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Case Report

Presumptive partial atrial standstill secondary to atrial cardiomyopathy in a Greyhound*

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KEYWORDS

Dog; Fibrofatty; Unilateral Abstract Persistent atrial standstill is a rare arrhythmia in both human and veterinary patients. In recent decades, cases of partial atrial standstill have been recognized in humans. We describe a case of presumptive partial atrial standstill in a Greyhound, in which there was disparate left and right atrial electromechanical function and rapid progression to congestive heart failure over the span of fourteen weeks. An atrial cardiomyopathy characterized by severe, diffuse, fibrofatty replacement of the atrial myocardium was identified histologically.

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A 4.5-year-old, 26.4-kg, female spayed Greyhound was presented to the Veterinary Teaching Hospital of the Virginia-Maryland College of Veterinary Medicine for evaluation of a newly identified heart murmur and arrhythmia. Enalapril had been prescribed by her primary care veterinarian at a dose of 10mg every 12 h; the patient was also receiving monthly heartworm, flea, and tick preventatives. Thoracic radiographs taken before presentation provided equivocal evidence of left atrial enlargement but the pulmonary parenchyma and vessels had a normal appearance. The vertebral heart score was 11.2. A point-of-care test for vector-borne disease^c had been negative two months prior. Systolic blood pressure was reported to be 140 mmHg.

On presentation, the dog was bright, alert, and panting. Her heart rate was 80 beats per minute and her body temperature was 100.7° F. A grade III/VI systolic heart murmur was heard best over the left cardiac apex and a grade II/VI systolic murmur was heard over the left heart base. The remainder of the physical examination was unremarkable.

A 6-lead electrocardiogram did not reveal evidence of atrial activity but a narrow QRS, presumed junctional rhythm, with rates between 50 and 95 beats per minute, was evident. Evaluation of precordial leads demonstrated low amplitude P waves and atrioventricular dissociation (Fig. 1). The P—P intervals were not consistent. Intravenous administration of atropine sulfate at a dose of 0.04mg/kg did not result in an appreciable increase in the ventricular rate and the previously identified low amplitude P waves became more difficult to identify due to a further decrease in amplitude.

echocardiography Transthoracic revealed severe left atrial enlargement. Left atrial-to-aortic root ratio was 2.0 (normal: 0.9-1.6) [1]. Left atrial volume indexed to body weight was 3.7 ml/kg (normal \leq 1.1 ml/kg) [2]. The left ventricle, right ventricle, and right atrium were deemed normal in size. Mild thickening of the mitral valve with mild mitral regurgitation was appreciated. Trace aortic regurgitation was also identified. Left ventricular outflow tract velocity was elevated at 2.8 m/sec (normal: 0.95–1.9 m/sec) [3]. The aortic valve and left ventricular outflow tract were structurally normal, and pulsed-wave Doppler interrogation failed to disclose a discrete velocity step-up suggesting that the left ventricular outflow velocity was attributable to breed-related characteristics [4] and increased stroke volume as a result of bradycardia. Pulsed-wave Doppler interrogation of mitral valve inflow did not reveal A waves. As waves were not identified during tissue Doppler interrogation of the lateral mitral valve annulus, though Aa waves were evident during lateral tricuspid annulus interrogation.

The patient was suspected to have an atrial cardiomyopathy that was progressing to complete atrial standstill. As there were no clinical signs, the owner declined additional diagnostic evaluation. Additional treatments were not recommended, but treatment with enalapril was continued as previously prescribed.

Fourteen weeks later, the patient was presented to the Veterinary Teaching Hospital with a 1–2 week history of increased breathing rates and recent onset of several syncopal episodes. During evaluation earlier in the day at an emergency facility, the dog was bradycardic and was diagnosed with pleural and peritoneal effusion. All electrolytes were normal. Evaluation of thoracic radiographs revealed cardiomegaly, (vertebral heart score 11.6) with evidence of left atrial enlargement, a small volume of pleural effusion and a mild to moderate interstitial pulmonary pattern suggestive of early pulmonary edema.

Physical examination revealed a heart rate of 35 beats per minute, hyperkinetic femoral arterial pulse, and unchanged heart murmurs. The patient was panting throughout the examination and had mild abdominal distention.

A recheck electrocardiogram revealed both narrow QRS and wide QRS escape rhythms that had average rates of approximately 30 beats per minute. Distinct, multiform atrial activity was appreciated only in the V4 lead (Fig. 1), with low voltage atrial depolarizations evident in the other chest leads. Evaluation of standard limb lead recordings did not reveal P waves. Given the changing morphology, these deflections were suspected to be P' waves arising from ectopic atrial foci. During echocardiography, a run of accelerated idioventricular rhythm followed by a period of asystole, presumed to reflect post-tachycardia inhibition of normal automaticity related to overdrive suppression was appreciated.

Echocardiography at this time revealed more marked dilation of the left atrium with a left atrial-to-aortic root ratio of 2.3 and a nearly doubled left atrial volume indexed to body weight of 7.1 ml/kg. Left ventricular end-diastolic dimensions had increased by 1.5 cm since the previous study, whereas end-systolic dimensions were unchanged. Based on subjective evaluation, the right ventricle remained normal in size, whereas

^c SNAP 4Dx Test; IDEXX, Westbrook, ME.

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