Lungworm, *Filaroides osleri*, Infection in a Dog in Japan

Takao KOTANI, Makio HORIE1, Shinobu YAMAGUCHI, Yasuhiro TSUKAMOTO, Takaumi ONISHI1, Fumihito OHASHI2, and Sadashige SAKUMA

Departments of Veterinary Pathology, 1Veterinary Internal Medicine, and 2Veterinary Surgery, College of Agriculture, University of Osaka Prefecture, 1-1 Gakuen-cho, Sakai, Osaka 593, Japan

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**ABSTRACT.** A 7-month-old male Pomeranian had severe dyspnea for 2 weeks. The lateral bronchogram showed a stenosis of the trachea. Insipid of supportive therapy including supplemental oxygen, the dog died 5 days later. Six pedunculated nodules were recognized in the mucosal surface of the trachea at necropsy. The tracheal nodules were histopathologically granuloma characterized by many coiled parasites containing a little collagen fibers, lymphocytes, plasma cells and macrophages. Female parasites had a lot of embryonated eggs in the uterus. Immature worms were observed in the dilated lymph vessels of bronchial and bronchial wall in the lungs. The worms were identified as *Filaroides osleri* based on parasitological examinations.—**KEY WORDS:** canine, *Filaroides osleri*, tracheal nodule.

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Filaroidiasis (lungworm disease or pulmonary nematodiasis) in the dog is uncommon in Japan. The dog is the host to 3 species of lungworms belonging to the genus *Filaroides*: *F. osleri* [1, 3, 4, 9, 11, 13, 15], *F. milksi* [2, 7, 9, 12, 16, 17] and *F. hirshi* [5, 6, 8, 10, 14]. The latter 2 species have been reported in Japan [8, 10, 16]. This is the first report that documents the presence of lungworm in a dog infested with *F. osleri* in Japan.

A 7-month-old, male Pomeranian, weighing 1.1 kg was referred to the Veterinary Medical Teaching Hospital, University of Osaka Prefecture for the evaluation of severe dyspnea, cyanosis, and spasms during the past 2 weeks. This dog had been raised inside a house with other 7 dogs including his mother. Seven dogs, 5 females and 2 males, were the same breed, 1.6 kg to 2.8 kg in body weight and 1.0 to 2.8 years old. They showed no clinical signs.

The lateral chest radiograph after the injection of contrast medium into the trachea revealed a stenosis in the ventral side of the trachea (Fig. 1). There was no significant change in the lungs. Results of a complete blood cell count and serum biochemistry analysis were within normal ranges except for a few basophils of the differential leukocytes. Inspite of a supportive therapy including supplemental oxygen, the affected dog died 5 days later, and a complete necropsy was performed.

At necropsy, six nodules were observed at the ventral and ventrolateral sides of the mucosa in the posterior one third of the trachea to the bronchial bifurcation (Fig. 2). The nodules were reddish with white winding streaks under the surface. They were 2 × 3 mm to 7 × 9 mm in size and showed a rise 2 to 4 mm from the normal tracheal mucosa. The nodule at the bronchial bifurcation was the largest and assumed a shape of a saddle, and it almost occluded the lumens of the left and right bronchi. Tracheal and bronchial lumens were filled with a large amount of foamy fluid.

The lungs showed a remarkable congestive edema and emphysema. The minute whitish foci were observed at the subpleura and the cut surface of the lungs. Tracheobronchial lymph nodes swelled edematously. Significant gross lesions were not observed in other organs except for the moderate inner hydrocephalus.

For histopathologic examination, tissues from the trachea, bronchi, lungs, tracheobronchial lymph nodes and other main organs were fixed in 10% neutral buffered formalin. Paraffin sections were prepared and stained with hematoxylin and eosin. Some sections were stained with elastic van Gieson and periodic acid Schiff method.

The pedunculated nodules of the tracheal mucosa contained coils masses of adult nematodes. The size of the nodules was related to the number of worms (Fig. 3a). The female parasites had a lot of embryonated eggs in the uterus (Fig. 3b). There was mild proliferation of the connective tissue with small blood vessels and infiltration of a few lymphocytes, plasma cells and macrophages. Eosinophils were not observed in these nodules. The parasitic nodules were covered by the flat epithelium. In some areas, there were desquamation of epithelial cells and proliferation of goblet cells.

The parasitic lesions were observed in each section cut from all lobes of the lungs (Table 1). Most of the nematodes were infested in the dilated lymph vessels of the bronchial and bronchiolar walls. The worms in these lesions were immature adult (without viviparity). There was no inflammatory cell in the surrounding tissues of lymphatics containing a few worms (Fig. 4). On the other hand, the lymph vessels containing many and/or coiled worms showed the granulomatous lesion with some lymphocytes and monocytes in the surrounding connective tissue (Fig. 5). There was congestive edema with alveolar macrophages into the alveoli. In some areas of the lungs, a few immature and gravid nematodes were observed in the lumen of bronchioles and emphysematous alveoli (Fig. 6). There was no significant inflammatory reaction in the surrounding pulmonary tissue. A focal lesion with a larva, consisting of some monocytes, macrophages, neutrophils and lymphocytes, was observed in one tissue section (Table 1).

Immature nematodes were observed in the sinuses of tracheobronchial lymph nodes (Fig. 7). There was no parasitic lesion in other organs.

