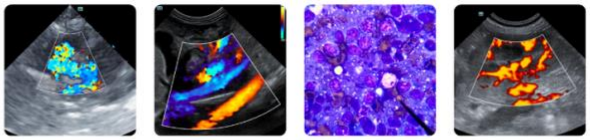

PATIENT

Karma Ferris

SPECIES

Canine

BREED

Pitbull

SEX

Spayed Female

AGE

9

WEIGHT

60

INTERPRETED BY

Eric Lindquist, DMV, DABVP (Canine & Feline), Cert. IVUSS

IMAGING PERFORMED BY

Dr. John Sampson

HOSPITAL NAME

Richboro VH

REFERRING VET

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INVOICE

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4/23/26

PRESENTING CLINICAL SIGNS

History: Presented on 4/20 for checkup after progressive respiratory issues (coughing, panting) and then a syncopal episode on 4/17. Has been on grain free diet for years - high suspicion for DCM

Abnormal PE/Chem/CBC/UA Results: Heart sounds were quiet and muffled but no arrhythmia noted Rest of PE was normal Radiographs showed enlarged heart - rads attached Started Vetmedin 10 mg po bid and Furosemide 60 mg bid at that visit. Cardalis 80/10 came in today and she is starting that today.

ULTRASONOGRAPHIC EXAMINATION OF THE HEART

CANINE CARDIAC PARAMETERS	**MR VMAX** (m/s)	**TR VMAX** (m/s)	**LA/AO** (M-Mode)	**LA/AO** (Heart Base; Swe)	**FS** (%)	**EF** (%)	**EPSS** (cm)
NORMAL PARAMETER	4.5-5.5	<2.7	1.3	Up to 1.6	28-40	40-100	<0.6
PATIENT	At least 5.0	At least 2.0	1.5	2.2	10	20	2.2
CANINE CARDIAC PARAMETERS	**HR** (BPM)	**AV VMAX** (m/s)	**PV MAX** (m/s)	**BODY WEIGHT**	**LAD** LA MAX 4 Chamber	**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				
PATIENT	--	--	.70	60	4.6	6.1	--

Cardiac Presentation

The echocardiogram for this patient presented excessive **left atrial size** expressed in 3 different LA measurement methods. Left atrial content was anechoic. No evidence of "smoke" or thrombotic activity was noted. The atrial septum was deviated owing to volume overload. The cranial and caudal **mitral** valve leaflets presented normal linear structure, yet insufficiency was noted. Mitral insufficiency was centralized and moderate. The **left ventricle** demonstrated excessive volume (LVIDd measurement above). Ventricular function was subnormal expressed by the fractional shortening measurement listed below. Myocardium appeared subjectively thin typical of DCM. Contractility was extremely poor in this patient. The **left ventricular outflow** tract demonstrated normal laminar flow and subjective structural integrity. The **right atrium** and auricle revealed normal size, structure and content. No evidence of masses was noted or chamber overload. **Tricuspid** insufficiency was noted, centralized and moderate. The **right ventricle** was of normal size (1/3 diameter of LV), chordae structure, myocardial echogenicity and thickness. **Pulmonic** tract assessment revealed normal valve

