Nutritional management of an American cocker spaniel with dilated cardiomyopathy secondary to nutritional carnitine, taurine and sulfur amino acid deficiency

Case Summary:

A 3 year old male castrated American cocker spaniel was referred to an emergency service for dyspnea and presumptive congestive heart failure. After stabilization, the dog was transferred to the cardiology service and a diagnosis of dilated cardiomyopathy was made. A nutritional consultation revealed a dietary history of having been fed a nutritionally unbalanced vegan diet, which resulted in low concentrations of plasma taurine and carnitine. A complete and balanced diet with taurine and L-carnitine supplementation was instituted, which resulted in cardiac improvement over the following ten months.

** This case report was originally submitted by an ACVN candidate but has been edited and reformatted to meet the Instructions for the Writing and Evaluation of ACVN Case Reports. The example case reports are intended to serve as an illustration of the report instructions, format and a clinical presentation that had been found acceptable in the past. Editorial liberties were taken to complete the information essential to the report. Example reports should not be taken as an ACVN's endorsement of any specific nutritional approach or rationale*

A 3 year old 15 kg male castrated American cocker spaniel, up-to-date on core vaccinations, was referred to a teaching hospital emergency service with a history of collapsing episodes. These episodes started a month earlier and had increased in frequency from once weekly to daily. The dog's most recent episode of collapse was accompanied by coughing and dyspnea. The patient was first seen by the dog's primary care veterinarian and a presumptive diagnosis of congestive heart failure (CHF) was made based on thoracic radiography. The dog was administered 2mg/kg of furosemide intravenously and referred to an emergency service for further diagnostics and treatment.

Upon presentation, the physical examination revealed abnormalities of mild dehydration (approximately 5%), tachycardia, weak pulses, pale mucus membranes, a grade III/VI left-sided apical systolic heart murmur at the apex, and increased lung sounds bilaterally characterized as late inspiratory crackles. The tachycardia was further defined by electrocardiogram as a sinus rhythm at a rate of 170 beats per minute (bpm). The blood pressure was 100 mmHg systolic 49 mmHg diastolic with a mean arterial pressure of 86 mm Hg. These findings in conjunction with previous diagnostic test results were consistent with a diagnosis of left sided congestive heart failure. Point of care bloodwork at this time revealed normal concentrations of blood urea nitrogen (BUN), glucose, and electrolytes and normal hematocrit, total solids, acid-base values. A dose of 30 mg furosemide[a] was administered intravenously 3h (hour) after initial administration by the referring veterinarian, and the patient was placed in an oxygen cage.

Differential diagnoses at this point included valvular, myocardial, pericardial, and vascular cardiac diseases. Congenital valvular diseases include aortic, subaortic, and pulmonic stenosis, and mitral/tricuspid valve malformation. Acquired valvular malformations include myxomatous atrioventricular valvular disease, ruptured chordate tendineae, and bacterial endocarditis. Myocardial diseases include ventricular and atrial septal defects, dilated, right ventricular, and hypertrophic cardiomyopathies, and myocarditis. Pericardial diseases include cardiac neoplasia causing pericardial effusion, infectious agents, and idiopathic pericardial hemorrhage. Vascular diseases include heartworm and a malformation such as patent ductus arteriosus.

Two hours after arrival the dog appeared more stable and 2-view thoracic radiographs were taken (Figure #1). The right-lateral radiograph revealed a heart that spanned 4 intercostal

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[a] Furosemide injectable, 50mg/ml, Butler Animal Health Supply, Dublin, OH

spaces and the dorsal-ventral radiograph showed a heart that encompassed 75% of the thoracic cavity in height with tracheal elevation. The radiologist's interpretation was a generalized cardiomegaly with left atrial enlargement and caudodorsal-to-diffuse airspace pattern; consistent with cardiogenic edema as a result of left-sided congestive heart failure secondary to dilated cardiomyopathy (DCM). The dog was returned to the intensive care unit and after placing an intravenous catheter[b] (right cephalic vein), continuous rate infusion (CRI) of furosemide was started (0.7mg/kg/h) while the dog remained in an oxygen cage.

Day 2 of hospitalization, the dog demonstrated an improved respiratory pattern and heart rate (decreased to 140 bpm). The patient was transferred to the cardiology service where blood was drawn for a complete blood count (CBC) and serum biochemistry profile. The abnormalities found in that laboratory data included increased concentrations of BUN (53 mg/dL) and creatinine (2.0 mg/dL) (Table #1, day 2). The azotemia was attributed to a prerenal etiology since these values were within normal limits prior to initiation of the furosemide CRI. To further evaluate heart function and degree of compromise, an echocardiogram was performed (Figure #2). The results of echocardiogram were left atrial and ventricular enlargement, enlarged pulmonary veins, decreased systolic function, and thickened mitral valve leaflets resulting in mitral regurgitation consistent with the thoracic radiographic assessment of DCM. Repeat thoracic radiographs were taken to assess efficacy of the overnight furosemide CRI treatment and an improvement in cardiogenic edema was noted.

