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Case Report

Hypertrophic cardiomyopathy in a dog: a systematic diagnostic approach☆

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Abstract A seven-year-old female neutered Parson Russel terrier was referred for syncopal episodes. An electrocardiogram revealed paroxysmal atrial flutter followed by periods of sinus arrest, suggesting sick sinus syndrome. Echocardiography showed severe biventricular wall thickening (hypertrophic cardiomyopathy (HCM) phenotype) with no signs of fixed or dynamic left ventricular outflow tract obstruction. Blood pressure, abdominal ultrasound, serum total thyroxin and thyroid-stimulating hormone, and insulin-like growth factor-1 were all within normal limits. Cardiac troponin I was elevated (1.7 ng/mL, ref<0.07). Serological tests for common infectious diseases were negative. A 24-h Holter confirmed that the syncopal episodes were associated with asystolic pauses (sinus arrest after runs of atrial flutter) ranging between 8.5 and 9.6 s. Right ventricular endomyocardial biopsies (EMB) were performed at the time of pacemaker implantation to assess for storage or infiltrative diseases that mimic HCM in people. Histological analysis

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of the EMB revealed plurifocal inflammatory infiltrates with macrophages and lymphocytes (CD3+ > 7/mm^2) associated with myocyte necrosis, but no evidence of myocyte vacuolisation or infiltrative myocardial disorders. These findings were compatible with myocardial ischaemic injury or acute lymphocytic myocarditis. Molecular analysis of canine cardiotropic viruses were negative. The dog developed refractory congestive heart failure and was euthanised 16 months later. Cardiac post-mortem examination revealed cardiomyocyte hypertrophy and disarray with diffuse interstitial and patchy replacement fibrosis, and small vessel disease, confirming HCM. We described a systemic diagnostic approach to an HCM phenotype in a dog, where a diagnosis of HCM was reached by excluding HCM phenocopies.

Abbreviations

BPM	beats per minute
ECG	electrocardiogram
EMB	endomyocardial biopsies
HCM	hypertrophic cardiomyopathy
LV	left ventricular
RV	right ventricular

A seven-year-old female neutered Parson Russel terrier was referred to the Queen's Veterinary School Hospital, University of Cambridge, for further investigation of syncopal episodes. The owners described several episodes of transient loss of consciousness with spontaneous recovery to normal mentation and activity levels within seconds.

On presentation, the dog was bright, alert and responsive, weighing 8.3 kg with a body condition score of 5/9. Mucous membranes were pink and moist. Cardiac auscultation revealed an arrhythmia with periods of relative bradycardia [80 beats per minute (bpm)] interspersed with a fast, regular tachycardia (200 bpm). A 2/6 left apical systolic heart murmur was also detected. Respiratory rate was 28 breaths per minute. Pulmonary auscultation revealed normal bronchovesicular sounds. Femoral pulses were weak but synchronous with heartbeat. Abdominal palpation, peripheral lymph nodes and the remaining physical examination were all unremarkable.

A six-lead electrocardiogram (ECG) revealed paroxysmal supraventricular tachycardia with ventricular rate around 200 bpm, followed by periods of sinus bradycardia (70 bpm) or sinus arrest. The later was interrupted by ventricular escape beats (Fig. 1). The supraventricular tachycardia was characterised by sudden onset and termination, F waves of variable morphology with no isoelectric line between consecutive F waves, very rapid atrial rate, and it was an unstable rhythm with rapid and spontaneous return to sinus rhythm. We speculated, therefore, this to be a functional atrial flutter (type II Wells) [1]. The ECG findings suggested sick sinus syndrome with a tachycardia—bradycardia pattern.

