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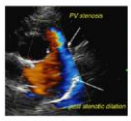
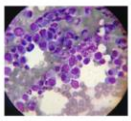
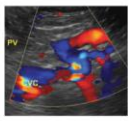

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PATIENT

Brodie Strzadala

SPECIES

Canine

BREED

Boxer

SEX

Male Neutered

AGE

8 years

WEIGHT

79lbs

INTERPRETED BY

Maggie Machen Lamy, DVM, DACVIM (Cardiology)

IMAGING PERFORMED BY

Kim Liedberg

HOSPITAL NAME

SVS Imaging WI

REFERRING VET

Dr. Senn

INVOICE

23607

DATE

4/12/22

PRESENTING CLINICAL SIGNS

History: Presented on 4/8/22 for collapsing episodes with prolonged recovery. History of collapsing episodes dating back to 5/27/17. He has shown some signs of spinal abnormalities at that time, so he was treated for IVDD.
-Abnormal PE/Chem/CBC/UA Results (4/9/2): PLT 151 HCT 56.6% Creatinine 2.0.

ELECTROCARDIOGRAPHIC FINDINGS *Note: Single lead ECGs are evaluated as a rhythm strip. Morphology/MEA cannot be definitively commented on.

A single lead ECG is available; 25mm/s, 10mm/mV. The underlying rhythm is sinus in origin with an average heart rate of 130bpm. P for every QRS complex and vice versa. The P and QRS morphologies are positive. Isolated VPCs throughout; 10 in a 30 second tracing. Singles only and monomorphic. No couplets, triplets or runs of VT. No supraventricular premature beats, pauses or other dysrhythmias observed.
ECG diagnosis: Normal sinus rhythm with isolated VPCs.

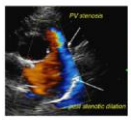
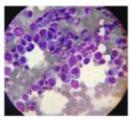
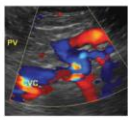
ECHOCARDIOGRAM FINDINGS

2D, m-mode, color flow and doppler imaging is available. The mitral valve appears normal in form and function, with no obvious prolapse into the left atrial lumen. Trace central MR. Mild left atrial dilation. Borderline LV diameter in both systole and diastole with mildly depressed myocardial function. Normal LV wall thickness. The tricuspid valve appears normal in form and function. No overt evidence of pulmonary arterial hypertension or right heart compensation, however right heart is prominent. No tricuspid regurgitation. The aortic valve appears normal. No subvalvular ridge present, mildly increased velocity. Mild aortic insufficiency. Normal pulmonic valve with no pulmonic insufficiency seen. No pericardial or pleural effusion noted. No obvious cardiac tumors. Irregular rhythm noted throughout the examination.

CARDIAC CHART

CANINE CARDIAC PARAMETERS	MR VMAX (m/s)	TR VMAX (m/s)	LA/AO (Boon method)	LA/AO (Heart Base; Swe)	FS (%)	EF (%)	EPSS (cm)
NORMAL PARAMETER	4.5-5.5	<2.7	1.3	<1.6	28-40	40-100	<0.6
PATIENT	5.2	2.0	1.3	1.5	22	40	0.9
CANINE CARDIAC PARAMETERS	**HR** (BPM)	**AV VMAX** (m/s)	**PV MAX** (m/s)	**BODY WEIGHT** (kg)	**LA** 2D short axis Base view (cm)	**LVIDd** Avg; 2D and m-mode short axis (cm)	**LVIDs** Avg; 2D and m-mode short axis (cm)
NORMAL PARAMETER	50-100	0.7-1.7	0.7-1.6	BELOW	BELOW	BELOW	BELOW
PATIENT	70	2.5	1.1	35.8	3.6	5.0	3.9
*Normal chamber parameters expressed as a mean value (SD)				3	1.27 (5.3)	2.46 (2.46)	1.36 (5.5)
BODY WEIGHT DEPENDENT PARAMETERS *Note: All measurements based upon multi-modal images and methods. An average value is reported.*				5	1.40 (4.5)	2.74 (5.2)	1.60 (4.7)
				10	1.50 (3.8)	3.27 (3.5)	2.06 (3.1)
				15	1.83 (2.0)	3.71 (2.4)	2.43 (2.1)
				20	2.02 (1.9)	4.14 (2.2)	2.80 (2.0)
				25	2.18 (2.4)	4.48 (2.9)	3.10 (2.5)
				30	2.33 (3.3)	4.83 (3.9)	3.39 (3.4)
				35	2.48 (4.3)	5.17 (5.0)	3.69 (4.5)
				40	2.62 (5.2)	5.48 (6.1)	3.96 (5.4)