The diagnosis was DCM with secondary congestive heart failure and the patient was started on a regimen of furosemide[c] (25 mg PO q8h x 3days, then 25 mg PO q12h thereafter), enalapril[d] (5 mg PO q12h), and pimobendan[e] (2.5 mg PO q12h). Furosemide is a loop diuretic commonly used for the treatment of congestive heart failure and pulmonary edema. Enalapril is an angiotensin converting enzyme inhibitor that is given to alleviate the effects of the renin-angiotensin-aldosterone-system. Pimobendan is a phosphodiesterase III inhibitor and inodilator that combine the central myocardial effects of a positive inotropic agent with those of a peripheral vasodilator.[1]

A nutrition consultation was requested day 3 of hospitalization due to the nature of the

[b] BD InsyteTM I.V. catheter, Becton Dickinson Inc., Sandy UT
[c] Furosemide, 50 mg tablet, Intervet, Inc., Summit, NJ
[d] Enalapril, 5 mg tablet, Wockhardt USA LLC, Parsippany, NJ
[e] Pimobendan, 5 mg tablet, Boehringer Ingelheim, St. Joseph, MO

problem list and breed predisposition to taurine deficiency. A body weight of 15 kg, with a body condition score (BCS) of 4/9 and a muscle condition score (MCS) of mild muscle wasting were determined by the Candidate.[2,3] While the majority of dogs that develop DCM are large and giant breeds including the Doberman pinscher, Great Dane, Scottish deerhound, and Irish wolfhound, low blood taurine concentrations have been reported in other breeds that develop DCM, which include the American cocker spaniel, Golden retriever, Labrador retriever, Newfoundland Portuguese water dog, and Irish wolfhound.[4] The Candidate obtained a diet history and discovered that the dog had been consuming a vegan diet (created by another vegan pet owner) for the past two years with no vitamin or mineral supplementation (Table #2). Blood was collected for whole blood taurine[f] concentrations, and plasma carnitine[g] concentrations. Oral supplementation of both taurine[h] (500 mg q12h) and L-carnitine[i] (1g q12h) were empirically started at standard doses while awaiting these results. An ophthalmic examination was also performed to investigate the possibility of bilaterally symmetric hyper reflective retinal lesions, which is similar to central retinal degeneration lesions in cats with low plasma taurine.[5] A focal triangular shaped cataract on the posterior cortical lens was found in both eyes but was not considered to be consistent with the diagnosis of taurine deficiency. One week after samples were drawn, results indicated whole blood taurine (18 nmol/mL) and plasma carnitine (12.6 μmol/L) were below normal reference ranges (Table #1, day 2).

The nutrient profile of homemade vegan diet was reconstructed using a computer software[j] and determined to be deficient in a number of nutrients when compared to AAFCO and NRC recommendations (Table #3).[6,7] Of particular interest in this cardiac case was that the vegan diet only met 31% of the sulfur based amino acid (methionine and cystine) requirements when compared to the NRC recommended allowance (RA) and only 42% of minimum AAFCO recommendation. Both NRC RA and AAFCO minimum recommendations suggest the concentration of dietary nutrients that includes a bioavailability factor. Therefore these were appropriate reference standards against, which to assess a vegan diet where nutrient bioavailability is known to be hindered by fiber interactions.[6] While a vegan diet may be used to

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[f] Amino Acid Laboratory at University of California, Davis, California
[g] Neuromuscular Laboratory at University of California, San Diego, California
[h] Taurine, 250 mg tablet, Pet Ag Inc., Hampshire, IL
[i] L-carnitine, 100 mg/mL, oral solution, Rising Pharmaceuticals, Inc., Allendale, NJ
[j] Balance IT®, Davis Veterinary Medical Consulting Inc, Davis, CA

feed a dog, the recipe should be formulated by a qualified individual to ensure the diet is nutritionally complete and balanced according to current daily nutrient intake recommendations appropriate for the life stage of the animal.

