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Trading name of SK Veterinary Services Ltd

Patient Data

Owner name	?????	**Animal name**	DOLLY
Identification	??????????	**Exam Date**	??/???/2023
Exam Description	??????????		
Performing Physician	S KEIR	**Report Date**	??/???/2023

Cardio Feline

M-Mode

Left Ventricle

IVSd	7.2	mm	LVIDd	11.4	mm
LVPWd	8.0	mm	IVSs	10.4	mm
LVIDs	3.8	mm	LVPWs	9.5	mm
EF	95	%	%LV FS	67	%
% IVS	44	%	%PW	19	%
LV Mass	15	g	Relative Wall Thickness	1.41	

Doppler

MV

MV E Vel [0.58, 0.66]	0.62	m/s	MV A Vel [0.47, 0.48]	0.48	m/s
MV E PG	1.5	mmHg	MV A PG	0.9	mmHg
MV PHT [22, 14]	18	ms	MVA (PHT)	12.11	cm²
MV E/A	1.29		MV Dec Time	62	ms
IVRT [76, 60]	68	ms	E/IVRT	0.91	

Pulmonary Vein

PVein S Vel	-1.00	m/s	PVein D Vel	-1.00	m/s
PVein S/D Ratio	1.00				

Pulmonary Capillary Wedge Pre sure m/s

MV E Vel [0.58, 0.66]	0.62

B-Mode

Aorta/LA

Ao Diam [12.8, 11.3]	12.0	mm	LA Diam [12.1, 13.0]	12.6	mm
LA/Ao	1.05				

Observations

Cardio remarks

Dolly was presented for echocardiographic examination and ECG assessment due to the finding of a gallop rhythm and raised pro-BNP on blood sampling.

Dolly was presented after being administered gabapentin but she was still extremely reluctant to stay still even with the temptation of Lik-e-Lix. After a brief initial assessment which ascertained the presence of HCM with normal sized LA and no free fluid, it was decided to sedate Dolly to allow a thorough echo exam. Dolly was sedated with Alflaxan IM and then topped up IV to keep her sedated. She recovered quickly and these medications will have minimal effects on the measurements obtained and the overall diagnosis and prognosis.

On examination, Dolly's heart rate was about 160bpm with no rate changes and a clear gallop rhythm audible. A concious ECG was performed at this time.

Subjective examination. There was no pleural or percardial fluid present. Obvious thickening of the LV wall was visible with good systolic function but no end-systolic LV cavity obliteration. There were no obvious valve abnormalities and the LA diameter major was 16mm so normal. There was noted the prescence of LVOTO obstruction and mitral valve regurgitation on colour doppler. There were no false tendons visible and the papillary muscles were not hypertrophied. The right side of the heart was in proportion and did not appear enlarged or thickened. There was no evidence of spontaneous echo-contrast or thrombus in the LA.

LV wall thickness. The LV free/posterior wall measured from 8.4-9mm and the interventricular septum from 5mm up to 6.5mm in end-diastole. Measurements over 6mm confirm hypertrophy.
LA size. The Left atrium was within normal size with LA:Ao of about 1 to 1.2 (>1.6 is dilation) and normal LAD. No visible spontaneous echocontrast or thrombus in the LA.
Presence of Left ventricular outflow tract obstruction. LVOTO was documented with turbulent flow in the aorta; this was due to documented systolic anterior movement (SAM) of the anterior leaflet of the mitral valve. This movement of the mitral valve also created a small mitral regurgitation that was visible in the R parasteral 4/5 chamber view and the left apical 4 chmaber view. The LVOTO is not severe enough to require medication.
Diastolic function. Normal LA size suggests normal diastolic function. IVRT, E wave and A wave, E:A ratio and E/IVRT are all normal evidencing normal diastolic function (i.e. relaxation) at this time. Systolic function normal.