For parasitological examination, three intact adult males and many fragments of adult males and females were collected from a formalin fixed tracheal nodule. Those worms were treated with the lactophenol, and
Fig. 1. Left lateral radiograph after injection of contrast media in the trachea (T). Narrowing of the tracheal lumen was prominent (arrows).

Fig. 2. Nodules containing adult *Filaroides osleri* in the tracheal mucosa.

Fig. 3a. Pendunculated nodule of the tracheal mucosa containing many coiled masses of worms. Hematoxylin and eosin (H-E) stain. × 25.

Fig. 3b. Enlarged view of site A in Fig. 3a. The female parasite has many embryonated eggs in the uterus. There is slight inflammatory reaction consisting of lymphocytes and plasma cells. H-E stain. × 125.

observed under the light microscope. Male and female worms had a tegminal sheath in cuticle, simple buccal cavity and a clearly defined flask-shaped esophagus. Males, 3.9 to 4.8 mm in body length (number of worms; n=3), with a maximum body breadth of 103 to 123 μm excluding tegminal sheath, and length of esophagus (n=4) 216 to 233 μm. The posterior end (Fig. 8), bluntly pointed and non-bulbate. Spicules (n=8), paired, subequal, 81 to 91 μm in length, with transverse ridges. Adult females were longer than the males in spite of the injured bodies. Body breadth, 228 to 247 μm and esophagus 226 to 230 μm in length. The posterior end, deeply cleft, with the anus and vulva opening close together in the cleft (Fig. 9). Uterus, including developing eggs filled with larvae encapsulated by thin shell membrane. Mean measurement of embryonated eggs (n=20) in the uterus, 98 by 73 μm.

Fecal examination was not done in the affected dog. First stage larvae, however, were recovered by the Baermann technique from the fecal samples in 4 of 7 dogs in the same household. These dogs showed no clinical signs. The larvae (n=40), 236 ± 21 μm (mean ± SD) in length and 14 ± 2 μm (mean ± SD) in breadth with a
Fig. 4. An immature nematode in a lymph vessel of bronchial wall. H-E stain. × 200.

Fig. 5. Nodular lesion containing coiled nematode in a dilated lymph vessel of bronchiolar wall. H-E stain. × 100.

Fig. 6. Gravid nematodes within a pulmonary alveoli. H-E stain. × 100.

Fig. 7. Immature nematodes (without viviparity) in dilated sinus of the tracheo-bronchial lymph node. H-E stain. × 100.

Fig. 8. Posterior end of an adult male which recovered from the tracheal nodule (SP = spicule). × 500.

Fig. 9. Posterior end of an adult female which recovered from the tracheal nodule. × 135.

Fig. 10. A first stage larva recovered from feces of a dog lived with the patient in the same house. × 310.
Table 1. Number of parasitic lesions on lung cross sections \(^a\) in the middle part of each lobe in a dog infected with lungworm

<table>
<thead>
<tr>
<th>Pulmonary lobes</th>
<th>Peribronchial lymph vessel</th>
<th>Lumen of bronchi and alveoli</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cranial lobe (Cr. L)</td>
<td>1</td>
<td>1b</td>
</tr>
<tr>
<td>Middle lobe</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Accessory lobe</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Caudal lobe</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Left</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cranial part of Cr. L</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Caudal part of Cr. L</td>
<td>1</td>
<td>1b</td>
</tr>
<tr>
<td>Caudal lobe</td>
<td>5</td>
<td>1b</td>
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</tbody>
</table>

\(^a\) Histopathologic section.
\(^b\) Gravid female in alveoli.
\(^c\) Larval lesion.

Distinct and kinked tail (Fig. 10).

Adult worms and 1st stage larvae in this report have been identified as *F. osleri* according to the morphological characteristics of anterior and posterior ends and measurements of body length, esophagus, and spicules [2–5, 8–11, 15–17]. Mature *F. osleri* forms the pedunculated nodules in the mucous membrane of near the bifurcation of trachea and bronchi [1, 3, 4, 9, 11, 15]. On the other hand, mature *F. milki* [2, 7, 9, 12, 16, 17] and *F. hirthi* [6, 8, 10, 14] are present in the alveolar spaces and bronchiolar lumen. There is no significant inflammatory reaction around intact nematodes, but a marked tissue reaction like granulomatous lesions against the degenerated worms. Immature worms of *F. osleri* are infested in the lymph vessels of the lungs [3, 9, 15]. In experimentally infected dingo and domestic dogs, 4th-stage larvae and 5th-stage immature nematodes were recovered from the alveoli and pulmonary parenchyma [3, 4]. In lungs of this case, the great majority of immature nematodes were located in the peribronchiolar and peribronchial lymph vessels. On the other hand, a few gravid females and immature nematodes were recognized in the alveoli and bronchioles. Since these nematodes were not examined parasitologically, there might be some migration of young adults of *F. osleri* from lymph vessels or might be a mixed infection with other species of lungworms.

Details of the life cycle and migration of *F. osleri* are still unknown. The dogs, however, have become infected following the experimental administration of 1st-stage larvae [3, 4, 13, 15]. The affected dog was always raised indoors since his birth with other 7 dogs. All these dogs were born in Japan. One of the four dogs in which larvae were observed in feces had been kept with imported dogs from U.S.A. for several months at a former owner's house. Although we can not decide the infectious route and source of the nematode, there might be direct infection from the 1st-stage larvae in feces of a carrier dog.

REFERENCES