Echocardiography showed severe concentric and symmetrical left ventricular (LV) wall thickening (Fig. 2, Video 1) (interventricular septum at end diastole measured 15 mm, normalised 0.94 (body-weight normalised, ref < 0.44)); LV free wall at end diastole measured 14 mm, normalised 0.85 (body-weight normalised, <0.47) [2], with hyperdynamic systolic function (LV fractional shortening 38%, ref > 25; LV ejection fraction 63%, ref > 40). The LV myocardium was subjectively hyperechoic. There was no LV outflow tract obstruction (Vmax 1.7 m/s, ref < 2.0), and an aortic coarctation was also not detected. Aortic valve was structurally normal (tricuspid) with a normal motion. There was mild mitral valve regurgitation, but the mitral valve apparatus was structurally normal and there was no systolic anterior motion of the mitral valve. Left atrium was mildly dilated (left atrium-to-aorta ratio 1.7, ref< 1.6). Right ventricular (RV) free wall was also subjectively thickened without evidence of RV outflow tract obstruction (Vmax 1.1 m/s, ref < 2.0). Mitral valve inflow showed a delayed relaxation pattern (mitral valve inflow ratio E/A 0.6, ref. 1—2) [3]. Non-invasive systolic blood pressure was within normal limits (140 mmHg) and fundic examination was unremarkable with no signs of hypertensive retinopathy. Haematology, serum biochemistry (including C-reactive protein), and urinalysis (including urine protein:creatinine ratio) were all within normal limits. Thoracic radiographs showed generalised cardiomegaly with normal lung fields.

Considering the presence of a hypertrophic cardiomyopathy (HCM) phenotype, our differential

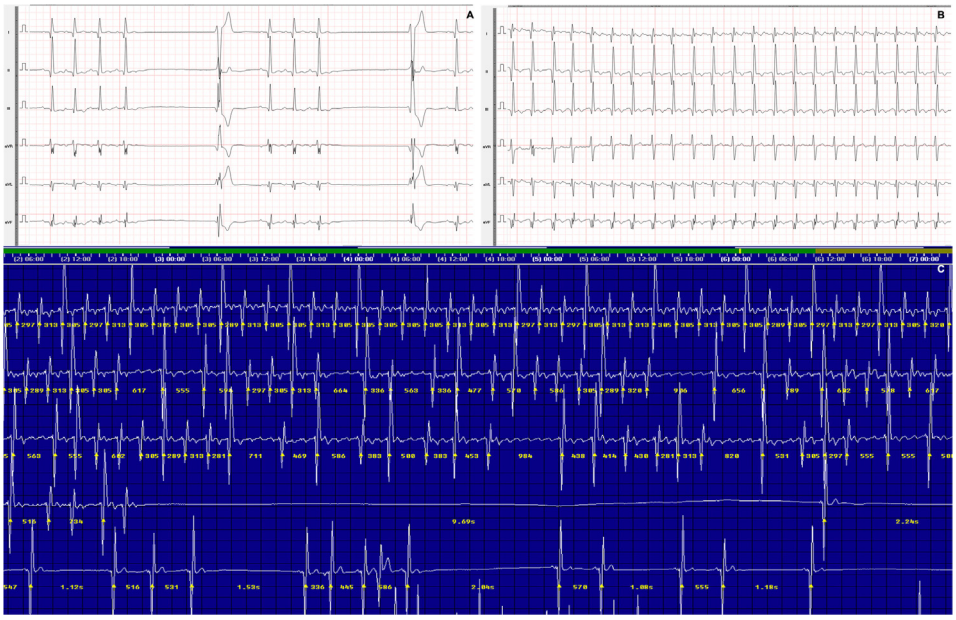

Fig. 1 (A) sinus rhythm with two episodes of sinus arrest interrupted by ventricular escape beats; (B): Atrial flutter with an average ventricular rate of 200 bpm; (C) ECG trace from a 24-h Holter recording showing paroxysmal atrial flutter followed by asystolic pause (sinus arrest) lasting 9.7 s that was associated with syncope. After the long pause, sinus rhythm resumes with frequent shorter pauses (ECG traces A and B recorded at 50 mm/s, 20 mm/mV).

diagnoses at this stage included (primary) HCM, hyperthyroidism, acromegaly, pheochromocytoma, acute myocarditis, diffuse myocardial neoplastic infiltration (e.g. lymphoma), and other conditions not yet described in dogs such as cardiac amyloidosis, storage diseases or mitochondrial cardiomyopathies. An abdominal ultrasound was unremarkable. Serum total thyroxin (33 mol/L, ref 13—52), thyroid-stimulating hormone 0.09 ng/mL (ref $<$ 0.41), and insulin-like