Adapted from June Boon, Veterinary Echocardiography, 1998
Rishniw M and Hollis NE, J Vet Intern Med 2000; 14:429-435
Hansson et al, Vet Rad and Ultrasound 2002
Bonagura et al. Echocardiography: principles of interpretation, Vet Clin North Am 15:1177, 1995

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	50	2.88 (7.1)	6.07 (8.3)	4.46 (7.4)

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Mild structural changes are identified in this study. The LV is borderline dilated with mildly depressed myocardial function and mild LA enlargement. These findings likely reflect early DCM-phenotype given the breed. There is mild right heart prominence in some views, which should be monitored going forward. Mildly elevated aortic outflow velocities are noted with a mild aortic insufficiency, and a baseline blood pressure is recommended. No additional issues are identified.

Frequent ventricular premature contractions were however confirmed as the cause of the noted arrhythmia. VPCs are generated from abnormal conductive or fibrotic tissue in the ventricles of the heart muscle, and even frequent single VPCs will often cause no clinical signs in dogs. When sustained however, ventricular tachycardia can lead to symptoms such as lethargy and collapse.

VPCs are a very non-specific finding. They can be primary in origin (such as ARVC), be secondary to significant cardiac disease (mild in this study) or be extra-cardiac in origin; i.e., due to pain, stress, inflammation, cancer, GI disease, DIC/sepsis, etc. In an 8-year-old Boxer, there is high suspicion for ARVC to explain both of the findings (most common age of onset 6-8yo, often asymptomatic). ARVC can occur with or without systolic dysfunction and structural issues and this should be monitored going forward for any progressive changes. It is always reasonable to rule out other differentials for VPCs (AUS, tick titers, troponin, etc.) however suspicion is low given the signalment of the patient. Unfortunately, there is always an elevated risk for collapse and sudden death in any arrhythmic patient, and even on medications this risk unfortunately still persists. ARVC carries a HIGHLY variable prognosis, with some dogs able to remain asymptomatic for extended periods of time, and others developing exercise intolerance, syncopal episode, and refractory arrhythmias/sudden death imminently.

Based strictly upon the amount of arrhythmia present on the available ECG, it is unclear if anti-arrhythmic therapy is warranted. Collapse episodes are noted in the history; however, it is mentioned that these have been occurring for years. Arrhythmias would be unlikely to cause intermittent collapse for that period of time and other possibilities should be considered. Boxers are also predisposed to vaso-vagal events (not to mention spinal disease) and there may be some combination of issues going on here. **Ideally, a holter monitor is recommended to screen for malignant arrythmias**. If declined or not possible, consider Sotalol as the conservative approach. Discussion with the owner is advised.

No obvious indication for Pimobendan prior to significant LA dilation. Consider ancillary contributing issues such as a non-traditional diet or hypothyroidism. Fish oil supplementation is recommended for dogs with arrhythmias (1000mg of omega 3 and 6 once to twice daily as tolerated).

Anesthetic risk is considered moderate. Avoid ketamine, telazol, Dexdomitor (or other alpha-2 agonists) and acepromazine. Recommend having lidocaine CRI available for use in the event of worsening ventricular arrhythmias under anesthesia (CRI 50—75mcg/kg/min).

Monitor at home for collapse, exercise intolerance, and/or lethargy. Anesthesia is not recommended until good arrhythmic control is achieved. Lifelong mild to moderate activity restriction is advised.

