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CASE REPORT

Aortic tear and dissection related to connective tissues abnormalities resembling Marfan syndrome in a Great Dane☆

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Abstract Aortic tears and acute aortic dissection are rarely reported in dogs. This report describes a case of aortic dissection and probable sinus of Valsalva rupture in a young Great Dane with associated histopathologic findings suggestive of a connective tissue abnormality.

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Abbreviations

CRI	constant rate infusion
RI	reference interval
UWVC	University of Wisconsin Veterinary Care

A six month-old intact female Great Dane was referred to University of Wisconsin Veterinary Care (UWVC), the veterinary medical teaching hospital of the University of Wisconsin, for further evaluation of severe pneumonia and hypoxemia [blood oxygen saturation (SpO_2) = 86–88%]. Twenty days prior to referral, the dog had been treated for cough with ampicillin/clavulanic acid (11 mg/kg PO, q12h for 7 days) for a tentative diagnosis of mild tracheobronchitis. No abnormal physical examination findings were noted at that time. Seventeen days later (3 days prior to referral), the dog was evaluated for acute lethargy and one episode of vomiting. Physical examination revealed an elevated rectal temperature (105.9 °F) and yellow–green vulvar discharge. A heart murmur was not ausculted. Free-catch urinalysis revealed 11–50 white blood cells per 40× field, approximately 20 red blood cells per 40× field, less than 20 cocci per 40× field, 3 to 10 struvite crystals per 40× field, urine specific gravity of 1.026 and a urine pH of 8.0. Culture results of the vulvar discharge revealed light growth of a mixture of urogenital and skin flora, but no predominant organism. A presumptive diagnosis of urinary tract infection or metritis was made. A 7-day course of cephalexin (22 mg/kg PO q12h) and a 2-day course of firocoxib (4.5 mg/kg PO q24h) were prescribed.

On the day of referral, the dog had been presented to the referring veterinarian for an acute onset of tachypnea (50–60 bpm) and harsh respiratory sounds. Complete blood count abnormalities included mild anemia (hematocrit 35%, reference interval (RI): 37–55%) and neutrophilic leukocytosis (white blood cells 26.25 × 10^6/L, RI: 6–17 × 10^6/L; neutrophils 22.5 × 10^6/L, RI: 3.5–12 × 10^6/L). The serum chemistry profile revealed a slightly elevated alkaline phosphatase (184 U/L, RI: 0–140 U/L), a low blood urea nitrogen (8 mg/dL, RI: 10–29 mg/dL), and mild hyponatremia (136 mmol/L, RI: 142–150 mmol/L). Thoracic radiographs obtained by the referring veterinarian revealed cardiomegaly (vertebral heart size = 12) and diffuse severe alveolar infiltrates in all lung fields. Blood oxygen saturation measured by pulse oximetry was 86–88%. The dog was referred to UWVC for further evaluation and treatment.

On presentation to the UWVC Emergency Service, the dog had an increased respiratory rate of 60 breaths/min, light pink mucous membranes, a normal temperature of 102.3 °F, a heart rate of 140 beats/min, and weighed 24 kg. Harsh respiratory sounds were present in all lung fields. A grade IV/VI systolic heart murmur was ausculted (location not recorded) with synchronous pulses. Bilateral enophthalmia was also noted. Abnormalities from an arterial blood gas included hypoxemia (PaO_2 = 53.8 mmHg, SaO_2 = 86.4%) and hypocapnia ($PaCO_2$ = 32.0 mmHg). Bilateral nasal cannulas were placed (oxygen flow set to 3 L/min), which increased the SpO_2 to 92%. A tentative diagnosis of infectious pneumonia was made and the dog was treated with fluids administered intravenously (Normosol-R, 5 mL/kg/hr) supplemented with 20 mEq KCl/L. Additional therapies initiated pending further diagnostic testing included ampicillin/sulbactam (20 mg/kg IV, q8h), and fluconazole (4 mg/kg IV, q12h). Nebulization and coupage were performed every 4 h.

The following morning increased respiratory rate and effort were noted and oxygenation was decreased despite oxygen support (SpO_2 88%). The dog's heart rate was elevated at 160 beats/minute with a regular rhythm on auscultation. Grade 4/6 systolic and diastolic murmurs were appreciated at the left and right heart base and the femoral pulses were hyperdynamic. Increased large airway sounds were heard over all lung fields. Arterial blood gas analysis revealed hypoxemia (PaO_2 = 59.9 mmHg, SaO_2 = 91.5%) and hypocapnia ($PaCO_2$ = 29.0 mmHg) similar to the admitting results. Thoracic radiographs revealed severe alveolar infiltrates bilaterally in the dorsal pulmonary fields, with bilateral interstitial to alveolar infiltrates in the caudal ventral lung fields and in the cranial lung fields. Pulmonary veins from the cranial lung lobes measured at the high end of the normal range. Due to the concern for pulmonary edema, the intravenous fluid rate was decreased to 3 mL/kg/hr, a constant rate infusion (CRI) of fentanyl (1 mcg/kg/hr) was initiated help alleviate anxiety associated with dyspnea,[1] and aminophylline (2.5 mg/kg IV) was administered once. Following these treatments, the dog appeared more comfortable and respiratory rate and effort reduced.

An echocardiogram was performed by the UWVC Cardiology Service[d]. A mobile hyperechoic lesion

[d] Vivid 7, General Electric Medical Systems, Waukesha, WI, USA.