Atrial cardiomyopathy in an adult Labrador retriever dog

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Abstract

A 7-year-old castrated male Labrador retriever was examined for a 10-day history of weakness and syncope. Physical examination revealed bradycardia and a grade III/VI left apical systolic heart murmur. Electrocardiography demonstrated bradycardia, absence of P waves and an atrio-ventricular nodal escape rhythm. Echocardiography revealed marked biatrial enlargement. Thoracic radiographs showed no evidence of pulmonary edema. Routine plasma biochemistry and electrolytes, basal serum cortisol, total thyroxin concentration, and complete blood count were within normal limits. Serum cardiac troponin I concentration was moderately increased. Serological examinations for antibodies against vector-borne diseases were negative. A pacemaker was implanted one month after the initial presentation due to worsening of the dog’s clinical condition despite medical treatment. The dog remained asymptomatic for 18 months but was then re-presented with a gastric dilatation volvulus and subsequently euthanized. Necropsy and histology of the heart yielded a diagnosis of atrial cardiomyopathy.

Keywords: artificial pacing, bradyarrhythmia, canine, heart

Atriale Kardiomyopathy bei einem erwachsenen Labrador Retriever


Schlüsselwörter: Bradykardie, Herz, Herzschrittmacher, Hund
**Case history**

A 7-year-old 35 kg, neutered male Labrador retriever dog was examined for a 10-day history of weakness and syncpe (5-6 per day). On physical examination, the dog was in good clinical condition. Cardiac auscultation revealed bradycardia (heart rate: 50 beats/min) and a grade III/VI left apical systolic heart murmur. The rest of the physical examination was unremarkable.

**Electrocardiography**

Standard 6-lead electrocardiography (ECG) showed bradycardia, absence of P wave and a regular (ie, RR interval variation less than 10%) atrio-ventricular nodal escape rhythm at a rate of 58 beats/minute (bpm, Fig. 1).

**Echocardiography**

Conventional echocardiography and standard Doppler examination were performed on the awake dog in standing position with an ultrasound unit (Megas and My Lab Twice, Esaote Biomedica, Firenze, Italy). The left ventricular M-mode echocardiogram obtained from the right parasternal transventricular short-axis view revealed a moderate left ventricular dilation in diastole (51.0 mm) with normal systolic diameter (26.5 mm) (reference ranges (RR) of 34.0-46.3 mm and 20.5-30.8 mm, respectively, Gonçalvez et al., 2002). The fractional shortening was moderately increased (48%, RR = 29-43%, Chetboul et al., 2005). The two-dimensional right parasternal transaortic short-axis view showed marked left atrial (LA) enlargement (LA to aorta ratio = 2.52, RR <1.6, Rishniw and Erb, 2000). The right atrium appeared dilated on the two-dimensional right parasternal long axis view with a ratio between the right and left atria of 0.8 (Fig. 2). The aspect of all cardiac valves was within normal limits except for mild thickening of the mitral leaflets. The left apical 4-chamber view revealed moderate mitral regurgitation using Color Doppler mode (area of the regurgitation jet signal to LA area < 50%). Finally, pulsed and continuous Doppler mode showed an increased E mitral wave velocity, reflecting LA pressure overload (2.68 m/s, RR: 0.61-1.13 m/s, Chetboul et al., 2005) and the absence of diastolic transmitral atrial phase flow.

**Radiography**

Thoracic radiographs revealed cardiomegaly (vertebral heart scale of 12.5, RR < 10.7, Buchanan and Bücheler, 1995) and pulmonary venous congestion but no signs of cardiogenic pulmonary edema.

**Holter examination**

Twenty-four-hour Holter monitoring (Fig. 3) confirmed the presence of persistent bradyarrhythmia (mean heart rate: 52 bpm) associated with ventricular escape beats at lower heart rate. No episode of syncope was reported during the Holter examination.

**Blood analysis**

Routine plasma biochemistry and electrolytes, serum basal cortisol, total thyroxin concentrations and complete blood count were within normal limits. Serum cardiac troponin I concentration was moderately increased (0.712 ng/mL, RR < 0.06 ng/mL, Laboratoire Idexx, Alfort, France). Finally, a qualitative ELISA test (SNAP 4Dx Test, Idexx Laboratories, Westbrook, USA) failed to detect any antibodies against vector-borne diseases such as erlichiosis, anaplasmosis, dirofilariosis and Lyme disease.