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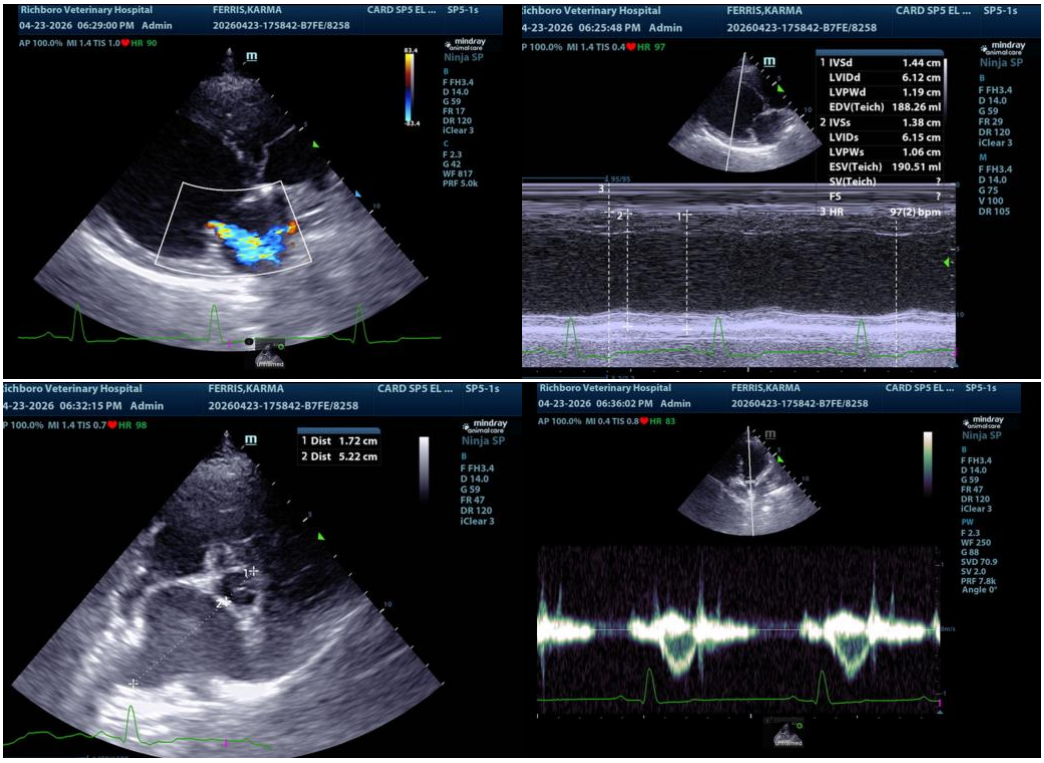
structure, laminar flow, and diameter (approx.1:1 pa/ao ratio). No visible **pericardial** or free pleura fluid was noted. The cranial **mediastinum and pericardial regions** were free of masses in the visible window.

ULTRASONOGRAPHIC FINDINGS

- DCM presentation

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Continuation of Vetmedin, furosemide, ACE inhibitor, and spironolactone all indicated. Assessment for taurine deficiency/nutritional cardiomyopathy and the cause of myocarditis should also be evaluated. Recheck echo is recommended in one month. Prognosis is guarded, depending upon response to therapy.



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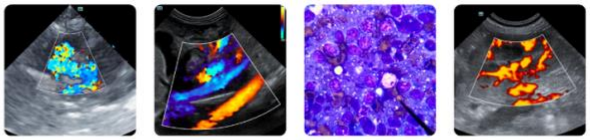

Clinical Sonography & Telecytology
Educational Teleconsultation Services™

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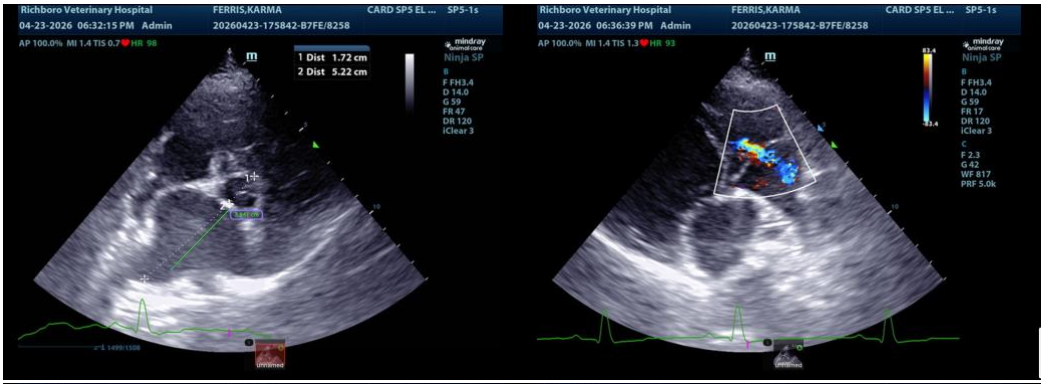

