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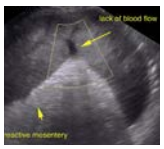
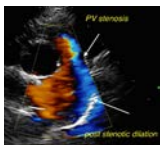
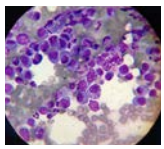
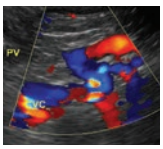

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1-800-838-4268 info@sonopath.com

PATIENT
Raven Thornton

SPECIES
Canine

BREED
Labrador Retriever X

SEX
Spayed Female

AGE
6 Years

WEIGHT
63 Pounds

INTERPRETED BY
Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY
Kelly Vazquez

HOSPITAL NAME
Westwood Regional

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Dr. Silver

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41665

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PRESENTING CLINICAL SIGNS

Pericardial effusion vs. pleural effusion. No reported meds/blood work. In O2 support cage.

ULTRASONOGRAPHIC EXAMINATION OF THE HEART & ABDOMEN

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	1.9	7	16	1.41
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		0.61	0.50		5.7	5.64	

Cardiac Presentation

The cardiac presentation presented severe volume overload in the left and right atrium with lack of contractility. Left ventricular dilation noted. Aortic velocity was subnormal. Severe mitral insufficiency noted, >4.0 m/sec. Tricuspid insufficiency noted at 3.0 m/sec. Pericardial effusion was not present. Some pleural effusion was noted as well as passive congestion leading to secondary ascites. The vena cava was severely dilated at the level of the diaphragm, measuring 2.19 cm.

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. The ureters were not visible which is normal. No uroliths or sediment were visualized and anechoic urine was present. No evidence of inflammatory or neoplastic changes were noted. Ureteral papillae were normal.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The right kidney measured 5.46 cm. The left kidney measured 5.42 cm.

Adrenal Glands

Both **adrenal glands** were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The right adrenal gland measured 1.98 cm x 0.77 cm at the caudal pole and 1.04 cm at the cranial pole. The left adrenal gland measured 2.08 cm x 0.72 cm at the caudal pole and 0.47 cm at the cranial pole.

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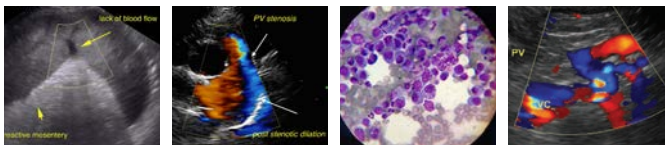

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Spleen

The **spleen** presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes were noted.

Liver

The **liver** was uniform. The gallbladder wall was mildly edematous owing to the ascites. Hepatic veins were dilated.

Gastrointestinal

Examination of the **gastrointestinal tract** revealed a stomach and intestine free of stasis, of normal wall thickness, acceptable curvilinear mural detail, and peristaltic activity. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively. No obstructive or overt infiltrative disease was noted. No associated abnormal lymphatic activity was noted.

Pancreas

The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

ULTRASONOGRAPHIC FINDINGS

- Bi-atrial enlargement with mitral and tricuspid insufficiency
- Pleural effusion
- Ascites
-

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The pleural effusion isn't typical of cardiac failure, yet can occur occasionally. Recommend treating for DCM in this patient.

Given the myocardial insufficiency noted in this patient Nutritional Cardiomyopathy (Taurine deficiency/grain free diet) and infectious agents should be considered. Infectious agents such as Bartonella, Leptospirosis, Parvo (current or historical), Bacterial sepsis and less likely and regional infectious agents such as Trypanosoma, Toxoplasmosis and Babesia should be considered as underlying clinical players.

Quadrotherapy indicated with Pimobendan at 0.3 mg/kg BID, ACE inhibitor 0.5 mg/kg SID progressing to BID, Lasix at 3-4 mg/kg BID, and Spironolactone at 1-2 mg/kg BID. Sildenafil could also be considered at 1-2 mg/kg BID. However, recommend waiting 48-72 hours. Cage rest warranted. This patient is at high risk for sudden death. EKG indicated and blood pressures. BUN/Creatine should be monitored carefully. Target sleeping respiratory rate of <25/min. Recheck echo in 10-14 days if the patient is able to survive this initial episode.