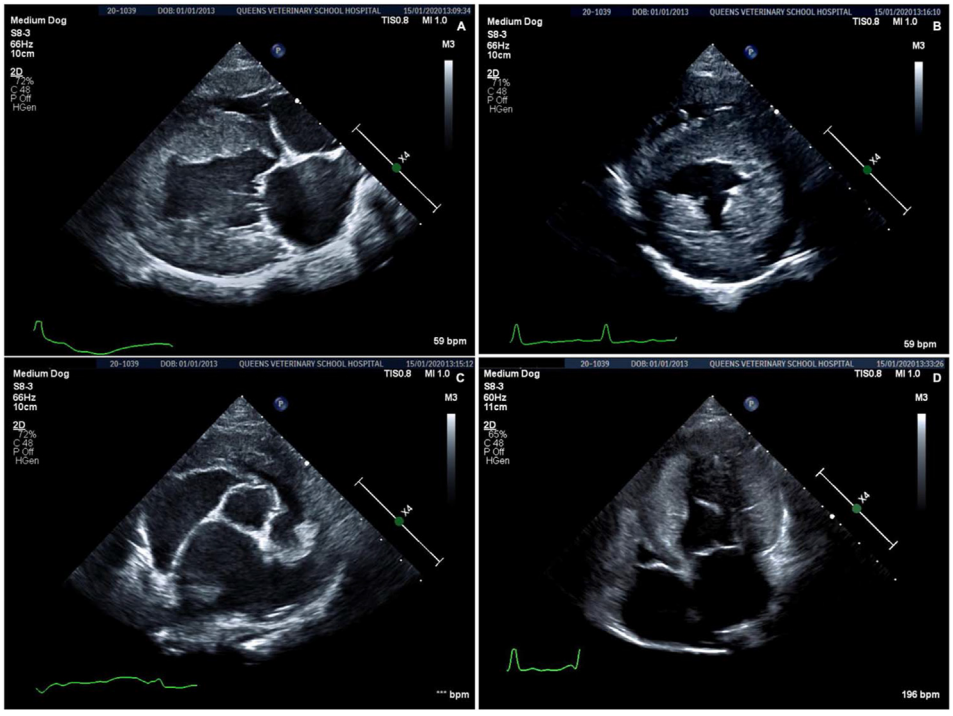

Fig. 2 Echocardiographic views of a dog with hypertrophic cardiomyopathy showing severe left ventricular and moderate right ventricular hypertrophy with mild left atrial dilation. Right parasternal long-axis view (A); right parasternal short-axis view at the level of the papillary muscles (B) and heart base (C); left apical four-chamber view (D).

growth factor-1 189 ng/mL (ref < 1000 ng/mL) were within normal limits. Serum cardiac troponin I was elevated (1.7 ng/mL, ref < 0.07). Serological tests for infectious diseases, including *Ehrlichia canis, Anaplasma phagocytophilum, Borrelia burgdoferi, Bartonella vinsonii* (immunofluorescence, IFA), *Bartonella henselae* (immunofluorescence, IFA), and *Bartonella koehlarae* (immunofluorescence, IFA), and *Toxoplasma gondii* were all negative.

A 24-h Holter ECG was performed to fully characterise the documented arrhythmia and the cause of the syncopal episodes. Frequent runs of paroxysmal atrial flutter, followed by sinus arrest were present throughout the 24-h recording. Three syncopal events were reported during the Holter monitoring and they were associated with asystolic pauses (sinus arrest) lasting up to 9.6 s that followed runs of atrial flutter (Fig. 1). There was one episode of sustained atrial flutter lasting 30 min. Additionally, there were 248 episodes of sinus standstill with a ventricular escape rhythm ranging between 30 and 40 bpm (mean 24-h HR was 66 bpm, max HR 270 bpm, and min HR 30 bpm). The Holter results confirmed sick sinus syndrome, with syncopal events caused by prolonged pauses (sinus arrest with ventricular asystole). Considering our findings, pacemaker implantation was recommended and RV endomyocardial biopsies (EMB) were planned at the time of pacemaker implantation to assess for storage and infiltrative myocardial diseases.