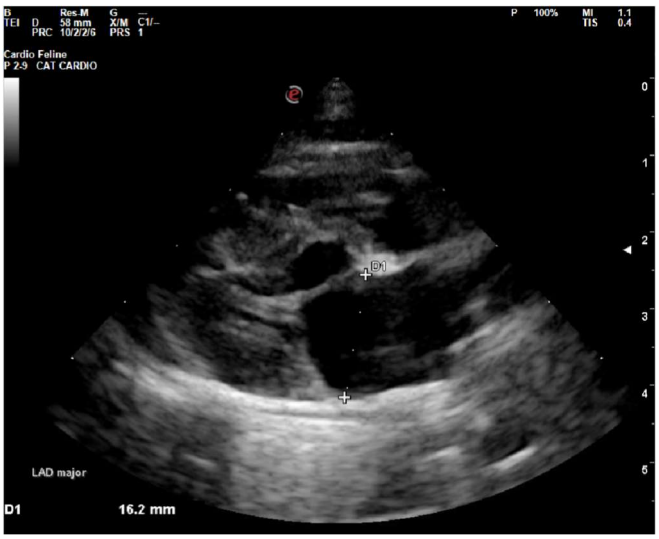
Conclusions

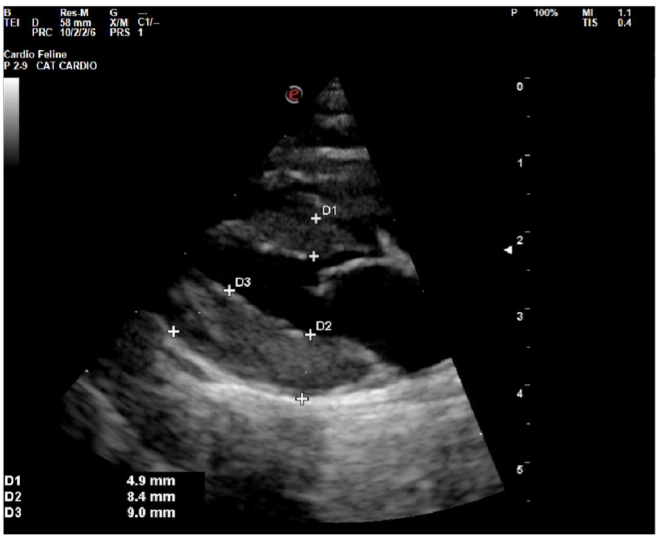
Dolly has hypertrophic cardiomyopathy (HCM) with LVOTO due to SAM, which is also called hypertrophic obstructive cardiomyopathy. Although some of the measurements of the LV free/posterior wall are getting towards the extreme end (classed as over 9mm), all other measurements of function are good and Dolly doesn't currently have any findings that would indicate a poor prognosis. Dolly is currently in stage B1, without LA enlargement, and so no medication is indicated at this time.

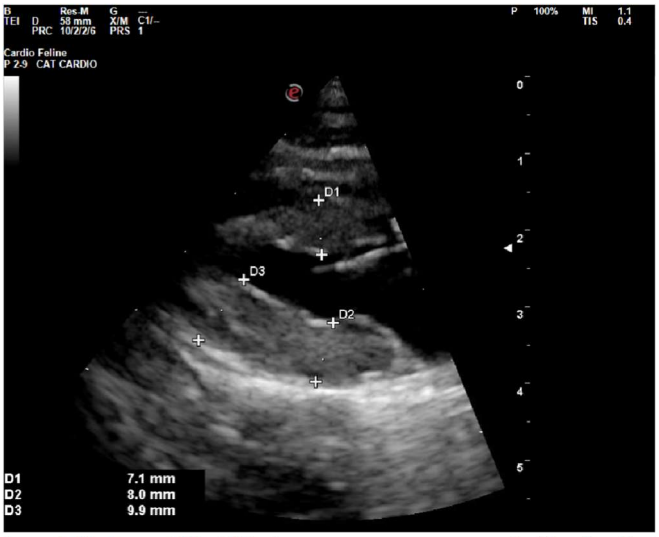
Idiopathic HCM is a diagnosis of exclusion, with the need to rule out other possible cardiac or systemic diseases which may be causing or contributing to a HCM phenotype such as systemic hypertension, hyperthryoidism, anaemia, acromegaly and aortic stenosis, the later ruled out by theis echo. This work up should include a blood presure and T4 measurement if these have not already been performed. Transient myocardial thickening appears identical to idiopathic HCM on echo but the changes disappear within 3-6 months; cats with this tend to present as acute congestive heart failure.