The Candidate recommended a dietary change to a canine veterinary therapeutic diet[k] (Table; #3). This diet was chosen for a several reasons. Although it did not contain taurine, the sulfur amino acid (methionine and cystine) content was twice the NRC RA. This was considered beneficial, since taurine is a sulfur containing amino acid synthesized from cystine, methionine is the precursor of cystine, and the methylation of lysine by a methionine is an intermediate step in the biosynthesis of carnitine.[7,8]

The recommended diet also had relatively high concentrations of omega-3 (n3) fatty acids: eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) at 30 times the NRC RA but also had an adequate concentration of omega-6 (n6) fatty acids (i.e., linoleic acid) for proper dermatologic maintenance. Dogs with CHF have been reported to have a relative deficiency of EPA and DHA compared to unaffected dogs.[9] EPA has been reported to dampen genetic transcription of inflammatory cytokines such as tumor necrosis factor alpha, interleukin-1β, interleukin-6, and NF-kβ that are related to protein catabolism seen in cachectic patients.[10,11,12,13] Additionally, omega-3 fatty acids may have antiarrhythmic effects and enhance appetite in cachectic canine patients.[9,14] Elevated concentrations of alpha tocopherol (10 x NRC RA) were present in this diet to help mediate the oxidative stress that accompanies the inflammation associated with cardiac disease. A moderate concentration of fat (~5 g/100 kcal) and high protein to calorie ratio (~10 g/100 kcal) were also beneficial to help maintain body weight, and in this case improve lean body mass, appropriate given the dog had no renal compromise (Table #1, day 4). This diet was nutritionally complete and balanced, and would replete the remaining nutrient deficiencies (calcium, copper, zinc, vitamin D, pantothenic acid and B_{12}) relative to either NRC or AAFCO recommendations that had not manifested clinically. The canned versus dry version of the diet was selected to make a dietary change most like the texture, consistency and palatability of the homemade diet the dog had been eating in an effort to

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[k] JM Joint Mobility Canine Formulas, Purina Veterinary Diets, Nestlé Purina PetCare Company, St. Louis, MO., canned with 23.8% moisture and 1.15 kcal/g as fed containing ingredients: Water meat by-products, salmon, liver, corn gluten meal, rice, turkey, oat fiber, fish oil, minerals and vitamins. 2011 manufacturer's product guide

reduce the likelihood of food rejection.

Since the dog was in good body condition and the daily energy intake of the vegan diet was approximately 830 kcal per day [= 863 g/d x 0.962 kcal/g] (Table #2), the initial food dose for the recommended diet was 2 cans (372 g/can) per day divided into 2 meals [426 kcal/can x 2 = 852 kcal/day]. This seemed a reasonable starting point as it was equivalent to 1.6 x Resting Energy Requirement (RER) [= 70 x $(15\ kg)^{0.75}$].[15] Given the dog would be consuming 852 kcal/day, and the dietary product selected contained 160 mg EPA/100 kcal, this dog would be consuming 1.36 g EPA/day. The current recommendation suggests 40 mg/kg EPA for dogs with cardiac disease, which would be a minimum of 600 mg EPA/day for this 15 kg dog.[9] While the EPA intake was high (1.36 g/d) relative to the recommendation for this dog (0.6 g/d), the commercial product still had an n6:n3 ratio[1] of 1.6:1, which has not been shown to inhibit platelet aggregation.[16]

The two major nutrients of concern for this cardiac case fed a vegan diet were taurine and carnitine. Taurine is typically found in high concentrations in cardiac and skeletal muscle. While having numerous roles in mammalian physiology, the importance of taurine in myocardial function has been recognized.[17] Proposed mechanisms of taurine function in the heart include calcium homeostasis; specifically modulation of sarcoplasmic reticular Ca^{2+} release required for normal systolic and diastolic function.[18] Taurine has been considered an essential amino acid in cats since 1987 when first described in the etiology of DCM.[19] Such a deficiency as an etiology of DCM in dogs was debated for some time, as dogs have the ability to synthesize taurine with increased activity of hepatic enzymes cystine dioxygenase and cystine sulfinic acid decarboxylase as compared to cats. This idea was first challenged when taurine deficiency was linked to DCM in foxes, a member of the family Canidae, in 1989.[20] Subsequently, the results of a Multicenter Spaniel trial found low plasma concentrations of taurine and normal concentrations of total, free, and esterified carnitine in American cocker spaniels diagnosed with DCM. Patients treated with both taurine and L-carnitine responded well. The conclusion drawn was that taurine supplementation resulted in a significantly better prognosis and improvement in cardiac function but whether L-carnitine supplementation was needed could not be definitely determined.[21]

L-carnitine is a water-soluble molecule obtained from protein in the diet or endogenous

