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Aortic chondroid neoplasia in two Labrador Retriever dogs☆

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Abstract In the same week, two Labrador Retriever dogs presented to The Ohio State University Veterinary Medical Center for cardiac evaluation. The presenting signs in both dogs included: weight loss, weakness, lethargy, and decreased femoral pulses. The first dog presented in cardiogenic shock and biventricular congestive heart failure, which initially responded to treatment; however, the dog was euthanized due to deteriorating clinical condition. In contrast, the second dog had a milder clinical course without signs of congestive heart failure, and remained stable over the 2-month period of clinical evaluation prior to euthanasia.

Echocardiographic evaluation revealed a dilated cardiomyopathy phenotype in the first dog, while a space-occupying intraluminal mass originating at the aortic valve with preserved left ventricular systolic function was observed in the second dog. At autopsy, each dog had a large obstructive luminal mass affecting the ascending aorta and arch. Histopathology revealed that the mass in the first dog was consistent with a benign chondroma, while in the second dog the morphologic

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characteristics, mitotic activity, and infiltrative growth justified a diagnosis of chondrosarcoma. This report presents the contrasting clinical disease progression and findings in two dogs with aortic neoplasia, with a proposed pathogenesis of cardiac failure secondary to aortic neoplasia.

Introduction

An approximately 10-year-old female, spayed Labrador Retriever (Dog 1) weighing 28.1 kg was presented to The Ohio State Veterinary Medical Center with a 2-week history of progressive weight loss, inappetence, cough, and dyspnea. The dog was tetraparetic with weak femoral pulses, a heart rate of 140 beats per minute, and a respiratory rate of 44 per minute. On auscultation, there were increased bronchovesicular sounds on the left and right sides of the thorax with no cardiac murmur detected. Arterial blood pressure measured 70 mmHg by indirect Doppler measurement from the left hind limb. Complete blood count and serum biochemistry results were within reference intervals. Thoracic radiographs revealed rounding of the cardiac silhouette with predominately left-sided chamber enlargement, and a diffuse and patchy interstitial-to-alveolar pattern throughout the lungs, as well as mild pleural effusion, which were compatible with biventricular congestive heart failure. An echocardiogram was performed, which revealed moderate left ventricular dilatation, impaired left ventricular systolic function, and left atrial enlargement — consistent with a dilated cardiomyopathy phenotype (left ventricular internal dimension in diastole of 54.5 mm; left ventricular internal dimension in systole of 49.5 mm; linear percent fractional shortening of 9.2%; and left atrial diameter from a right parasternal long axis view of 52.4 mm; Fig. 1, Video 1). Retrospectively, an abnormal soft tissue echogenicity was apparent in the aortic arch, but was not appreciated at the time of initial evaluation.

The dog was hospitalized and received furosemide 60 mg IV q 12 h, and positive inotropes including pimobendan at 10 mg PO q 12 h, and dobutamine as a continuous IV infusion at 3—6 mcg/kg/min to maintain a systolic arterial pressure of >100 mmHg. The respiratory rate normalized within 24 h and the dog was more willing to ambulate. The dog then began to vomit repeatedly, so ondansetron 6 mg IV q 8 h, and maropitant citrate 30 mg SQ q 24 h were administered. Repeat thoracic radiographs showed resolution of the pleural effusion with a mild, resolving interstitial pattern in the right caudal lung lobe. The dog then developed diarrhea and repeat serum biochemistry showed moderate azotemia, with a serum urea nitrogen level of 52 mg/dL (reference interval 5—20 mg/dL) and a creatinine of 4.4 mg/dL (reference interval 0.6—1.6 mg/dL). However, the dog remained inappetent and lethargic. A complete abdominal ultrasound was performed, which revealed decreased caudal aortic blood flow without evidence of a thrombus or mass. Due to its deteriorating clinical condition, the dog was euthanized.

The following week, a 6.5-year-old male, castrated Labrador Retriever (Dog 2) weighing 28.6 kg presented to The Ohio State University Veterinary Medical Center with a 2-month history of collapse after exercise and intermittent, yet progressive, hind limb weakness that was worsened by activity. Physical examination documented an absent right femoral pulse, with mild bilateral hind limb weakness and muscle atrophy. Neurologic examination revealed mild hind limb ataxia, a decrease in hind limb conscious proprioception, mild hind limb weakness, and thoracolumbar pain. On thoracic auscultation, a Grade IV/VI left basilar systolic heart murmur with radiation to the right thorax was evident. Complete blood count and serum biochemistry results were unremarkable. Two-dimensional and Doppler echocardiography revealed a large, intraluminal, hyperechoic mass lesion extending from the non-coronary cusp of the aortic valve to the descending aorta, resulting in mild obstruction (peak left ventricular outflow velocity of 3.25 m/s, corresponding to a peak transaortic valve systolic pressure gradient of 45 mmHg) and mild aortic valve insufficiency (Fig. 2, Video 2). Left ventricular wall thickness and luminal dimensions were normal. Thoracic radiographs were unremarkable. Indirect systolic arterial blood pressure obtained by Doppler was normal in the right and left forelimbs (115 mmHg), decreased in the left hind limb (80 mmHg), and not attainable in the right hind limb. The dog was prescribed clopidogrel 75 mg PO q 12 h. Hemoptysis was noted 2 weeks after starting clopidogrel, at which time the packed cell volume was 55%, prothrombin time was 56 s (reference interval

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