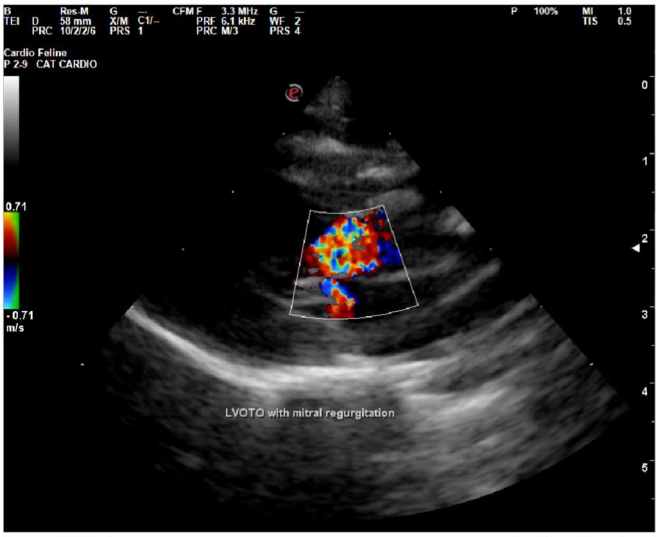
The disease progression with HCM is highly variable and often has a prolonged symptomatic, pre-clinical phase. Fortunately, while HCM is a very common disease, the overall 5 year cardiac mortality is 25%. Repeated heart scans over time are needed to monitor for changes associated with increased risk factors and preemptive medication or increased monitoring for congestive heart failure can be instigated. I recommend a repeat heart scan in 6-12 months. The changes over time will also give a better idea of prognosis.

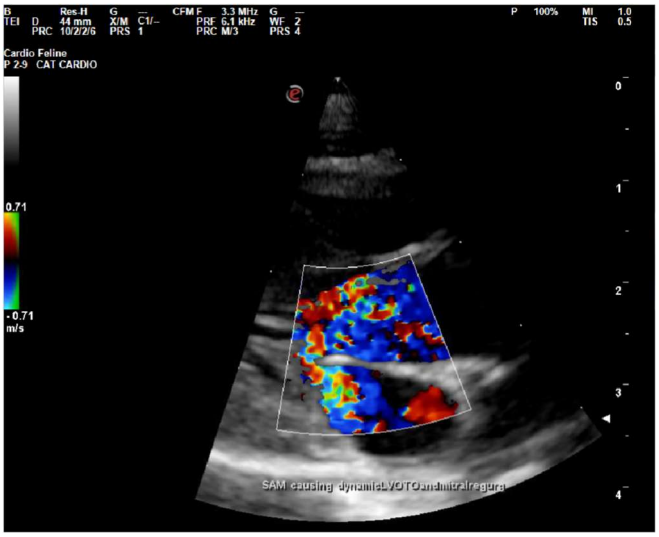
	The arrhythmia is currently mild and will be asymptomatic. ECG abnormalities are commonly seen in all forms of cardiomyopathy. The prescence of ectopy does not increase the risk of sudden death. The threshold for treating arrhythmias in cats is higher than dogs due to the reduced feasibility of medicating the patient, limited treatment options due to toxicity and limited ability to measure any daily arrhtyhmia burden and hence the response to treatment. This arrhythmia will be asymptomatic currently and so treatment is not recommended.
SIGNATURE	Dr Sarah Keir BVMS PGCertSAM MRCVS RCVS Advanced Practitioner in Small Animal Medicine
New field	ECG - obtained while concious with gabapentin, 1 minute trace as wandering around, kept walking around. HR averaging 160bpm, sinus rhythm with a couple of VPCs seen (around 2 in the One minute trace).