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[1] n6 to n3 ratio = linoleic acid + arachidonic acid to eicosapentaenoic acid + docosahexaenoic acid + alpha linolenic acid concentrations

synthesis in the liver, and approximately 95-98% is stored in the skeletal and cardiac muscle of dogs.[22] A primary function of L-carnitine is as a cofactor to other enzymes that allow for "shuttling" of long chain fatty acids (LCFA's) across the inner membrane of the mitochondria into the matrix where beta-oxidation occurs and energy is produced. Approximately 60% of net energy production in heart muscle is acquired from beta-oxidation of LCFA's demonstrating the importance of L-carnitine. L-carnitine concentrations in plasma do not necessarily correspond to amounts present in the myocardium; however, obtaining an endomyocardial biopsy to measure those concentrations was considered neither practical nor in the best interest of this patient.[23] It is known that people with damaged myocardiocytes will "leak" L-carnitine out of cells resulting in low myocardial carnitine concentrations, suggesting that low concentrations occur as a consequence rather than as an etiology of cardiac disease.[22]

The owner was informed that taurine and L-carnitine supplementation may result in partial reversal of the disease and a better prognosis. While cardiac structure may not necessarily return to normal, some patients may be weaned off cardiac medications. If taurine supplementation was implemented and cardiac structure did not improve, then taurine deficiency was unlikely the etiology of this patient's DCM, which would mean a poor prognosis, survival time of 3-24 months, and potential sudden death. While cats may have a return to normal cardiac function within 3-6 months with taurine supplementation, dogs have not had as a dramatic improvement. Despite clinical improvement, improved echocardiogram parameters may not be noted for 4 months or longer.[21,24]

The dog returned to the teaching hospital for rechecks at 2, 4, 8, and 10 months post diagnosis. At each recheck the owner reported the dog had a good appetite, there were no palatability issues with the recommended diet and the body weight fluctuated within 0.5 kg of the dog's weight on presentation and maintained a BCS of 4/9. The MCS status; however, was considered normal by 4-month recheck visit. The dog's activity improved; walking up to 3 miles per day as of the 4-month recheck appointment with no further collapsing episodes. The cardiac murmur had decreased in intensity but stabilized at a grade II/VI with mitral regurgitation, and heart rate ranged from 84 to 96 bpm.

The echocardiogram at all 4 recheck visits supported a positive progression in systolic function with continued mild improvement in the size and function of the left atrium and ventricle; though the left ventricle remained globoid in shape due to chamber dilation as would be expected. At the 8-month recheck visit, the left atrium size was considered within normal limits. As the dog improved clinically and diagnostically, furosemide and pimobendan were discontinued. A recheck visit at 10 months confirmed discontinuation of the furosemide and pimobendan was possible as the dog remained stable. The 10-month radiographs (Figure #3) showed improvement in heart size and no evidence of CHF with a significant reduction in mitral regurgitation as viewed via echocardiogram (Figures #4). Renal panels to assess side effects of enalapril (Table #1 at 8 and 10 months) indicated no decrease in kidney function. Blood collected for taurine and L-carnitine concentrations at the 2- and 8- month recheck appointments, revealed increasing concentrations of each. Due to cost of the L-carnitine supplement and the number of medications, the L-carnitine was discontinued, the taurine supplement dose was reduce by 50% but the enalapril was continued as originally prescribed.

In conclusion, the dietary change with targeted supplementation of taurine and L-carnitine resulted in an improved cardiac function and quality of life for this dog as evidenced by walking 3 miles daily. The dog's prognosis improved from poor to fair with an expected plateau to be reached in cardiac remodeling over the next year. Given no evidence of clinical decline, a 6-month recheck schedule with blood renal values was suggested, while blood taurine and plasma carnitine concentrations rechecks were recommended annually.

References:

[1] Bowles D, Fry, D. Pimobendan and its use in treating canine congestive heart failure. Compend Contin Educ Vet. 2011 Nov;33(11):E1.

[2] Laflamme DP. Development and validation of a body condition score system for dogs: A clinical tool. Canine Practice - Nutrition. 1997;22(4):10-15.

[3] WSAVA. Muscle Condition Score. Muscle condition score - a new tool for patient assessment http://www.wsava.org/sites/default/files/Muscle%20condition%20score%20chart%202013.pdf. 2013 Accessed 3/1/2014.

[4] Meurs, KM. Canine Dilated Cardiomyopathy-Insights into Diagnosis and Management. In Proceeding of the North Amer Vet Conf. 2005. Jan 8-15, Orlando, FL p119-121.

[5] Hayes, KC. Nutritional problems in cats: taurine deficiency and vitamin A excess. Can Vet J. 1982;23:2-5.

[6] Association of American Feed Control Officials Incorporated. AAFCO Official Publication. c2008. Chapter 4, Model Regulations for Pet food and Specialty Pet food Under the Model Bill: p.150-151.