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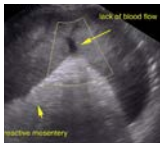
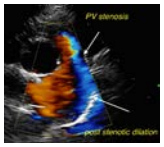
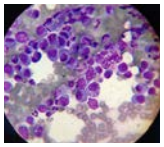
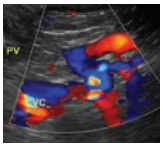

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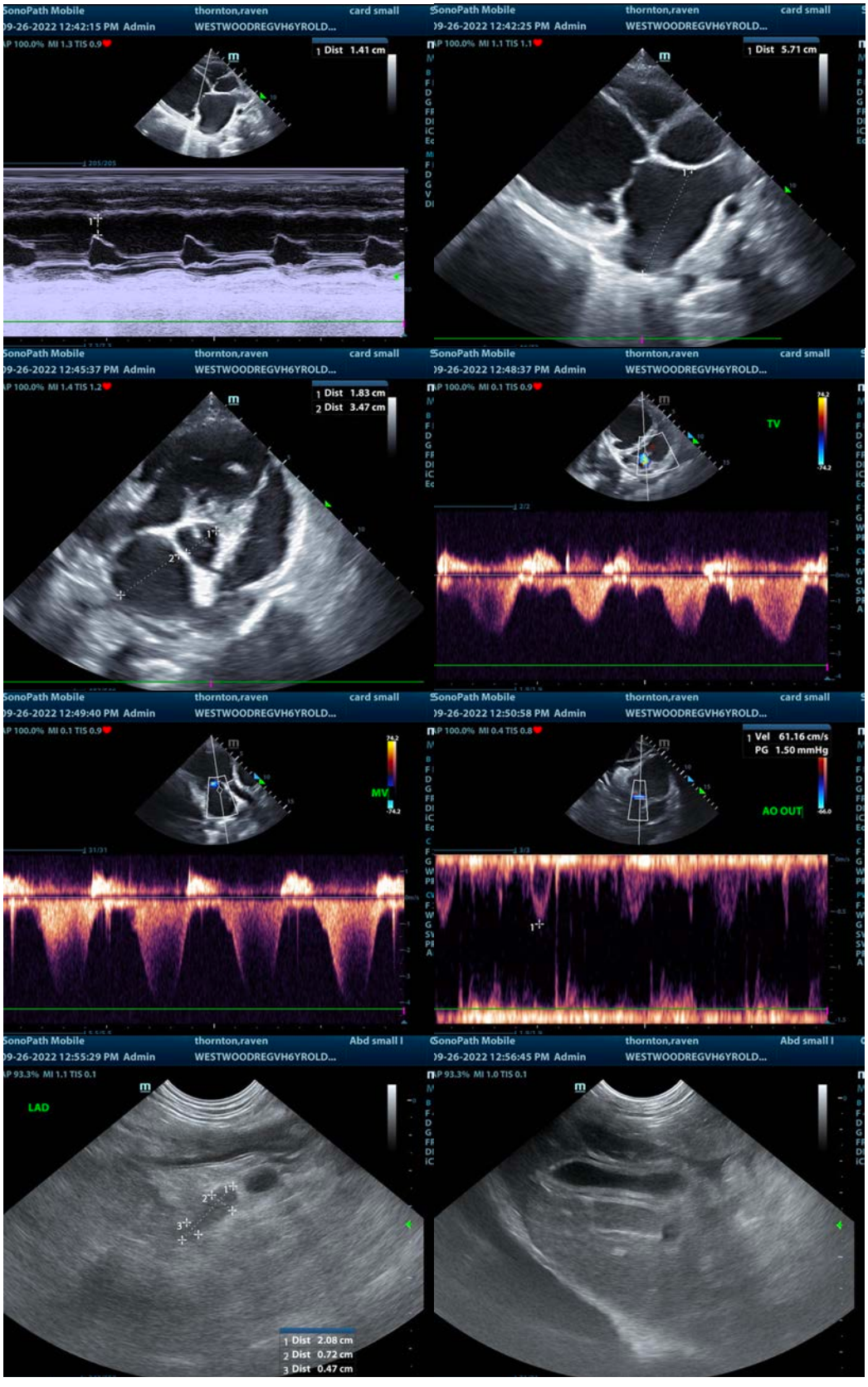

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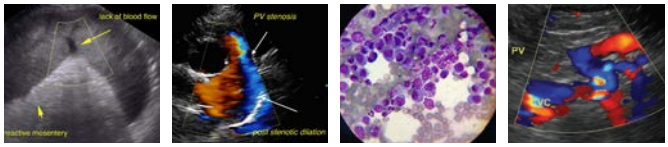