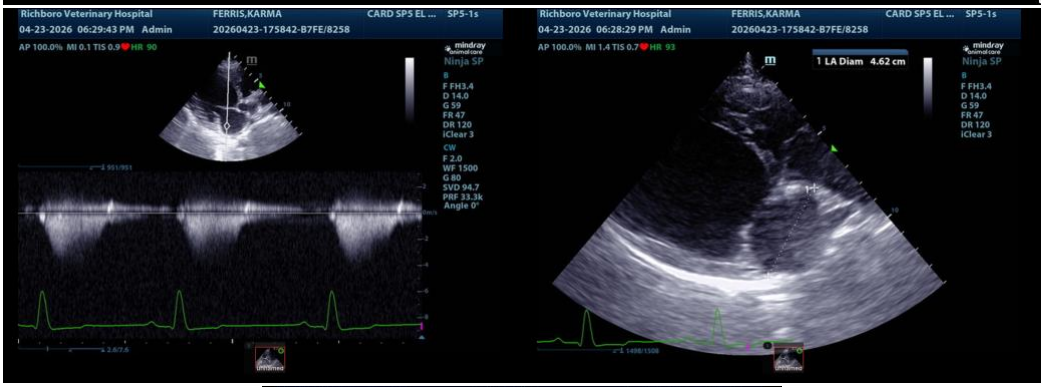

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Eric Lindquist, DMV, DABVP(CFM), Cert. IVUSS,
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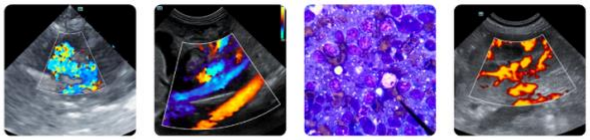

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Canine Dilated Cardiomyopathy

http://www.sonopath.com/DilatedCMY

Description: Dilated cardiomyopathy (DCM) is the most significant acquired cardiac disease of large to giant breed dogs; it especially affects Doberman Pinschers, Great Danes, and Labrador Retrievers. DCM is primarily a systolic dysfunction caused by a progressive decrease in myocardial contractility. As contractility worsens, all chambers of the heart progressively dilate and the walls become increasingly stiff as intracardiac pressure rises. The net result is reduced cardiac output, which causes a decrease in muscle perfusion. As the disease progresses, the left ventricle continues to dilate, causing the mitral valve annulus to widen; cardiac output is further reduced as a result of mitral regurgitation. The reduced cardiac output stimulates the sympathetic nervous system to increase preload (diastolic volume), heart rate, and peripheral vascular resistance. Consequently, there is an increase in volume and pressure, and clinically, it is at this stage of the disease that most animals develop pulmonary edema.

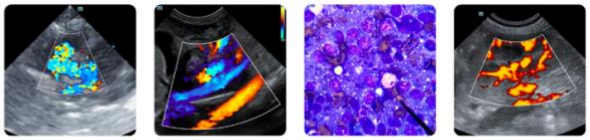
Although the most common etiology is idiopathic, it can also be the result of hypothyroidism, taurine deficiency, L-carnitine deficiency, parvovirus, Lyme disease, and possibly myocarditis. In Doberman Pinschers there is some evidence that there may be a causative genetic mutation. Doxorubicin chemotherapy may induce a DCM-type presentation due to its myocardial toxicity. In Boxers, it is possible for primary myocarditis to precipitate DCM in the later stages of the condition. Hypothyroidism may also be a complicating factor in DCM as it has the potential to further aggravate the systolic dysfunction.

Clinical signs: Clinical signs include weakness, lethargy, exercise intolerance, coughing, anorexia, ascites, syncope, tachypnea, and dyspnea. Pulses are weak and rapid, and often there are pulse deficits. Pulmonary crackles may be auscultated. Jugular distention and muffled heart sounds may also be present.

Diagnostics: It is possible that NT-proBNP testing will be utilized in the future to detect occult DCM. One recent study showed that NT-proBNP levels were higher in dogs with DCM as well as apparently healthy patients that went on to develop DCM within 1.5 years of the samples being obtained.

The echocardiographic parameters for diagnosing DCM include an increased left ventricular (LV) diameter, depressed fractional shortening, and increased EPSS (> 0.8 cm); there is the possibility of an enlarged left atrium. A recent study showed that the best discriminating value for differentiating healthy Doberman Pinschers from those with DCM was the LV diastolic diameter (> 48mm for male dogs and > 46 mm for female dogs).

Ideally, an ECG should be performed with a Holter monitor over a 24-hour period. Fifty to 100 VPCs are considered diagnostic for DCM in Doberman Pinschers when other systemic causes for arrhythmias have been excluded. The absence of detectable VPCs in the 5-minute ECG, however, should not lead to the assumption that the dog is healthy. Even one VPC in a 5-minute



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ECG strongly warrants further examination.