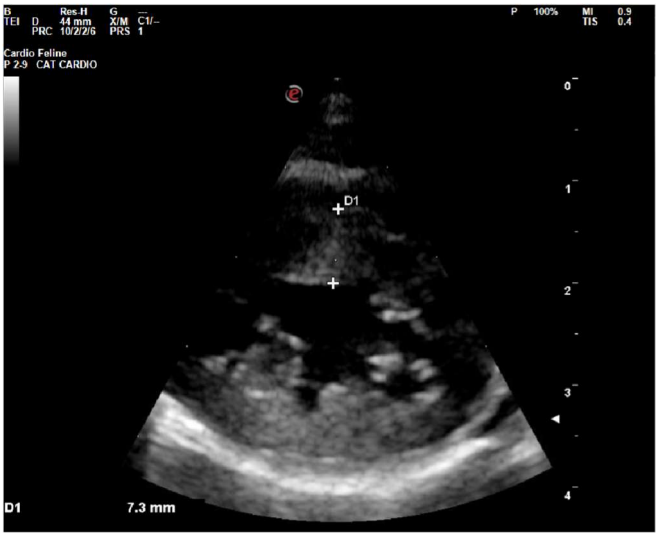

Cardio Feline
P 2-9 CAT CARDIO
LAD major
D1 16.2 mm

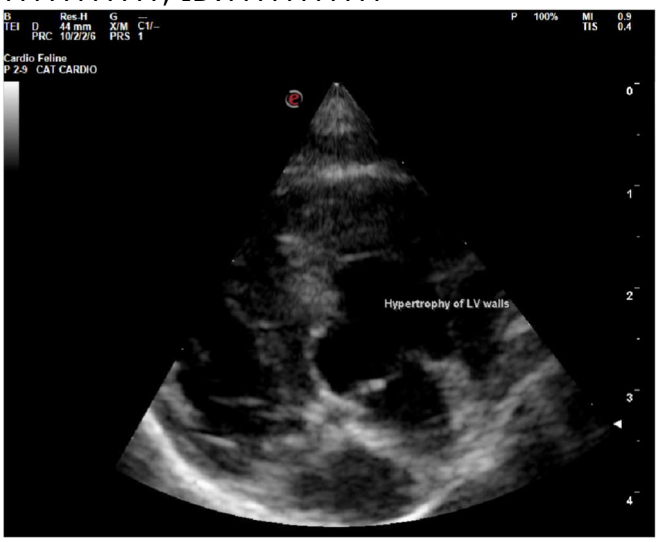

Cardio Feline
P 2-9 CAT CARDIO
D1
D3
D2
D1 4.9 mm
D2 8.4 mm
D3 9.0 mm

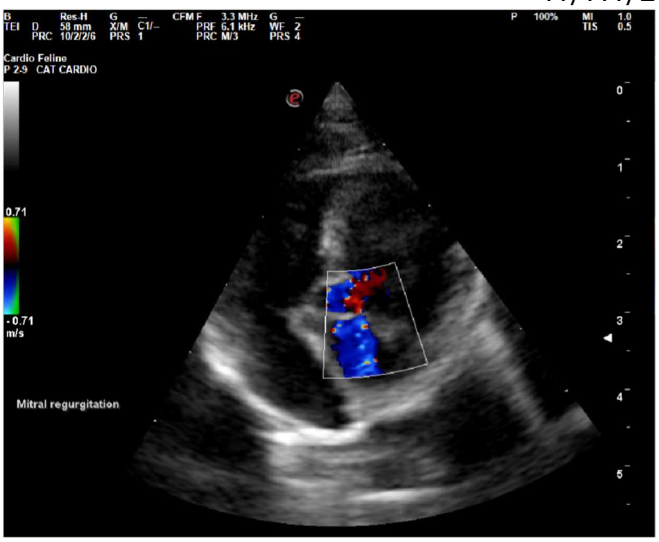

Cardio Feline
P 2-9 CAT CARDIO
D1
D3
D2
D1 7.1 mm
D2 8.0 mm
D3 9.9 mm

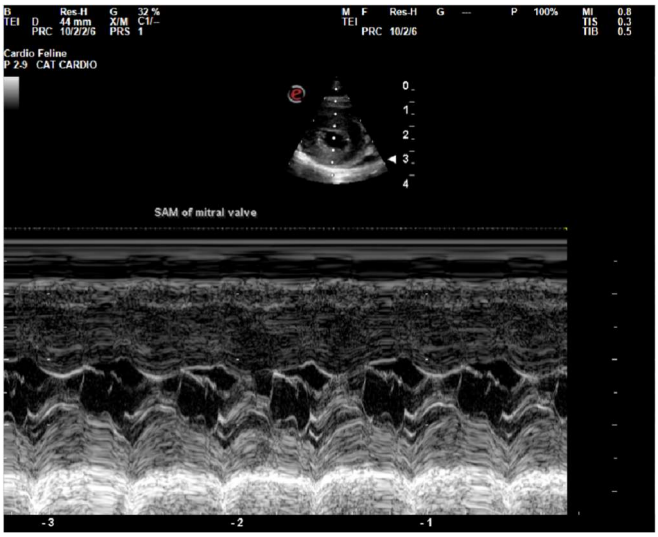

Cardio Feline
P 2-9 CAT CARDIO
0.71
- 0.71
m/s
LVOTO with mitral regurgitation


Cardio Feline
P 2-9 CAT CARDIO
0.71
- 0.71
m/s
SAM causing dynamic LVOTO and mitral regurg


Cardio Feline
P 2-9 CAT CARDIO
D1
D1 7.3 mm


Cardio Feline
P 2-9 CAT CARDIO
Hypertrophy of LV walls


Cardio Feline
P 2-9 CAT CARDIO
0.71
- 0.71
m/s
Mitral regurgitation


Cardio Feline
P 2-9 CAT CARDIO
SAM of mitral valve