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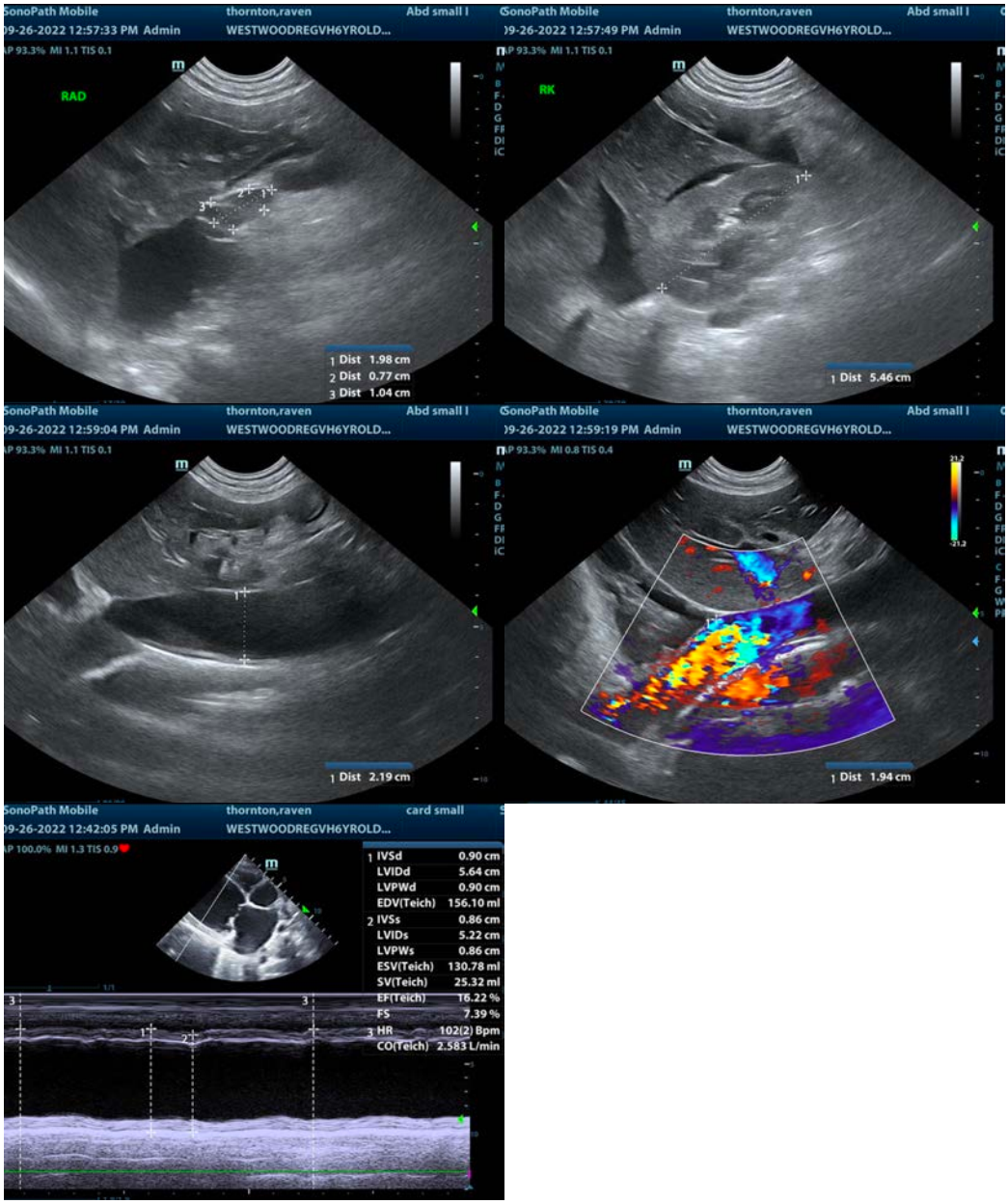

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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, Cert. IVUSS, CEO of SonoPath.com

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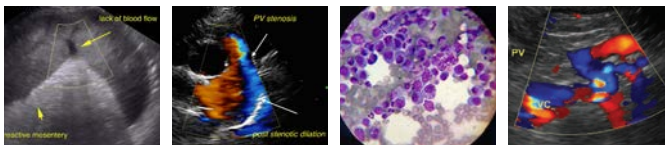

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Canine Dilated Cardiomyopathy

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.

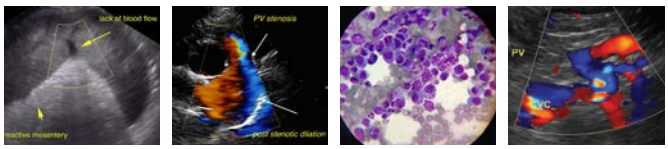
Although the most common etiology is idiopathic, it can also be the result of hypothyroidism, taurine deficiency, L-carnitine deficiency, parvovirus, Lyme disease, and possibly myocarditis. In Doberman Pinschers there is some evidence that there may be a causative genetic mutation. Doxorubicin chemotherapy 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



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assumption that the dog is healthy. Even one VPC in a 5-minute 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.

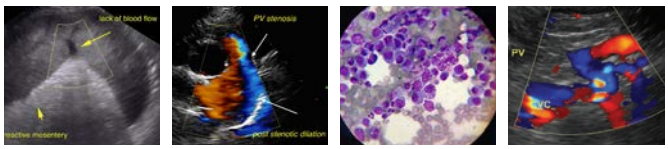
Angiotensin-converting enzyme (ACE) inhibitors: Enalapril (0.5 mg/kg BID) or benazepril (0.5 mg/kg PO Q12-24hr) therapy decreases systemic afterload, which diminishes the chamber stretching effects on the myocardium and thereby improves oxygen carrying capacity. A recent retrospective study showed that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

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



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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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