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BRIEF COMMUNICATION

Third degree atrioventricular block and sudden death secondary to acute myocarditis in a dog

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Abstract Third degree atrioventricular (AV) block in dogs is thought to be most frequently characterized by non-specific fibrotic changes in the AV node. However, it may occur secondary to an undiagnosed inflammatory process. We report a case of third degree AV block in a dog, secondary to acute lymphocytic-plasmacytic myocarditis that resulted in sudden death. This dog had cardiac troponin I levels of 44.65 ng/mL (normal <0.11 ng/mL). The serum cardiac troponin I level was five times higher than any other AV block patient measured in our laboratory, and was also substantially higher than in dogs with chronic valve disease or dilated cardiomyopathy. The severe myocardial necrosis observed at necropsy correlated with the degree of cardiac troponin I elevation. This report suggests that measurement of cardiac troponin I may be an indicated test before pacemaker implantation in dogs with third degree AV block.

The majority of cases of third degree atrioventricular (AV) block in dogs are idiopathic and are characterized by non-specific fibrotic changes in the AV node.[1] We report a case of third degree AV block with ventricular arrhythmias secondary to acute lymphocytic-plasmacytic myocarditis that resulted in sudden death shortly after permanent pacemaker implantation. Though myocarditis is often associated with cardiac conduction disturbances, it is rarely

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identified as the proximate cause of acquired third degree AV block. Endomyocardial biopsy is required for definitive antemortem diagnosis of myocarditis but is rarely performed, in part, because of poorly defined clinical indications warranting its application. In this case, high serum troponin I concentrations, discovered after the fatal outcome, suggest that such assays might be helpful in distinguishing dogs with AV block due to active myocarditis.

Case report

An 8-year-old, male, castrated, 24 kg, Springer Spaniel presented to the referring veterinarian with a 2-week history of cough and a 3-day history of decreased activity, dyspnea, inappetence, and polydypsia. Based on the physical exam finding of bradycardia, an ECG was performed, revealing third degree AV block. The dog was subsequently referred to the University of Illinois Veterinary Teaching Hospital for evaluation as a candidate for pacemaker implantation. The patient was fed a lamb and rice diet for food allergies, had not traveled out of the state of Illinois, and was receiving no medications other than lufenuron for flea prevention. On presentation, auscultation revealed a heart rate of 36 bpm with occasional premature beats and periods of paroxysmal tachycardia (140 bpm). No murmurs or gallop rhythms were detected. The respiratory rate was 60/min and increased bronchovesicular sounds were detected. An ECG revealed third degree AV block with a monomorphic ventricular escape rhythm, frequent premature ventricular complexes (Fig. 1) and paroxysms of ventricular tachycardia. Thoracic radiographs demonstrated moderate pulmonary venous congestion, a moderate perihilar interstitial pattern, but a normal cardiac silhouette. A complete blood count revealed a stress leukogram and serum chemistries were within normal limits. An echocardiogram revealed mild hypertrophy of the left atrium (LA/Ao = 1.5), mild mitral regurgitation with thickening of both mitral leaflets, and normal fractional shortening (29.3%). There was decreased systolic wall motion of the anterior portion of the interventricular septum.

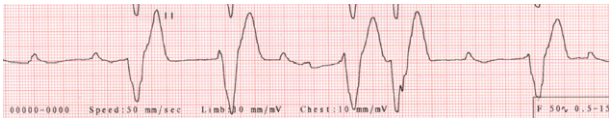

Figure 1 Electrocardiogram at presentation to University of Illinois VTH Emergency Service, showing third degree AV block with a monomorphic ventricular escape rhythm and frequent premature ventricular complexes.

Implantation of an artificial pacemaker was recommended. Initial therapy included placement of a transvenous temporary pacemaker, intravenous furosemide (2 mg/kg), nasal oxygen supplementation and intravenous cefazolin (22 mg/kg). The following morning, a permanent bipolar 58 cm tined lead[d] was placed in the right ventricular apex and VVI pacing was accomplished using a pulse generator[e] programmed to 95 bpm. Post-operatively the temporary pacing lead was removed and the intravenous furosemide and cefazolin were continued. Thoracic radiographs were performed immediately after permanent pacemaker implantation to document lead position within the right ventricular apex. A post-operative ECG revealed abnormally prolonged QRS complexes that were negatively deflected in leads II, III and aVF (Fig. 2). The QRS duration was 140 ms with a QT interval of 320 ms. One ventricular couplet was noted on a post-operative rhythm strip recording. An electrocardiographic telemetry system was used to monitor heart rate and rhythm while the patient recovered in the intensive care unit.

The patient paced consistently at 95 bpm and slept comfortably with a respiratory rate of 28/min until 9 h post pacemaker implantation when the patient suddenly developed ventricular fibrillation. External electrocardioversion was temporarily successful in restoring a normal paced rhythm, but the dog died approximately 30 min after resuscitation attempts were instituted.

Client consent was granted to perform a post-mortem examination. Gross necropsy of the heart revealed multifocal pale regions of apparent myocardial necrosis of the left ventricular posterior wall, papillary muscles, interventricular septum and right ventricular wall. The pacing lead was appropriately positioned within the right ventricular apex. Mild degenerative mitral valve disease was present and there was no evidence of pulmonary thromboembolism. Due to the gross necropsy findings and apparent myocardial necrosis, cardiac troponin I concentration was measured[f] using serum that had been obtained prior to pacemaker implantation. Serum cardiac troponin I concentration was approximately 400 times greater than the upper reference value for our laboratory (44.65 ng/mL, normal $<$0.11 ng/mL). Histopathologic examination of the necrotic myocardial regions revealed severe lymphocytic-plasmocytic myocarditis with mild to moderate numbers of eosinophils, evidence of fibrinous vasculitis, and large areas of

[d] Capsure Z 5034, Medtronic, Minneapolis, MN, USA.
[e] Kappa KSR 401/403, Medtronic, Minneapolis, MN, USA.
[f] Access AccuTnI, Beckman Coulter Inc., Fullerton, CA, USA.