Pacemaker implantation and EMB were performed three weeks after the initial presentation. The dog was anaesthetised, and vascular access was performed through a surgical cut-down of the right jugular vein. Once isolated, vascular access was obtained using a modified Seldinger technique with a 9 Fr vascular introducer.[d] The RV was catheterised using a 9 Fr guiding catheter[e] and a 260 cm long 0.035 J-tip guidewire.[f] Four EMB were taken under fluoroscopy and transoesophageal echocardiography guidance targeting the interventricular septum using a 7 Fr biopsy forceps[g] (104 cm, jaw volume 5.2 mm^3). A run of atrial flutter with high grade atrioventricular block developed during EMB which spontaneously terminated after a few minutes and returned to normal sinus rhythm. Following the EMB, a bipolar, transvenous pacemaker[h] was implanted with a passive lead in the RV apex and the pulse generator was routinely positioned on a SC pocket on the right cervical region. Pacemaker was programmed to VVI mode with a pacing rate of 70 bpm with hysteresis set at 40 bpm, output 3.5 V/0.4 ms, and sensitivity 5.6 mV. Recovery from anaesthesia was uneventful. Cefazolin was administered intravenously during the procedure and the dog was then kept on oral amoxicillin with clavulanic acid for two weeks (16 mg/kg PO q 12 h). Seven hours post-procedure the dog developed tachypnoea and sustained tachycardia, a point-of-care ultrasound showed moderate pleural effusion, a severely dilated right atrium with spontaneous echocardiographic contrast, and caudal vena cava and hepatic veins distension. An ECG revealed sustained atrial flutter with a ventricular rate of 245 bpm. Thoracocentesis yield 120 mL of a 'milky white' fluid. Fluid analysis revealed chylous effusion. The dog was started on furosemide[i] (2 mg/kg IV q 6 h for 24 h, and then changed to 1.8 mg/kg PO q 12 h), benazepril/spironolactone[j] (0.3–2.5 mg/kg PO q 12 h), sotalol (1.2 mg/kg PO q 12 h) and clopidogrel (2.3 mg/kg PO q 24 h). An echocardiogram 48-h post-procedure revealed similar findings to the previous scan: severe LV and RV thickening with mild-moderate biatrial enlargement, persistent spontaneous echocardiographic contrast in the right atrium but no evidence of thrombosis. Pleural effusion had resolved. Pacemaker lead was well positioned at the RV apex.

Histological analysis of EMB samples revealed plurifocal inflammatory infiltrates with macrophages and lymphocytes (CD3+ > 7/mm^2) associated with myocyte necrosis (Fig. 3). Mean cardiomyocyte diameter was 15 μm (range 13–19 μm). Ultrastructural cardiomyocyte analysis (transmission electron microscopy) showed focal loss of sarcomeres and interstitial inflammatory lymphocytic cells. There was no evidence of myocyte vacuolisation or infiltrative myocardial disease. These findings were compatible with a myocardial ischaemic injury or an acute lymphocytic myocarditis [4,5].

Molecular analysis of common canine cardiotropic viruses was performed on the myocardial samples. Polymerase chain reaction for canine coronavirus, canine herpesvirus 1, canine

[d] Standard sheath introducer AVANTI+, Cordis, Cardinal Health UK, Buckinghamshire, UK.
[e] Guiding catheter MPA I, Vista Brite Tip, Cordis, Cardinal Health UK, Buckinghamshire, UK.
[f] J-tip Fixed-Core Wire Guide, Safe-T-J, Cook Medical, Limerick, Ireland.
[g] Standard biopsy forceps, Cordis, Cardinal Health UK, Buckinghamshire, UK.
[h] IPG Sphera SR MRI Surescan, Medtronic, Medtronic Limited, Watford, UK.
[i] Dimazon, MSD Animal Health UK Ltd, Milton Keynes, UK.
[j] Cardalis, Ceva Santé Animale, Libourne, France.

