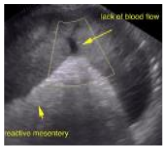
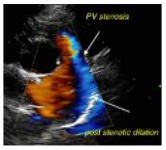
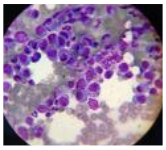
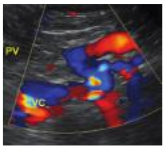
SonoPath
Clinical Sonography & Telecytology
EDUCATIONAL TELECONSULTATION SERVICES™

SonoPath.com
1-800-838-4268 info@sonopath.com

PATIENT
Dante Wilson

SPECIES
Canine

BREED
English Bulldog

SEX
Male Neutered

AGE
9 years

WEIGHT
27.3

INTERPRETED BY
Maggie Machen Lamy, DVM, DACVIM (Cardiology)

IMAGING PERFORMED BY
Kelly Romero, DVM

HOSPITAL NAME
FC Veterinary Emergency Hospital

REFERRING VET
Dr. Romero

INVOICE
23947

DATE
4/29/22

PRESENTING CLINICAL SIGNS

History: Regurgitated over weekend. Became lethargic, but still eating. Started coughing (none previous). Went to rDVM and had radiographs taken on Tuesday. Presented to the ER on 4/27. Initial radiographs interpreted as moderate interstitial to alveolar lung pattern in right middle lung lobe and mild generalized diffuse interstitial pattern overall. Heart and vasculature read out as normal size. History of alopecia and splenectomy secondary to splenic torion. Patient is not on a grain free diet. Abnormal PE/Chem/CBC/UA Results: Normal temperature. No murmur. Blood pressure 110 systolic. Increase respiratory rate and effort with harsh lung sounds and more difficult to hear heart on the right. CBC - WNL. Chemistry - ALP 419. EPOC/blood gas - lactate 4.2 Initial treatment was focused on aspiration pneumonia with antibiotics and IVF. After echo, have continued Unasyn, discontinue IV fluids and Baytril, added pimobendan 7.5mg BID and currently giving furosemide 3mg/kg IV.

ECHOCARDIOGRAM FINDINGS

2D, m-mode, color flow and doppler imaging is available. The left ventricular is significantly dilated with diminished systolic function. Severe left atrial enlargement. The mitral valve appears normal in form and function, with no obvious prolapse into the left atrial lumen. Mild central mitral regurgitation secondary to annular stretch. Decreased velocity. Decreased LV wall thickness and increased sphericity. The tricuspid valve appears normal in form and function. Mild right atrial and ventricular dilation. Trace tricuspid regurgitation. Normal velocity. The aortic valve is normal in morphology and mobility. No subvalvular ridge present. Decreased LVOT and RVOT velocities. No aortic insufficiency. Normal pulmonic valve with no pulmonic insufficiency seen. No pericardial effusion. Small pockets of pleural effusion noted. No obvious cardiac tumors.

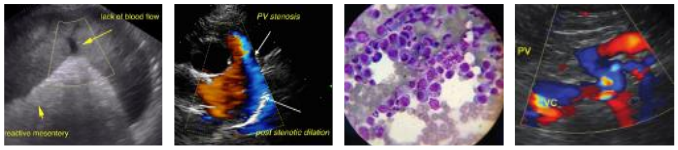
CARDIAC CHART

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	4.3	2.2	2.3	2.5	9	21	1.1
CANINE CARDIAC PARAMETERS	**HR** (BPM)	**AV VMAX** (m/s)	**PV MAX** (m/s)	**BODY WEIGHT**	**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	NM	0.7	0.5	27.3	4.2	5.2	4.7
*Normal chamber parameters expressed as a mean value (SD)				3	1.27 (5.3)	2.46 (2.46)	1.36 (5.5)
				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)
				50	2.88 (7.1)	6.07 (8.3)	4.46 (7.4)

BODY WEIGHT DEPENDENT PARAMETERS

**Note: All measurements based upon multi-modal images and methods. An average value is reported.*

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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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Unfortunately, this patient has significant cardiomyopathy and systolic dysfunction. This is causing dilation and overload of all four chambers resulting in insufficiency of the AV valves. The degree of dilation and pump failure is suspected to be causing congestive heart failure (pleural effusion). The right heart is also affected, with mild dilation; however, no obvious pulmonary hypertension is observed.

