

## Salivary mucocele associated with dirofilariasis in a dog

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A 4-year-old male Pekingese dog was admitted for examination of a circumscribed cervical mass. The mass had been first noticed the previous evening. Examination of the mass revealed a fluctuant mass, 8 cm in diameter, in the cranioventral cervical region.

Initial differential diagnoses included salivary mucocele, abscess, neoplasia, and lymphadenopathy. Aspiration of the cervical mass yielded mucoid, blood-tinged fluid. Direct microscopic examination of the fluid revealed 3 to 5 microfilariae/hpf, and numerous leukocytes and RBC. Cytologic examination of smears of the fluid stained with a modified Wright's stain<sup>a</sup> revealed predominantly eosinophils (10 to 15/hpf). A membrane filter test for microfilariae was performed on formalinized blood, and numerous microfilariae with morphologic features characteristic of *Dirofilaria immitis* were observed. Circulating *D immitis* antigen was confirmed, using an immunoassay kit.<sup>b</sup>

A 20-gauge needle was used to drain 75 ml of mucoid fluid from the mass. As the needle was withdrawn, a filariid worm was seen protruding from the aspiration site. The live worm was removed with a hemostat. The worm was approximately 125 mm long. A tentative diagnosis of salivary mucocele caused by aberrant migration of an adult *D immitis* was made.

Aspiration of the mass the following day yielded only 3 ml of fluid. Palpation of the ventral cervical region revealed a poorly defined fluctuant area on the ventral midline, just cranial to the angle of the mandible. The dog was discharged with instructions that acetylsalicylic acid (5 mg/kg of body weight, q 24 h) be given as a prophylactic treatment for pulmonary thromboembolism associated with adulticide therapy.<sup>1,2</sup> The dog was scheduled for adulticide therapy at 10 days after discharge.

The dog was readmitted 10 days after the initial examination. Radiography revealed right ventricular and pulmonary arterial segment enlargement, and perivascular parenchymal lung disease. Results of hematologic and serum chemical analyses were normal except for a low serum alkaline phosphatase activity (18 IU) and an increased aspartate aminotransferase activity (85 IU).

Adulticide therapy consisted of thiacetarsamide sodium<sup>c</sup> (0.22 mg/kg, iv, q 12 h) for 4 treatments.<sup>3</sup> Aspirin was continued for 30 days following the termination of adulticide therapy. Microfilaricidal therapy was instituted 3 weeks after adulticide therapy using ivermectin<sup>d</sup> (0.05 mg/kg, PO).<sup>2</sup> At the time of microfilaricide treatment, swelling was not seen in the ventral cervical region and aspiration of the area of the cervical mass yielded only 0.5 ml of mucoid fluid. Microfilariae were not observed on microscopic examination of the fluid. Four weeks after microfilaricide treatment, *D immitis* microfilariae were not found in a membrane filter test performed on the dog's blood. Preventive therapy, using ivermectin (68 µg, PO, q 30 days), was recommended. Eight weeks after adulticide therapy, antigen of *D immitis* was not detected by immunoassay. Examination of the dog at that time, and again at 6 months after the initial examination, revealed no abnormalities.

Finding characteristic microfilariae in the blood and in the fluid of the mucocele supports the premise that the observed adult filariid was *D immitis* and that obstruction of the salivary duct by the filariid or by the host's reaction to the filariid resulted in the formation of a salivary mucocele. Although most salivary mucoceles result from trauma to the sublingual salivary gland,<sup>4</sup> there was no evidence of trauma at the time of the initial examination. We confirmed that the dog was infected with heartworms by detecting circulating *D immitis* antigen. Aberrant migration of adult *D immitis* has been documented in the brain, spinal cord, abdominal cavity, lymphatic system, and the skin.<sup>2,5-8</sup>

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<sup>a</sup>Diff-quick, AHS del Caribe Inc, Aguando, Puerto Rico.

<sup>b</sup>Cite semi-quant heartworm antigen test kit, IDEXX Corp, 100 Fore St, Portland, Me.

<sup>c</sup>Caparsolate, Abbott Laboratories, North Chicago, Ill.

<sup>d</sup>Ivomec, MSD Agvet, Rahway, NJ.

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### Correction

In the article "Epidemiologic features of von Willebrand's disease in Doberman Pinschers, Scottish Terriers, and Shetland Sheepdogs: 260 cases (1984-1988)" (*JAVMA*, Apr 15, 1992, pp 1123-1127), on page 1125, the legends for Figures 1 and 2 were published in error. The *JAVMA* regrets the error. The figures, with correct legends, are as follows:

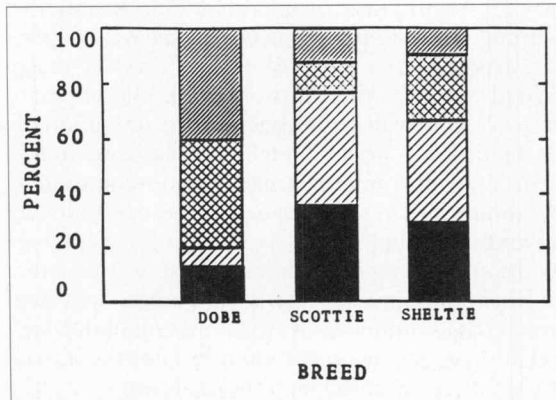


Figure 1—Percentage distribution of age at time of diagnosis in Doberman Pinschers (Dobe), Scottish Terriers (Scottie), and Shetland Sheepdogs (Sheltie) affected with von Willebrand's disease (vWD).  
Age (years): ■ = 0 to 0.5; ▨ = 0.6 to 1; ▩ = 1.1 to 5; ▤ = >5.

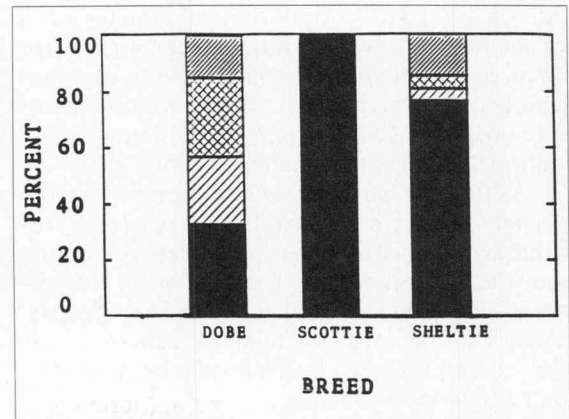


Figure 2—Percentage distribution of von Willebrand's factor antigen (vWF:Ag) concentration in vWD-affected Doberman Pinschers, Scottish Terriers, and Shetland Sheepdogs.  
vWF:Ag %: ■ = 0 to 7; ▨ = 8 to 14; ▩ = 15 to 35; ▤ = 36 to 55.