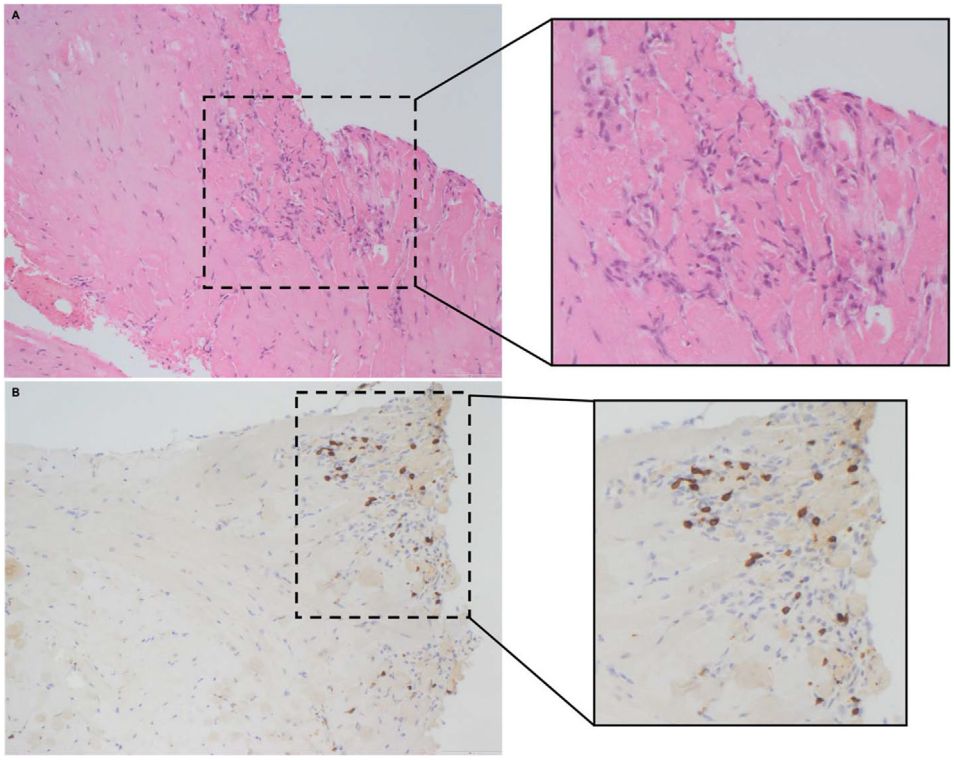

Fig. 3 Histopathologic images of endomyocardial biopsy samples in a dog with hypertrophic cardiomyopathy phenotype showing plurifocal inflammatory infiltrates and myocyte necrosis, but no evidence of storage or infiltrative diseases. (A) The inset represents magnified area showing the inflammatory infiltrate (cells with blue nuclei and scanty cytoplasm) encircling necrotic cardiomyocytes (myocytes with hypereosinophilic cytoplasm and loss of nuclei); Haematoxylin and eosin staining; (B) Immunohistochemistry showing T lymphocytes CD3+ ($>7/mm^2$) stained in brown (inset represents magnified area).

distemper virus, canine adenovirus 1 and 2, and canine parvovirus 2 were all negative.

The dog was rechecked two weeks after the procedure. The syncopal events had resolved, and she had a good exercise tolerance. Serum cardiac troponin I remained elevated (1.4 ng/mL, ref $<$ 0.07). Pacemaker interrogation revealed adequate lead impedance, sensing and capture thresholds. A 24-h Holter monitor revealed a paced rhythm 58% of the time with occasional paroxysmal atrial flutter (maximal HR 187 bpm). The dog was kept on the same treatment regime.

The dog was followed for 1.5 years with frequent echocardiograms, Holter monitoring and pacemaker interrogations. The LV and RV remained severely thickened over time. The dog had frequent relapses of congestive heart failure (chylous pleural effusion), which resolved with increased diuresis. There was progressive bi-atrial enlargement and atrial fibrillation developed 10 months after the initial presentation. Pacemaker function remained adequate throughout the follow-up period. Cardiac troponin I remained elevated (1.4 ng/mL, 1.8 ng/mL and 1.3 ng/mL, respectively, three, seven and 10 months after initial presentation). Twelve months after pacemaker implantation, a small non-obstructive thrombus was detected attached to the pacemaker lead.

The dog was euthanised 16 months after initial presentation due to refractory congestive heart failure and azotaemia; treatment regime at the time was: torasemide[k] (0.8 mg/kg q 12 h), furosemide (3.3 mg/kg q 24 h SC), sotalol (1.3 mg/kg q 12 h), sacubitril/valsartan[l] (3 mg/kg − 3.3 mg/kg q 24 h)[x], clopidogrel (2.3 mg/kg q 24 h), aspirin (2.3 mg/kg q 24 h), and potassium gluconate[m] supplementation (0.6 mEq/kg q 12 h). Post-mortem examination was performed with the owner's consent. Gross pathology revealed severe biventricular hypertrophy (interventricular septum of 14 mm, LV free wall thickness of 13 mm and RV free wall 7 mm) (Fig. 4). Heart weight was 136 g, heart weight/body weight ratio 1.7% (ref 0.7%) [6]. The pacemaker lead was attached to the RV apex with a small thrombus at the level of

[k] UpCard, Vetoquinol SA, Lure, France.
[l] Entresto, Novartis Pharmaceuticals UK, London, UK.
[m] Kaminox, VetPlus, Lytham, UK.

the right atrium. On histopathology there was cardiomyocyte hypertrophy and disarray with diffuse interstitial and patchy replacement fibrosis at the LV free wall. There were also patchy areas of myocytolysis, and intramural small vessels with medial hypertrophy and intimal thickening (small vessel disease) (Fig. 5). Focal inflammatory infiltrates not associated with myocyte necrosis were observed. Histological findings confirmed HCM.

