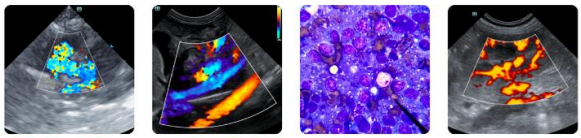

PATIENT

Callie Gala

SPECIES

Feline

BREED

DSH

SEX

Female Spayed

AGE

15.7 years

WEIGHT

7.08lbs

INTERPRETED BY

Maggie Machen Lamy, DVM, DACVIM (Cardiology)

IMAGING PERFORMED BY

Loetitia Saint-Jacques, LVT

HOSPITAL NAME

VCA Feline Animal Hospital

REFERRING VET

Dr. Fleming

INVOICE

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PRESENTING CLINICAL SIGNS

History: Possible cardiomegaly. Lethargy. CKD IRIS Stage 2-3. Hyporexia. Hypermagnesemia – mild. Weight loss - progressive. Sedated for exam.
-Abnormal PE/Chem/CBC/UA Results (12/30/2025): CBC: HCT 38, platelets 156 (d/t clumping), mild lymphopenia 8166 (ref 1200-8000) CHEM: BUN 53, creatinine 1.9, SDMA 25.3, ALT 46, ALP 28, precisionPSL 14, hypermagnesemia 3.1 (ref 1.5-2.5) T4: 2.7 UA: USG 1.019, protein 2+, occult blood 3+, RBC 11-20, amorphous phosphate crystals 2-3.

ELECTROCARDIOGRAPHIC FINDINGS

A six lead ECG is available at 25mm/s; 10mm/mV. The underlying rhythm is suspected to be sinus in origin; however, P waves are extremely difficult to confidently identify. The average heart rate is 220bpm with a largely regular rhythm. The QRS is inverted. The MEA is shifted left. VPCs are noted; singles only. No APCs, pauses or other dysrhythmias observed.
ECG diagnosis: Suspect normal sinus tachycardia with isolated VPCs; however, P waves cannot be visualized. LAFb.

ECHOCARDIOGRAM FINDINGS

2D, m-mode, color flow and doppler imaging is available. The left ventricular wall is moderately increased in dimension. The LV chamber is decreased in dimension. There is a diffusely hyperechoic endocardium consistent with fibrosis. Mild symmetric papillary muscle hypertrophy and remodeling. The right ventricle is subjectively normal in size and morphology. There is no left atrial enlargement present. No right atrial enlargement present. Normal RVOT velocity. Trace TR. Normal LVOT velocity. There is no obvious systolic anterior motion (SAM) of the mitral valve present. No MR. No significant AI or PI. There is no pericardial effusion noted. No pleural effusion appreciated. No obvious cardiac tumors.

CARDIAC CHART

FELINE CARDIAC PARAMETERS	BODY WEIGHT (kg)	HR (BPM)	IVSd (cm) (Moise, Pipers)	LVIDd (cm) (Moise, Pipers)	LVWd (cm) (Moise, Pipers)	FS (%)	EF (%)
NORMAL PARAMETER	------	150-240	0.35-0.55	<2 (mean 1.5)	3.5-0.55	35-67	80-100
PATIENT	3.2	NM	0.73	0.9	0.74	47	90
FELINE CARDIAC PARAMETERS	**LA/AO** (Boon)	**LA/AO HEART BASE (Swe)** (Abbott)	**LA** 2D short axis Base view (cm) (Abbott)		**LVOT VEL** (m/s)	**RVOT VEL** (m/s)	**E max** (m/s)
NORMAL	<1.5	<1.3	<1.2		<1.6	<1.3	<0.9
PATIENT	NM	1.1	1.1		1.4	1.0	NM

**Note: All measurements based upon multi-modal images and methods. An average value is reported.*
Adapted from June Boon, Veterinary Echocardiography,1998
Abbott J & MacLean H JVIM 2006;20: 111-119, Moise et al. Am J Vet Res 47:1476, 1986. Pipers et al. Am J Vet Res 40:882, 1979.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Hypertrophic cardiomyopathy (HCM) is a rule out diagnosis once a patient is deemed normotensive and euthyroid. Both should be considered in this case as contributing factors. The LV chamber is decreased, and pseudohypertrophy should also be considered, particularly given CKD (i.e. volume depletion leading to a transiently hypertrophied appearance). Regardless, the

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degree of disease is mild, with moderate LVH and no LA dilation. This would indicate the risk for clinical issues is low at this time.