[7] National Research Council. Ad hoc Committee on Dog and Cat Nutrition. National Research Council Nutrient Requirements of Dogs and Cats. Washington DC: The National Academies Press; c2006. Table 15-5, Nutrient Requirements for Adult Dogs for Maintenance; p.359-360.

[8] Cox RA, Hoppel CL. Biosynthesis of carnitine and 4-N-trimethylaminobutyrate from lysine. Biochem J. 1973 Dec;136(4):1075-82.

[9] Freeman LM, Rush JE, Kehayias JJ, et al. Nutritional alterations and the effect of fish oil supplementation in dogs with heart failure. J Vet Intern Med. 1998;12:440–448.

[10] Endres S, Ghorbani R, Kelley VE, et al. The effect of dietary supplementation with n-3 polyunsaturated fatty acids on the synthesis of interleukin-1 and tumor necrosis factor by mononuclear cells. N Engl J Med. 1989;320:265–271.

[11] Caughey GE, Mantzioris E, Gibson RA, et al. The effect on human tumor necrosis factor alpha and interleukin 1 beta production of diets enriched in n-3 fatty acids from vegetable oil or fish oil. Am J Clin Nutr. 1989;63:116–122.

[12] Kang JX and Weylandt KH. Modulation of inflammatory cytokines by omega-3 fatty acids. Subcell Biochem. 2008;49:133–143.

[13] LeBlanc CJ, Horohov DW, Bauer JE, et al. Effects of dietary supplementation with fish oil on in vivo production of inflammatory mediators in clinically normal dogs. Am J Vet Res. 2008;69:486–493.

[14] Smith CE, Freeman LM, Rush JE, et al. Omega-3 fatty acids in Boxer dogs with arrhythmogenic right ventricular cardiomyopathy. J Vet Intern Med. 2007;21:265–273.

[15] Debraekeleer J, Gross KL, Zicker, SC. Small Animal Clinical Nutrition. 5th ed. Topeka, KS: Mark Morris Institute; c2010. Chapter 13, Feeding Young Adult Dogs: Before Middle age; p.260.

[16] Lenox CE, Bauer JE. Potential adverse effects of omega-3 Fatty acids in dogs and cats. J Vet Intern Med. 2013 Mar-Apr;27(2):217-26.

[17] Schaffer SW, Chian JJ, Ramilla KC, et al. Physiologic roles of taurine in heart and muscle. J Biomed Sci. 2010; 17(Suppl 1):S2.

[18] Entman ML, Bonnett EP, Bressler R. The effect of taurine on cardiac sarcoplasmic reticulum. Life Sci. 1977;21:543-550.

[19] Pion PD, Kittleson MD, Rogers, QR, et al. Myocardial failure in cats associated with low plasma taurine: a reversible cardiomyopathy. Science. 1987;237(4816):764-768.

[20] Moise NS, Pacioretty LM, Kallfelz FA, et al. Dietary taurine deficiency and dilated cardiomyopathy in the fox. Am Heart J. 1991;121(2 Pt 1):541-547.

[21] Kittleson MD, Keene B, Pion PD, et al. Results of the multicenter spaniel trial (MUST): taurine- and carnitine-responsive dilated cardiomyopathy in American cocker spaniels with decreased plasma taurine concentration. J Vet Intern Med. 1997;11(4):204-211.

[22] Neely JR, and Morgan HA. Relationship between carbohydrate metabolism and energy balance of heart muscle. Annu Rev Physiol. 1974;36:413-459.

[23] Keene BW, Kittleson MD, Rush JE, et al. Frequency of myocardial carnitine deficiency associated with spontaneous canine dilated cardiomyopathy. Proceedings of the Sixth Annual Veterinary Medical Forum. Washington, DC; 1988:757.

[24] Gavaghan BJ, Kittleson MD. Dilated cardiomyopathy in an American cocker spaniel with taurine deficiency. Aust Vet J. 1997;75(12):862-868.