Discussion

We described a dog with an HCM phenotype and sick sinus syndrome where a systematic and thorough diagnostic approach confirmed primary HCM.

Hypertrophic cardiomyopathy is defined as LV thickening in the presence of normal loading conditions [7]. It is the most common heart disease in cats and the most common inherited cardiovascular disease in humans, but it is rare in dogs [2,7,8]. In people, HCM is a familial disease caused by sarcomeric mutations [7]. In cats only a few mutations have been described [9—11], and the aetiology in dogs remains unknown. In people, several diseases cause myocardial wall thickening mimicking HCM, such as acute myocarditis [7,12], neoplastic infiltration [13], storage diseases [14] and amyloidosis [7,15]. However, there are only scarce descriptions of HCM phenocopies in cats and dogs [16—20].

Echocardiography is the clinical gold standard to diagnose HCM [2,7], but further investigations are required to exclude HCM phenocopies [7,21]. Endomyocardial biopsies are not routinely used for the diagnosis of HCM in people, but it can be considered when there is a suspicion of

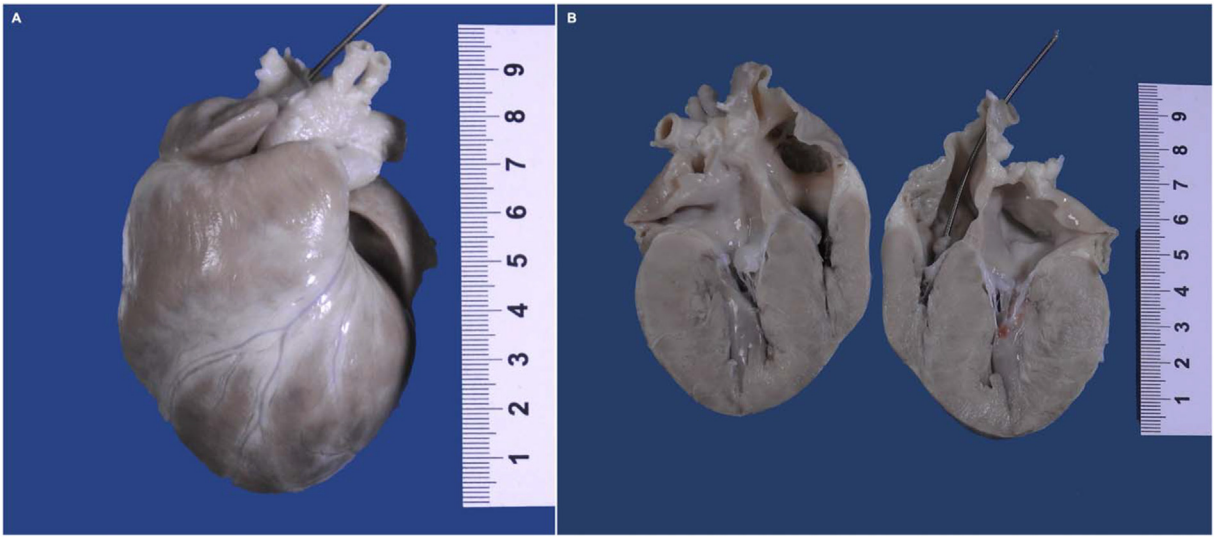

Fig. 4 Gross pathologic images of a dog with hypertrophic cardiomyopathy showing severe left ventricular and moderate right ventricular hypertrophy with moderate bi-atrial dilation. A pacemaker lead can be seen attached to the right ventricular apex with a small thrombus at the level of the tricuspid valve.