The ECG does confirm an arrhythmia, with isolated ventricular premature contractions (VPCs) noted. 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. When sustained however, ventricular tachycardia can lead to symptoms such as lethargy and collapse. **It is worth noting that P waves are difficult to visualize; however, a sinus origin is suspected in spite of this. Atrial fibrillation is ruled out as the rhythm is regular; however, an ancillary supraventricular rhythm remains a possibility.** Consider repeating the ECG tracing with light sedation, a vagal maneuver, etc.

VPCs are a very non-specific finding. They can be primary in origin (a rule out dx), 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 this cat with systemic illness, this is suspected to be the likely cause. Further systemic workup is advised. The changes to the LV may of course also be contributing; however, this is speculative.

When assessing VPCs, we must not only consider why they are happening, but if we should treat them. Markers of malignancy are assessed, in addition to patient history and signalment. Based upon what is seen here in addition to the highly sensitive nature of cats to anti-arrhythmic medications, I would not institute therapy at this time. Assuming the patient remains asymptomatic, reassessing the finding periodically through auscultation/ECG monitoring is a reasonable approach.

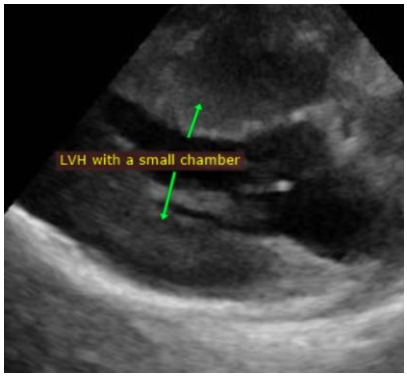
Anesthesia is not advised due to the complexity of the findings.

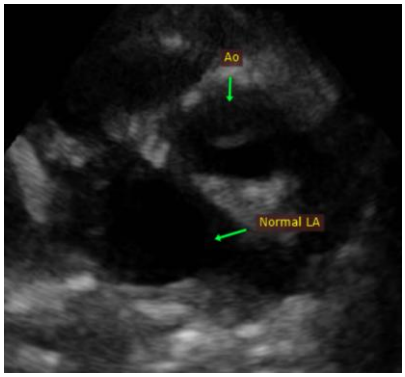
PLAN

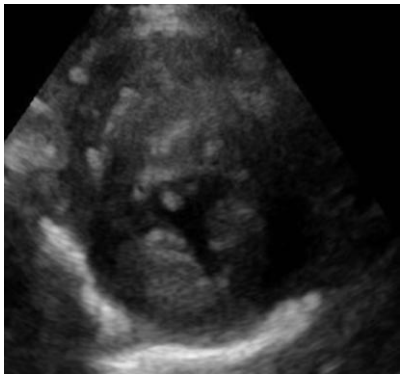
Monitor BP and T4 every 6 months. Full systemic evaluation and baseline senior lab work as discussed. Reassess BP as discussed.

A recheck echocardiogram and ECG are recommended in 6 months to monitor for progression, sooner if any issues arise in the interim.

IMAGES







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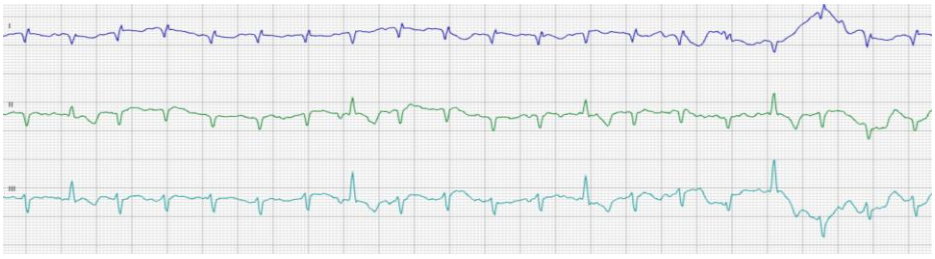

The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

Thank you for this referral. This report was generated using transcription software, and minor dictation errors may be present. 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