



PATIENT PRESENTING CLINICAL SIGNS

Lilu Ruggieri History: Respiratory distress, abnormal breathing, labored breathing with increased abdominal effort for 2 days Tachypnea/tachycardia for 2 days. Anorexia for 2 days, drinking normal. Lethargy summary of PE findings (MM pm <2s Increased lung sounds Crackles bilaterally Tachycardia and wheezing). No murmur.

SPECIES

Canine -Current medications: Furosemide 4mg/kg IV, Butorphanol 0.1mg/kg IM.
-Abnormal PE/Chem/CBC/UA Results: CBC: WBC 29.76 HIGH BAND * Suspected * PCT 0.59 Chem/lytes: SDMA 17, Phosphorus 2.76, Chloride 106.

BREED

Shih Tzu -Radiographs: The cardiac silhouette appears generally enlarged on the ventral dorsal view and also appears wide and tall on the lateral images. A diffuse patchy unstructured interstitial pattern is present throughout the pulmonary parenchyma with alveolar pattern noted in the caudal and dorsal thorax that he also has a patchy appearance. There are a few rounded more nodular appearing soft tissue opacities in the cranial and ventral thorax on the lateral images. The diaphragm is intact. The trachea is uniform in diameter with no evidence of collapse. Thin pleural fissure lines are noted.

SEX

Female Spayed

AGE

14 years **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;505mm/s, 20mm/mV. The average heart rate is 180bpm with a largely regular rhythm. The rhythm is sinus in origin, with a p for every QRS complex and vice versa. The P and QRS morphologies are positive. No ectopic beats, pauses or other dysrhythmias observed.

WEIGHT

13.7lbs ECG diagnosis: Normal sinus tachycardia.

ECHOCARDIOGRAM FINDINGS

2D, m-mode, color flow and doppler imaging is available. Normal mitral valve with no obvious prolapse into the left atrial lumen. No mitral regurgitation with a small left atrial dimension. Small LV diameter with adequate myocardial function. The tricuspid valve appears mildly thickened with mild to moderate tricuspid regurgitation. Moderate right atrial enlargement; severe right ventricular dilation and hypertrophy consistent with pulmonary arterial hypertension. Septal flattening in systole. The pulmonic and aortic valves are normal in morphology and mobility. Moderate main PA and branch dilation. No pulmonic insufficiency. Normal pulmonic and aortic outflow velocities. No pericardial and small volume pleural effusion. No cardiac tumors observed.

INTERPRETED BY

Maggie Machen Lamy,
DVM DACVIM
(Cardiology)

IMAGING PERFORMED BY

Crystal Hill, RVT

CARDIAC CHART

HOSPITAL NAME

Beatties Pet Hospital
Burlington

REFERRING VET

Dr. Ruggieri

INVOICE

27757

DATE

12/1/22

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		NM	1.1	1.1	49	84	0.3
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	150	1.2	0.7	6.2	1.3	1.3	0.7



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*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	5	1.40 (4.5)	2.74 (5.2)	1.60 (4.7)
<i>*Note: All measurements based upon multi-modal images and methods. An average value is reported.</i>	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)
Adapted from June Boon, Veterinary Echocardiography, 1998	25	2.18 (2.4)	4.48 (2.9)	3.10 (2.5)
Rishniw M and Hollis NE, J Vet Intern Med 2000; 14:429-435	30	2.33 (3.3)	4.83 (3.9)	3.39 (3.4)
Hansson et al, Vet Rad and Ultrasound 2002	35	2.48 (4.3)	5.17 (5.0)	3.69 (4.5)
Bonagura et al. Echocardiography: principles of interpretation, Vet Clin North Am 15:1177, 1995	40	2.62 (5.2)	5.48 (6.1)	3.96 (5.4)
	50	2.88 (7.1)	6.07 (8.3)	4.46 (7.4)

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Severe pulmonary hypertension (PAH) is suspected, as evidenced by significant right heart/MPA enlargement. The estimated systolic pulmonary arterial pressure is >80mmHg, with normal being <25mmHg. This is causing hypertrophy and dilation of the right heart and MPA (indicating right-heart pressure overload). The left heart dimensions are normal to small. No tumors or effusions are appreciated. The ECG is unremarkable with a sinus tachycardia.

Clinical signs of weakness, heavy breathing, cyanosis, and syncope are attributed to PAH. The underlying genesis of PAH is poorly understood in cases other than heartworm infestation, though it occurs with increased frequency in a variety of forms of chronic lung disease and in patients with idiopathic pulmonary fibrosis. If not performed, a heartworm antigen test is recommended. Given the chronicity of the disease seen here (no chronic case history provided), COPD/chronic bronchitis and/or primary PF as an underlying cause with an acute secondary exacerbating insult (infectious or inflammatory) is suspected. Patients with this degree of PAH and pulmonary disease can develop right-sided congestive heart failure (ascites/pleural effusion), debilitating cyanosis, labored breathing and exertional syncope if poorly controlled.