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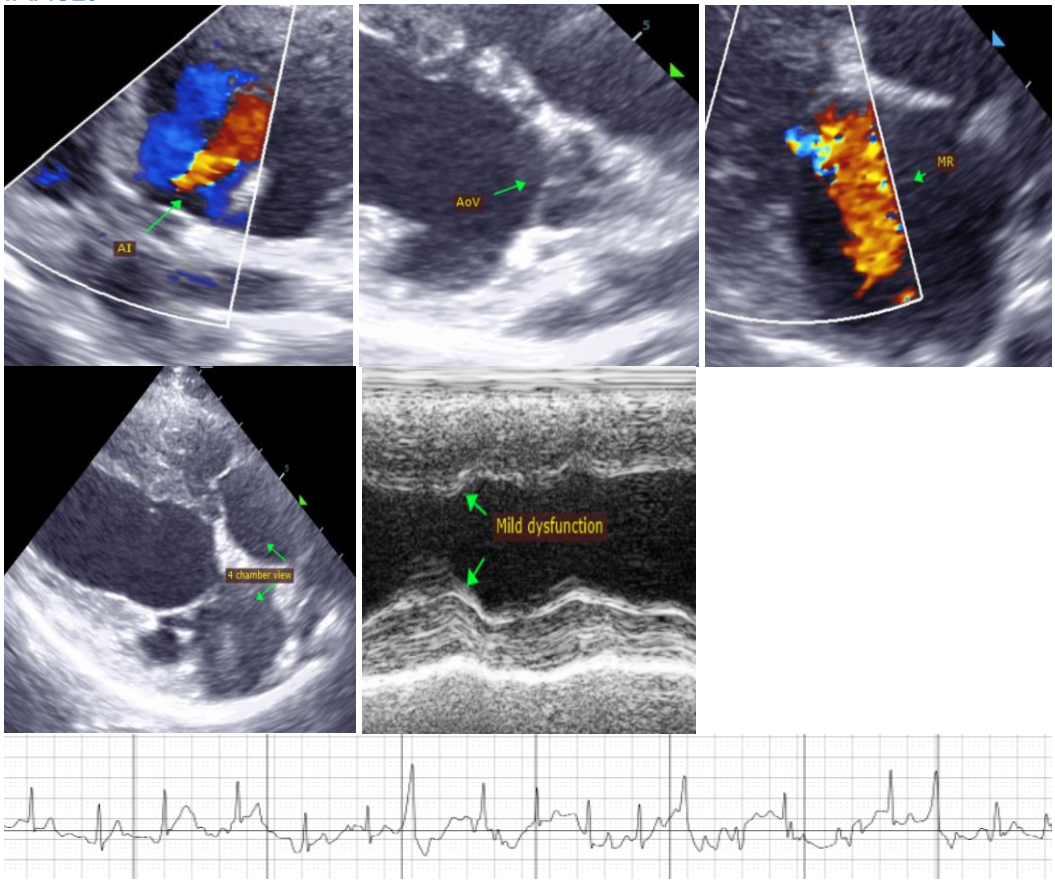
PLAN

A holter monitor is recommended. If declined, institute sotalol 1-2mg/kg PO q12h. Recheck ECG or holter in 1-2 weeks to assess response (goal is significant reduction in ectopy without a significant change in underlying sinus rate). No obvious indication for Pimobendan. Consider diet/thyroid status as discussed. Institute Taurine 1000mg PO q12h.

Recheck ECG and echocardiogram is recommended in 6 months to determine progression/control, sooner if any development of associated clinical signs.

IMAGES



The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance, please contact me.

Maggie Machen Lamy, DVM
Diplomate of the American College of Veterinary Internal Medicine (Cardiology)
info@sonopath.com

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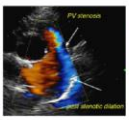
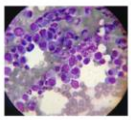
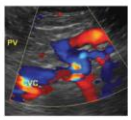

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