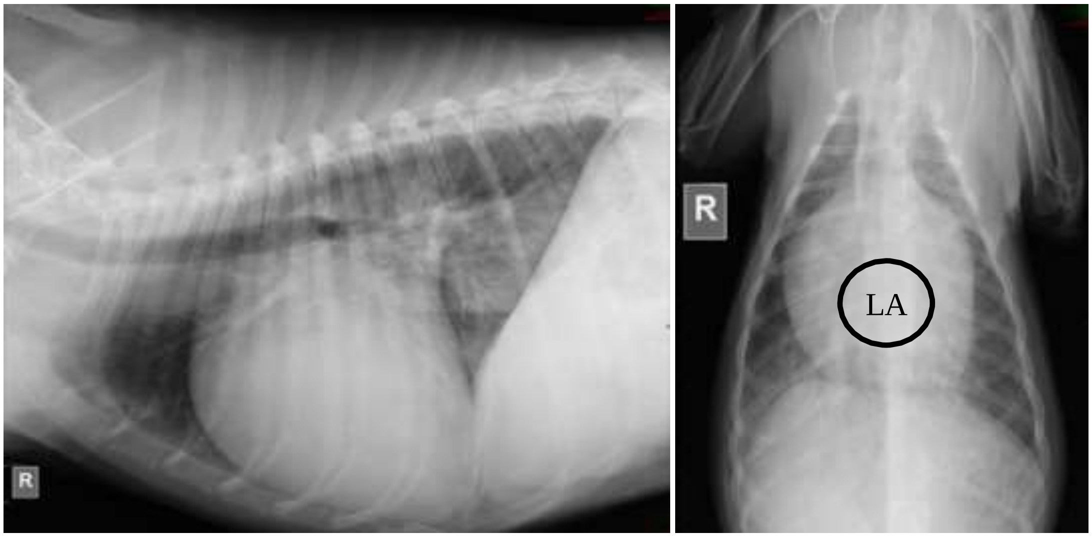

Figure #1: Right lateral and dorsoventral thoracic radiographs at time of diagnosis demonstrating generalized cardiomegaly with left atrial enlargement and caudodorsal-to-diffuse airspace pattern characteristic of pulmonary edema due to congestive heart failure. Right lateral shows a heart that is 4 intercostal spaces (reference range 2.5-3.5 IC spaces) and 75% height of thoracic cavity (reference range <66% thoracic cavity). Dorsoventral view shows enlarged radiopaque circular left atrium (LA).

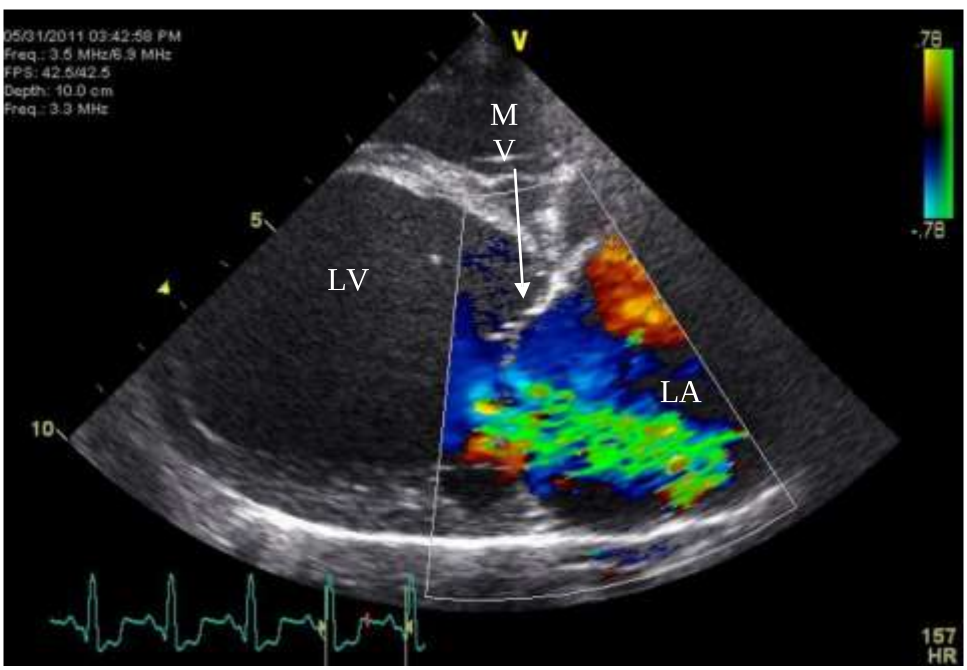

Figure #2: Two-dimensional echocardiogram long-axis view at time of diagnosis. This echocardiogram demonstrates the enlarged left ventricle (LV) and left atrium (LA). The thickened mitral valve leaflet is labeled (MV). Color flow Doppler demonstrates mitral regurgitation.