Given the recent history of respiratory signs and crackles, the most common cause is an infectious or inflammatory insult causing a decline in already poor oxygenation status. A PTE cannot be ruled out. Coverage with broad spectrum pulmonary antibiotic (fluoroquinolone) is recommended, in addition to aggressive vasodilation using pimobendan and sildenafil. I would not utilize a diuretic, as this patient has no risk for left-sided CHF making the radiographs likely more consistent with airway disease. There may be risk for right-sided CHF in the future; however, no effusions are noted making this unlikely. If the patient experiences any additional respiratory compromise, continued hospitalization for oxygen support and IV antibiotics may be necessary. Finally, the patient is volume contracted, likely due to Lasix therapy and cautious fluid administration may be beneficial.

Once stable, use of theophylline and/or taper course of anti-inflammatory steroids can also be beneficial in these cases, to treat exertional dyspnea or acute flare ups and decrease the inflammatory component as much as possible. PRN use of cough suppressants may also be beneficial. Unfortunately, the prognosis overall is poor, however I am hopeful we can provide some medical relief going forward.

Omega fatty acid supplementation (anti-inflammatory) may be of some long-term benefit. Monitor for worsening of labored breathing, exercise intolerance or collapse episodes.



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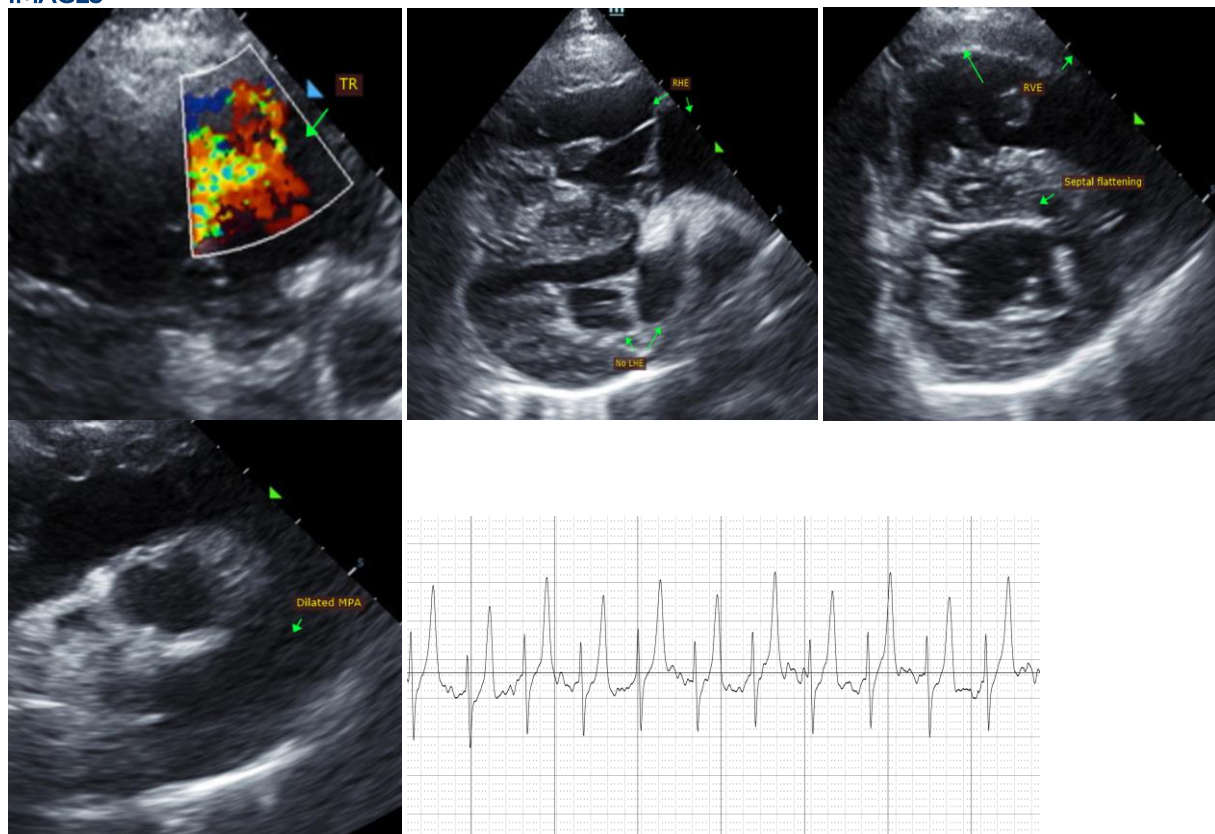
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PLAN

Continue hospitalization for oxygen support. Discontinue Lasix as discussed. Consider fluids. Institute Pimobendan 0.3mg/kg PO q12h. Institute sildenafil (Viagra) 1-2mg/kg PO q8h. Consider course of Baytril or similar. Can also use hydrocodone and/or theophylline depending on chronic clinical signs of cough/exertional dyspnea.

Recommend recheck echocardiogram in 6 months to reassess pulmonary pressures, sooner if any development of 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. 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.

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