual evidence of adult worm protrusion into the esophageal lumen or the typical nipplelike orifice associated with parasitic nodules, differentiating between granulomas, sarcomas, and other primary neoplasms (eg, leiomyomas) is rather difficult because of their many macroscopic similarities.