Table #1: Blood and plasma parameters measured over 10 months*†

	Time after ER Admission				
Parameter Measured (units)	Day 2	Day 4	2 Months	8 Months	10 Months
Sodium (142-150 mEq/L)	145.0	143.0		148.0	146.0
Potassium (3.8-5.4 mEq/L)	4.1	4.0		3.8	4.1
Chloride (105-116 mEq/L)	105.0	106.0		115.0	115.0
Bicarbonate (15-25mEq/L)	22.0	20.0		19.0	20.0
Anion gap (14-24 mEq/L)	24.0	23.0		18.0	15.0
Urea N (10-32 mg/dL)	**53.0**	11.0		17.0	16.0
Creatinine (0.6-1.4 mg/dL)	**2.0**	1.0		0.9	0.8
Calcium (9.3-11.4 mg/dL)	11.1	10.0		10.5	9.9
Phosphate (2.9-5.2 mg/dL)	4.8	2.8		2.9	3.2
Albumin (3.1- 4.2 g/dL)	3.4	3.2		4.2	3.6
Cholesterol (138-332 mg/dL)	143.0	151.0		247.0	229.0
Taurine (60-120 nmol/mL)	**18.0**		**194.0**	**259.0**	
Total carnitine (17-43 µmol/L)	**12.6**		**70.2**	**273.6**	
Free carnitine (17-38 µmol/L)	**10.8**		**62.5**	**209.4**	
Esters-carnitine (0.0-23 µmol/L)	1.8		7.7	**64.2**	
Ratio ester/free (0.0-0.54)	0.2		0.3	0.1	

* Blank = test not done

† Bolded values are outside of reference normal range

Table #2. Homemade vegan diet composition

Ingredients	USDA NDB #*	Amount consumed/day	Amount converted to g/d†
Rice, brown long rain, cooked	20037	1 cup	202
Barley, pearled, cooked	20006	1 cup	158
Lentils, mature seeds cooked w/o salt	16070	1 cup	200
Oatmeal, instant, regular cooked w/water	08121	8 vol oz	118
Pumpkin, canned w/o salt	11424	12 vol oz	184
Olive oil	04053	¼ tsp	1.1
Total			863‡

* USDA National Food Composition Database https://ndb.nal.usda.gov/ndb

† Dry matter 24.2%; 0.962 Kcal/Kg as fed

‡ Estimated daily energy intake = 830 kcal [863 g/d x 0.962 kcal/g]

Table #3: Nutrient Profile comparison of NRC recommended allowance (RA), AAFCO Adult, Owner Vegan Diet and Candidate Recommended Veterinary Therapeutic Diet for Dogs.*

Nutrient (units/100 kcal)	NRC RA[7]	AAFCO Adult Min[6]	Vegan Diet From Owner†	JM Veterinary Therapeutic Diet‡
Protein (g)	2.50	5.14	4.09	9.62
Methionine + cystine (g)	0.16	0.12	**0.05**	0.32
Fat (g)	1.38	1.43	**1.17**	4.92
Linoleic acid (g)	0.28	0.29	**0.09**	0.46
α-linolenic acid (g)	0.01	NR$	**0.00**	0.04
EPA + DHA (g)	0.01	NR	**0.00**	0.31
Carbohydrate (g)	NR	NR	18.61	7.00
Fiber (g)	NR	NR	3.30	1.22
Calcium (g)	0.10	0.17	**0.02**	0.40
Phosphorus (g)	0.08	0.14	0.11	0.30
Sodium (g)	0.02	0.02	0.05	0.08
Chloride (g)	0.03	0.03	NA**	0.26
Potassium (g)	0.10	0.17	0.14	0.33
Magnesium (g)	0.02	0.01	0.04	0.02
Iron (mg)	0.75	2.30	1.35	6.64
Copper (mg)	0.15	0.21	**0.08**	0.44
Zinc (mg)	1.50	3.40	**0.75**	6.67
Manganese (mg)	0.12	0.14	0.30	0.43
Selenium (µg)	8.75	3.00	**3.00**	29.00
Iodine (µg)	22.00	43.00	NA	97.00
Vitamin A (IU)	126.33	142.90	3190	22860
Cholecalciferol (IU)	13.60	14.30	**0.00**	76.60
Thiamin (mg)	0.06	0.03	0.15	0.92
Riboflavin (mg)	0.13	0.06	**0.05**	0.39
Pyridoxine (mg)	0.04	0.03	0.08	0.33
Niacin (mg)	0.43	0.33	0.67	3.53
Pantothenate (mg)	0.38	0.29	**0.23**	1.01
Cobalamin (µg)	0.88	0.60	**0.00**	4.00
Folic Acid (µg)	6.75	5.00	24.99	51.00
Choline (mg)	42.50	34.30	NA	48.40
Alpha tocopherol (IU)	1.13	1.40	**0.30**	12.36

* Bolded values are below NRC RA Recommendations

† Reconstructed from USDA National Food Composition Database https://ndb.nal.usda.gov/ndb

‡ Reconstructed using data obtained from Nestlé Purina PetCare Company and 2011 product guide

