Journal of Veterinary Cardiology (2015) **17**, 229—236

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Journal of Veterinary Cardiology
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CASE REPORT

Myocardial infarct associated with a partial thickness left atrial tear in a dog with mitral insufficiency☆

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Received 8 October 2014; received in revised form 23 February 2015; accepted 1 April 2015

KEYWORDS
Endocardial splitting;
Congestive heart failure;
Thromboembolism;
Myocardial dyskinesis;

Abstract A 10-year-old male neutered cavalier King Charles Spaniel with a 1-year history of degenerative mitral valve disease presented for dyspnea and severe weakness. He was diagnosed with congestive heart failure, systolic dysfunction, presumptive myocardial infarction and a left atrial thrombus based on thoracic radiographs, electrocardiogram and echocardiographic findings. Clinical signs also suggested right foreleg embolism. The dog was euthanized due to the grave

Presented in part at the 18th International Veterinary Emergency Critical Care (IVECCS) Symposium, San Antonio, Texas, September 2012.

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http://dx.doi.org/10.1016/j.jvc.2015.04.003

Coronary artery occlusion

prognosis and a postmortem evaluation was performed. The postmortem examination confirmed myocardial infarction and was thought to be due to embolic showering from the thrombus attached to a partial thickness left atrial endocardial tear.

Abbreviations

CKCS	Cavalier King Charles Spaniel
H&E	hematoxylin and eosin
LA	left atrium or left atrial
LV	left ventricle or left ventricular
MI	myocardial infarct
MMVD	mitral valve degeneration

A 10-year-old, 9-kg, male neutered cavalier King Charles Spaniel (CKCS) was brought to the Cardiology Service of the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania with a one week history of progressive exercise intolerance, difficulty rising, inappetence and a 4-year history of a cardiac murmur (grade II/VI left apical systolic murmur). Approximately 1 year prior to presentation, the heart murmur was noted to have progressed to a holosystolic, left apical grade IV/VI murmur when he was evaluated for gastrointestinal clinical signs. At that time, no coughing was described, the respiratory rate and efforts were normal, and strong and synchronous femoral pulses were present. Thoracic radiographs revealed mild left atrial (LA) enlargement with a thin pleural fissure line noted within the left hemithorax. No evidence of congestive heart failure was noted and no cardiac medications were recommended. For several months prior to the current visit, the owners described occasional, non-productive coughing.

On physical examination the dog was mentally dull and depressed. He was normothermic (rectal temperature 38.7 °C) and laterally recumbent. Increased respiratory effort was present and the respiratory rate was 60 breaths per minute. Mucous membranes were cyanotic. Thoracic auscultation revealed a grade II/VI holosystolic murmur with the point of maximum intensity over the left apical precordium and increased intensity of bronchovesicular sounds in all fields. Femoral and dorsal metatarsal pulses were not palpable. Furosemide (2 mg/kg) was administered intravenously, and he was placed in an oxygen cage. Oxygen was supplemented via an oxygen mask during diagnostic studies.

An ECG demonstrated a mild sinus tachycardia at a rate of 150 beats per minute with occasional ventricular premature complexes and ST segment elevation. Thoracic radiographs revealed marked generalized cardiomegaly (vertebral heart size = 13.2) with more severe LA enlargement than on previous radiographs. Caudal lobar pulmonary vascular congestion, pleural fissure lines and a moderate alveolar—interstitial pulmonary pattern were also evident (Fig. 1). Two-dimensional echocardiography[d] confirmed severe LA enlargement (aortic root diameter = 1.5 cm; LA diameter = 3.8 cm; LA/aorta = 2.6), with moderate left ventricular (LV) dilation (diastolic LV internal diameter = 4.4 cm; systolic LV internal diameter = 3.7 cm; see Table 1 for a comparison of this patient's echocardiographic measurements normalized to body weight) and poor systolic function (shortening fraction: 15.5% and peak mitral regurgitant velocity 220.8 cm/s). The caudal region of the LV free wall was akinetic in addition to generally reduced LV contractility (Fig. 2). Mitral valve thickening was apparent, with decreased motility of the posterior leaflet and severe mitral regurgitation. The tricuspid valve leaflets also appeared moderately thickened with moderate tricuspid regurgitation and mild pulmonic insufficiency. Two mixed echogenicity masses were visible within the LA and appeared to be adhered to the LA wall in the right parasternal and left apical views. Based on the anatomic position within the LA, they were deemed most consistent in appearance with atrial thrombi (Fig. 3). The aortic peak flow velocity was severely decreased (44.3 cm/s), as was the mitral regurgitant peak flow velocity (221 cm/s). The tricuspid regurgitant jet velocity was consistent with normal pulmonary pressure (234 cm/s). No clinically important pericardial effusion was noted.

Following diagnostics and within 1 h of presentation, significant postural deficits in the right forelimb were identified that had either occurred since presentation or were not previously noted due to the dog being recumbent. When encouraged into a standing position, the right foreleg remained knuckled over but the other limbs were able to provide support. Metacarpal arterial pulses in this limb were not palpable (and femoral pulses were

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