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**Figure 1:** Standard 6-lead electrocardiogram tracing obtained at 25 mm/s and 10 mm/mV. Note the absence of consistently definable P waves and the narrow-complex junctional escape rhythm at a rate of 58 bpm.
Atrial cardiomyopathy in an adult Labrador retriever dog

E. Bomassi et al.

Therapy and follow-up

The dog was medicated per os with benazepril (Fortekor, Novartis Santé Animale, Rueil Malmaison, France), 0.28 mg/kg SID, furosemide (Dimazon, Intervet, Beaucouze, France), 0.57 mg/kg BID, prednisolone (Megadose, Merial, Lyon, France), 0.57 mg/kg BID and doxycycline (Ronaxan, Merial, Lyon, France), 8.6 mg/kg SID. Due to the increase of syncopal episodes (ie, >10 per day), a pacemaker implantation was scheduled one month after the initial presentation. A single chamber, permanent pacemaker (Adapta ADSR01, Medtronic, Minneapolis, USA) with VVIR (ventricular pacing, ventricular sensing, inhibition response and rate-adaptive; lower heart rate: 70 bpm; upper tracking rate: 130 bpm; amplitude: 3.5 V; pulse width: 0.64 ms) combined with a bipolar epicardial wire (CapSure Epi, Medtronics Boulogne-Billancourt, France) was implanted using a standard surgical approach (Monnet 2003). Several left ventricular myocardial biopsies were taken during the procedure. Histopathological examination failed to reveal any degenerative, inflammatory or infectious disease of the ventricular myocardial and epicardial tissues. Prednisolone and doxycycline were discontinued 10 days after surgery. Follow-up included clinical, ECG, Holter and echocardiographic examinations. Despite a decrease in serum cardiac troponin I concentration (0.105 versus 0.712 ng/mL) and ventricular diameter (48.5 versus 51.0 mm) 4 weeks after pacemaker implantation, the atria remained dilated with an LA to aorta ratio of 2.19 (versus 2.52 at first examination). The dog remained asymptomatic and in good clinical condition for 18 months but was then presented with gastric dilatation volvulus resulting in euthanasia. Gross examination revealed the atria to be highly enlarged and discolored with thin walls. The ventricles were moderately dilated and the atrio-ventricular valve leaflets were thickened and irregular. Histological examination of both atria revealed severe extensive replacement fibrosis and atrophic modifications of the residual myofibers that were dissociated by interstitial collagen deposition (Fig. 4). Foci of mild interstitial fibrosis were found in the ventricles.

Discussion

The present report describes an exclusive atrial cardiomyopathy (AC) characterized by fibrous infiltration of the atrial myocardium and atrophy of atrial cardiomyocytes in an adult Labrador retriever dog. The AC was associated with a bradyarrhythmia causing weakness and syncope, which are frequent presenting complaints in dogs with bradyarrhythmia (Wess et al., 2006). Similar AC have been previously described in dogs, including one male crossbred Chow-chow (Une et al., 1998),

Figure 2: Image of a two-dimensional right parasternal long axis 4-chamber view. Note the biatrial enlargement and the moderate thickening of the mitral valve leaflets. LV: left ventricle, RV: right ventricle, RA: right atrium, LA: left atrium.

Figure 3: Extract of the 24-hour Holter examination (leads I and II) obtained at 25 mm/s and 10 mm/mV. Note the absence of consistently definable P waves, the narrow-complex junctional escape rhythm (mean rate of 58 bpm) and the periods of ventricular escape beats.
Atrial cardiomyopathy
in an adult Labrador
triever dog
E. Bomassi et al.