Systolic failure can be primary in nature (DCM) or secondary to taurine deficiency, myocarditis, tachycardia-induced cardiomyopathy, or infiltrative disease such as lymphoma. In a predisposed breed such as a Bulldog, this is considered genetic primary DCM until proven otherwise. A diet history should be sought; however, a grain free option is not mentioned in the history. A thyroid level should be assessed if not recently performed. Regardless, prognosis is poor to grave at this stage in the disease process, with an average survival time of <6 months. Most DCM patients will succumb to either refractory CHF or sudden arrhythmic death at any time, and this risk should be relayed regardless of therapy.

Initiation of full cardiac supportive medications is recommended as below. It is somewhat surprising to see pleural effusion rather than pulmonary edema, given the appearance of the left heart. A possible explanation may be aspiration pneumonia was the primary issue with subclinical cardiomyopathy present, and fluid overload led to development of pleural effusion. Because of this possibility, consider continuing broad spectrum antibiotic therapy. If the patient is refractory to therapy, reassessing CXR is strongly recommended for comparison. Sampling and submission of the effusion for cytology may also be beneficial in this instance. Cases of systolic failure are at high risk for malignant tachyarrhythmias (such as VT) and sudden death. A baseline ECG is recommended.

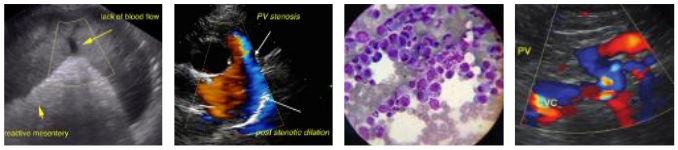
Omega fatty acid supplementation and mild salt restriction may be of some long-term benefit. Monitor for development of a cough, worsening labored breathing, exercise intolerance or collapse episodes in the future. Monitoring of sleeping breathing rates at home is recommended to assess response to medications and recurrence of CHF in the future.

Elective anesthesia is not advised, as there is high risk for complication. Risk:benefit ratio should be considered. Consider consultation with and/or referral to a facility with an anesthesiologist. Should you elect to proceed, cardiac protective drug choices (opioid/benzodiazepine premedication, propofol or alfaxalone induction, iso or sevoflurane gas) are recommended. Pre-oxygenate for 5-10 minutes prior to induction and recover in O2 cage. Monitor for arrhythmias, hypotension, and hypoxia both intra and post-operatively and intervene as necessary. Moderate IV fluid restriction is recommended to avoid fluid overload, while considering comorbidities, hydration status, BP, etc. Avoid heart rate stimulating drugs such as atropine unless clinically indicated.

PLAN

Baseline ECG and BP as discussed. Recommend the following oral medications: Furosemide 1-2mg/kg PO q12h. Pimobendan 0.25-0.3mg/kg PO q12h. Spironolactone 1-2mg/kg PO q12h. Recommend taurine 1000mg PO q12h. Recommend continued Baytril therapy.

Recheck renal panel, BP and clinical response in 5-7 days, sooner if any decline. If the BP is >130mmHg, institute ACE-I 0.5mg/kg PO q12h. Monitor every 3-4 months lifelong. If effusion is



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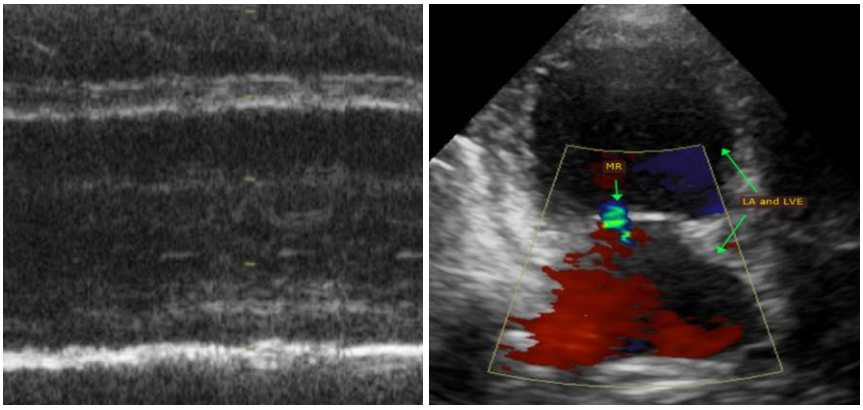
refractory to therapy and/or the patient quality of life is not improving, other possible contributing issues should be considered.

Recheck echocardiogram in 6 months to screen for progression, sooner if clinical signs arise in the interim.

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.

Maggie Machen Lamy, DVM
Diplomate of the American College of Veterinary Internal Medicine (Cardiology)
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