What Is Your Diagnosis?

A 7-year-old 30-kg (66-lb) sexually intact female Rhodesian Ridgeback was referred because of ptyalism and hind limb paresis. Two days earlier, the dog was seen by the referring veterinarian because of ptyalism and retching; the dog was treated with maropitant and minocycline. Once at home, the patient began vomiting, which continued for 24 hours and prompted a visit to a local veterinary emergency clinic. Abdominal radiographs were obtained to rule out a gastrointestinal foreign body, and the dog received fluids IV.

A loud referred upper airway noise was detected on physical examination at the time of hospital admission. Persistent hypersalivation was evident with no observable masses or lesions in the oral cavity. Auscultation of the lungs revealed a crackling sound. The patient had not eaten or urinated in 3 days. The dog had hind limb ataxia with muscle atrophy and absent patellar reflexes bilaterally. Conscious proprioception responses were delayed with intact superficial pain responses bilaterally in the hind limbs. The patient was estimated to be 7% to 10% dehydrated. Mucous membranes were light pink with some yellow discoloration. No other abnormalities were detected on physical examination. Findings on CBC were within reference limits, and the PCV was 50%. Serum biochemical analysis revealed a high blood glucose concentration (120 mg/dL; reference range, 60 to 110 mg/dL) and a low BUN concentration (6 mg/dL; reference interval, 7 to 25 mg/dL). Previously obtained radiographic images of the abdomen were reviewed; no evidence of a gastrointestinal obstruction was seen. Ultrasonography of the abdomen and thorax was performed; no evidence of free fluid, pericardial effusion, or pleural effusion was found (images not shown). Radiographs of the thorax were obtained (Figure 1).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

Figure 1—Left lateral (A) and ventrodorsal (B) radiographic views of the thorax of a 7-year-old sexually intact female Rhodesian Ridgeback evaluated for ptyalism, retching, and hind limb ataxia of 3 days’ duration.

This report was submitted by Mikayla M. Schroeder, BA; Christopher P. Ober, DVM, PhD; Kristina Kiefer, DVM, PhD; and Jasmine Tom, DVM; from the Department of Veterinary Clinical Sciences, College of Veterinary Medicine, University of Minnesota, Saint Paul, MN 55108 (Schroeder, Ober, Kiefer); and Blue Spruce Animal Clinic, 101 Briscoe St, Castle Rock, CO 80104 (Tom).

Address correspondence to Ms. Schroeder (schr1146@umn.edu).
Radiographic Findings and Interpretation

The esophagus is diffusely dilated with gas, allowing visualization of the dorsal and ventral esophageal walls and causing ventral displacement of the trachea. A tracheal stripe sign is also noted, compatible with esophageal gas. The lungs are radiographically normal, with no evidence of pneumonia (Figure 2).

Differential diagnoses for the radiographic findings included megaesophagus secondary to myasthenia gravis, idiopathic megaesophagus, hypoadrenocorticism, or hypothyroidism. With the concurrent hind limb weakness and ataxia, myasthenia gravis was considered the most likely diagnosis.

Treatment and Outcome

Intervenous fluid therapy was initiated. The dog was treated with maropitant (1 mg/kg [0.45 mg/lb], SC) and famotidine (1 mg/kg, IV) and discharged from the hospital. Anti-acetylcholinesterase treatment was discussed at the time of hospital discharge, but declined. Client education for management of megaesophagus was provided. The patient had a severe decline in condition overnight and underwent respiratory arrest and died.

A serum sample had been submitted to measure the anti-acetylcholine receptor antibody titer; 5 days later, results were positive for acquired myasthenia gravis with a serum antibody titer of 5.22 mmol/L (reference limit, < 0.6 mmol/L).

Comments

Myasthenia gravis is a neuromuscular disease characterized by exercise-associated weakness. There are both congenital and acquired forms; however, the congenital form that has an onset at 3 to 8 weeks of age in puppies is not as prevalent. Acquired myasthenia gravis is rarely reported in dogs < 1 year of age. There appears to be 2 distinct age peaks for acquired myasthenia gravis: between 2 and 4 years of age and between 9 and 12 years of age. Any breed of dog may be affected by acquired myasthenia gravis; however, retrospective studies suggest that German Shepherd Dogs, Akitas, German Shorthaired Pointers, Chihuahuas, and terrier group breeds may be overrepresented. Myasthenia gravis is rare in cats. Occasionally, acquired myasthenia gravis in dogs is associated with a thymoma.

Dogs with acquired myasthenia gravis often have clinical signs that include ptyalism, regurgitation, and progressive fatigue that worsens with exercise. When exercised, these animals tend to develop a choppy gait before sitting or lying down and refusing to move. Generally, with rest, they are able to rise and ambulate again. With concurrent clinical signs of muscle weakness, regurgitation, and neurologic signs, thoracic radiography aids in identification of megaesophagus, aspiration pneumonia, or thymoma. Therefore, thoracic radiography should be included in a comprehensive workup for dogs suspected of having myasthenia gravis. A finding of megaesophagus can be a sequela to many disease processes, of which myasthenia gravis is relatively uncommon. Most cases of megaesophagus are considered idiopathic in nature or related to metabolic disease. Although the following list is not exhaustive, specific differential diagnoses for radiographic.

Figure 2—Same radiographic images as in Figure 1. Notice the walls of the dilated esophagus (black arrows) that are indicative of megaesophagus. A tracheal stripe sign (white arrows) is evident as a result of gas in the esophagus and the silhouetting of the ventral esophageal wall and the dorsal tracheal wall. There is also mild ventral displacement of the trachea caused by esophageal distension.
signs of megaesophagus with concurrent paresis include botulism, organophosphate toxicoses, polymyositis, and polyneuropathy. Esophageal foreign body and vascular ring anomalies are common rule outs for regurgitation but exhibit focal rather than diffuse esophageal dilation radiographically. Given the variety of differential diagnoses for megaesophagus, serologic testing for anti-acetylcholine receptor antibody titers must be performed to confirm myasthenia gravis.

In affected dogs with recurrent or severe aspiration pneumonia, prognosis is usually guarded to poor. However, if not complicated by such sequelae, many dogs will respond favorably to anticholinesterase and treatment with prednisone and can have a favorable outcome. Surgical excision of thymic tumors can result in dramatic improvement of signs. Occasionally affected dogs will achieve complete remission and no longer require lifelong treatment.

Dogs with acute fulminating myasthenia gravis have a sudden onset of frequent regurgitation and radiographic evidence of megaesophagus. In these affected dogs, generalized weakness was reported to progress to complete recumbency and death in a matter of days. The most common cause of death in these patients was aspiration and loss of strength in respiratory muscles with resultant respiratory arrest. Unfortunately, no necropsy was performed in the dog of the present report, so the cause of death is unknown; however, the history, clinical signs, and diagnostic results strongly supported a diagnosis of acute fulminating myasthenia gravis.

References