2 mongrels and 4 Springer Spaniels (Buchanan, 2005),
two Labrador retrievers (one female and one male,
Schmitt and Lefbom, 2016) and one Greyhound (Wesse-
sowski et al., 2017). Most of these dogs had clinical
signs compatible with congestive heart failure and brad-yarythmia, such as abdominal distention and syn-cope. In all cases, necropsy and histopathological exam-
ination revealed enlarged, thin-walled and pale atria,
fibrous infiltration of the atrial myocardium (with or
without fatty degeneration) and atrophy and loss of the
atrial cardiomyocytes (Une et al., 1998; Buchanan,
2005; Schmitt and Lefbom, 2016; Wesselowski et al.,
2017). Some dogs showed different degrees of chronic
inflammation of the atrial myocardium, associated in a
few dogs with concomitant atrial and ventricular in-
volve ment (Buchanan, 2005). Unlike the dog in the
present report, most of these dogs were young, i.e., be-
tween 12 and 36 months old, and one dog was 4.5 years
(versus 7 years in the present case). This suggests that
AC may have different etiologies, as observed in humans
(Goette et al., 2016). Interestingly, 8 of the 10 dogs were
diagnosed with atrial standstill, confirmed by ECG (Une
et al., 1998; Buchanan, 2005; Wesselowski et al., 2017).
Thus, this arrhythmia seems to be frequently associ-
ated with AC, as already highlighted in several other reports
in which AC was suspected (MacAulay, 2002; Nakamu-
ra et al., 2012; Serene, 2012; Thomason et al., 2016;
Cervenec et al., 2017). In the present case, ECG exam-
ination revealed an absence of definable P waves, a near-
ly regular junctional escape rhythm and ventricular
escape beats. Differentials included therefore atrial
standstill, sinus arrest and atrial fibrillation with com-
plete atrio-ventricular block that might have been dif-
ferentiated by carrying out an electrophysiologic study
(not performed here for practical reasons). In the present
case, and in agreement with previous reports (Schmitt
and Lefbom, 2016; Wesselowski et al., 2017), echocar-
diography was relatively non-specific of AC and showed
marked bia trial enlargement, mild ventricular diastolic
dilation (which could be attributed to bradycardia) with
nearly normal fractional shortening and moderate mit-
ral valve regurgitation. The latter was probably sec-
dary to both mitral annulus dilation and degeneration of
the mitral valve leaflets because this LA enlargement
could not be explained by the magnitude of regurgita-
tion alone. Moreover, the moderate increase of serum
troponin I concentration reflected mild myocardial in-
jury and excluded active myocarditis. Nevertheless, the
evolution of previous subclinical atrial myocarditis into
chronic AC could not be excluded with certainty. Final-

Figure 4: Microscopic aspect of the right atrial wall at low magnification. Severe extensive interstitial fibrosis dissociating and
replacing the myocardial fibers. Residual myofibers are dissociated by interstitial collagen deposition and exhibit atrophic changes.
Masson trichrome stain, original magnification ×10 (A), ×40 (B), ×100 (C), ×200 (D)
Atrial cardiomyopathy in an adult Labrador retriever dog

E. Bomassi et al.

Fallberichte | Case reports

Atrial cardiomyopathy (AC) is a chronic cardiac disease characterized by structural, architectural, contractile or electrophysiological changes affecting the atria with the potential to produce clinically-relevant manifestations. In humans, AC may be primary (e.g., isolated atrial amyloidosis, ‘lone’ atrial fibrillation, hereditary muscular dystrophies, mutation of the gene encoding for the precursor protein for atrial natriuretic peptide) or secondary to various cardiac and extra-cardiac diseases such as congestive heart failure, cardiac valvulopathies, myocarditis, diabetes mellitus, obesity and ageing. In the present case, no underlying cause was identified and the AC was considered idiopathic. This report has several limitations. Firstly, skeletal muscle biopsies could have been done to explore muscular dystrophy but were not envisaged in view of the absence of clinical signs related to muscular disease. Moreover, an electrophysiological study would have been interesting to characterize the bradycardia as well as advanced echocardiographic techniques such as speckle tracking imaging to evaluate atrial function. In conclusion, the present case is the third AC confirmed for managing bradycardia-associated syncope in this uncommon heart disease. Further studies are needed to better understand AC and possibly identify a breed predisposition in Labrador retriever dogs, as already suggested in English Springer Spaniels.

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References


Atrial cardiomyopathy in an adult Labrador retriever dog

E. Bomassi et al.


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Atrial cardiomyopathy in an adult Labrador retriever dog
E. Bomassi et al.