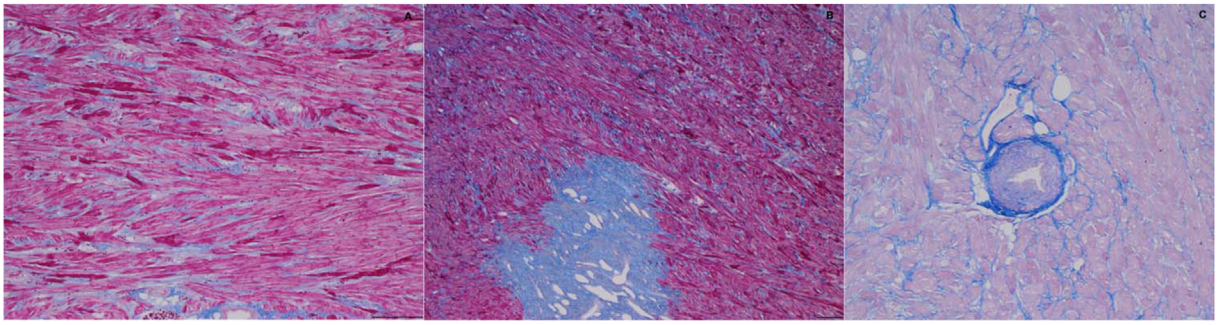

Fig. 5 Histopathologic images of a dog with hypertrophic cardiomyopathy showing cardinal features of this disease, namely myofiber disarray (A), a large area of replacement fibrosis (Masson's trichrome staining showing fibrosis in blue) (B), and an intramural small coronary artery with marked medial hypertrophy and intimal thickening (small vessel disease) (C). Masson's trichrome staining.

infiltrative or storage diseases or myocarditis [7,22]. There is scarce data on EMB in dogs with two studies showing a low rate of complications [23,24], but cardiac arrest during EMB is reported in a dog with myocarditis [19]. The dog here described had an HCM phenotype characterised by severe LV and RV thickening and sick sinus syndrome. As an HCM phenotype is rare in dogs, we performed EMB at the time of pacemaker implantation to exclude HCM phenocopies commonly described in humans, namely infiltrative (e.g. amyloidosis, neoplasia), storage (e.g. Fabry's) and mitochondrial diseases and acute myocarditis [7]. Several RV EMB were taken for histological analysis and transmission electron microscopy, and there were no signs of infiltrative, storage or mitochondrial diseases. Histopathological analyses showed focal interstitial inflammatory infiltrates associated with myocyte necrosis suggesting either a myocardial ischaemic injury or acute myocarditis. Interstitial inflammatory infiltrates with myocyte necrosis have been frequently described in human HCM associated with ischaemic damage [5]. Similarly, focal myocardial inflammatory cell infiltrates, predominantly lymphocytes, have been described in cats and dogs with HCM [25,26]. The dog here presented was euthanised 1.5 years after presentation due to refractory congestive heart failure (stage D), and a full cardiac post-mortem examination showed classic histological features of HCM, namely myocardial disarray, small vessel disease, and myocardial fibrosis [5] confirming primary HCM.

This dog's heart failure manifested as chylous effusion with caval and hepatic congestion suggesting right-sided heart failure. Cats with HCM in stage C can frequently have pleural effusion, but pleural effusion is an uncommon manifestation of heart failure in dogs [27]. In our case, several factors might have contributed to the occurrence of right-sided heart failure, namely presence of marked right ventricular thickening/dysfunction, brady- and tachyarrhythmias, and non-physiologic ventricular-demand (single chamber) cardiac pacing. Additionally, the presence of pulmonary hypertension cannot be completely ruled out.

A recent study in dogs with HCM showed an overrepresentation of terrier breeds, and both supraventricular tachyarrhythmias and atrioventricular blocks were described in that cohort [2], which matches our findings. We have not performed histological analysis of the conduction system or atrial tissue, which is a limitation of this manuscript.

We described a systematic diagnostic approach in a dog with an HCM phenotype. Endomyocardial biopsies were safely performed and ruled-out storage, infiltrative and mitochondrial diseases that can mimic HCM in people. Primary HCM was diagnosed after exclusion of HCM phenocopies and confirmed by post-mortem cardiac examination.

Conflict of Interest Statement

The authors do not have any conflicts of interest to disclose.

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Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.jvc.2023.10.002.

Video 1	Transthoracic echocardiography from a dog with hypertrophic cardiomyopathy. There was severe left ventricular hypertrophy without left ventricular outflow tract obstruction, moderate left atrial dilation, and subjective right ventricular hypertrophy

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