When congestive heart failure (CHF) develops, patients will exhibit an increased respiratory rate and there will be evidence of pulmonary edema on survey radiographs. One might also note elevated NT-proBNP levels.

Treatment: Pimobendan (0.25-0.3 mg/kg PO BID) is a phosphodiesterase inhibitor and positive ionotropic medication. (Note: The latter is a more powerful positive ionotrope than digoxin.) Studies have shown that pimobendan significantly prolongs survivability in Dobermans with DCM and CHF; their mean survival time was 329 days compared to 50 days for those in the placebo group. There was also clinical improvement in dogs that had CHF.

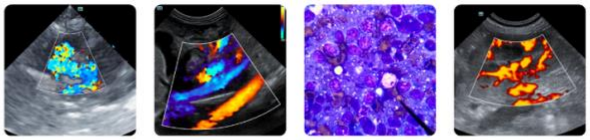
Diuretics: Lasix should be administered at 2-4 mg/kg PO BID-TID as needed and subsequently reduced to the lowest tolerated dose to maintain respiratory comfort, which is approximately 2 mg/kg PO BID (higher doses will be required as the disease progresses). Spironolactone (0.5 mg/kg PO BID) is used primarily for its anti-aldosterone effect and to decrease remodeling of the heart. In human trials, the addition of spironolactone to standard therapy has been shown to increase life span in cases of CHF.

Angiotensin-converting enzyme (ACE) inhibitors: Enalapril (0.5 mg/kg BID) or benazepril (0.5 mg/kg PO Q12-24hr) therapy decreases systemic afterload, which diminishes the chamber stretching effects on the myocardium and thereby improves oxygen carrying capacity. A recent retrospective study showed that the early use of benazepril prolonged the onset of overt DCM in Dobermans with occult DCM.

Anti-arrhythmics: These should be used as needed. In cases of ventricular tachycardia where severe systolic dysfunction is a possibility, amiodarone (10 mg/kg PO Q24hr until stabilized and then 8 mg/kg PO Q24hr) and mexilitine (5-8 mg/kg PO TID) can be used. Although beta blockers, such as sotalol, can be used, they must be prescribed with extreme caution in dogs with severe systolic dysfunction as they have a decreased ionotropic effect. Therapy for atrial fibrillation would include diltiazem (start at 0.5 mg/kg PO TID and increase to a target dose of 2 mg/kg PO TID) and/or digoxin, or amiodarone.

Carvedilol: Carvedilol is a calcium channel blocker, acts as a positive inotrope and free radical scavenger, and has ACE inhibiting effects. The target dose is approximately 1 mg/kg BID; it should be titrated up gradually from 0.25 mg/kg with dose adjustments made every 2 weeks until the ideal dose is reached.

Morphine: Morphine (0.025-0.05 mg/kg IV) decreases anxiety and improves respiratory efficiency and oxygen consumption. Morphine also dilates splanchnic vasculature and increases venous capacitance thereby reducing pulmonary edema.



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Supplements: These may aid in some cases of DCM. In taurine-deficient patients (based on plasma or whole blood taurine levels), one should supplement taurine at 500 mg PO BID and L-carnitine at 175 mg/kg PO BID. Coenzyme Q10 is also used as a means of myocardial ischemic prevention (30-90mg BID). Omega-3 fatty acid supplements can help with cell membrane stabilization and be used as inflammatory mediators (EPA 40mg/kg, DHA 25mg/kg PO Q24hr).

Salt restriction: An optimized high-quality protein diet (4gm/100kcal) that is mildly restricted in salt should be considered to minimize the onset of sodium-induced volume overloads.

Monitoring: Serum chemistry (i.e., BUN/creatinine, electrolytes), blood pressure, and thoracic radiographs should be performed as needed 5-7 days after therapy has been initiated. One should conduct a follow-up echocardiogram in 1-6 months to assess myocardial function and the possible progression of the systolic dysfunction; medication should be changed or modified as needed. An electrocardiogram should be repeated as needed to monitor arrhythmias and the patient's response to therapy. The best means of monitoring progress at home is to observe the resting respiratory rate. A study showed that the resting respiratory rate obtained at home was the strongest predictor of improving or worsening of CHF and/or pulmonary edema.

Prognosis: The prognosis is variable and largely dependent on the individual animal and its response to therapy.

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