$ No requirement

** Data not available (NA) in USDA National Food Composition Database used by Balance IT® software, Davis Veterinary Medical Consulting Inc, Davis, CA

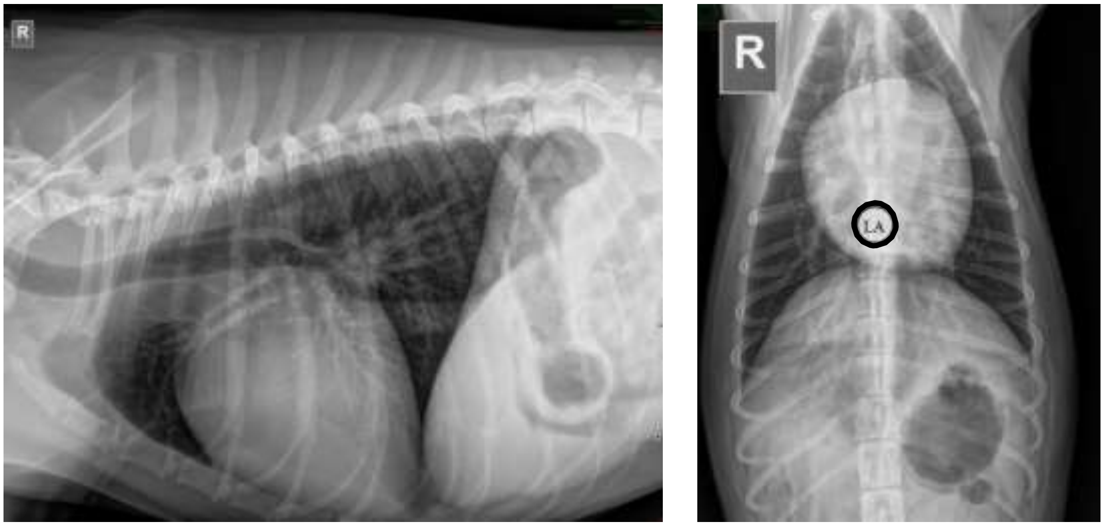

Figure #3: Right lateral and dorsoventral thoracic radiographs at 10 month recheck. Right lateral view demonstrates improvement in cardiac size to 3 ½ intercostal spaces and 66% height of thoracic cavity (within reference range). Dorsoventral view demonstrates reduction in size of left atrium (LA) compared to Figure #1.

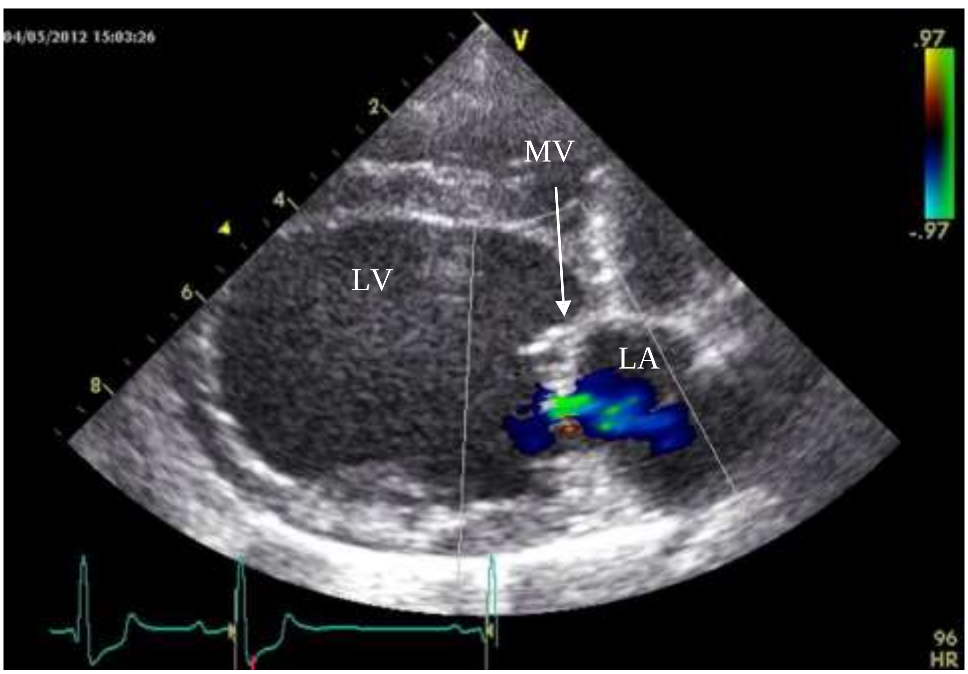

Figure #4: Two-dimensional echocardiogram long-axis view at 10 month recheck. This echocardiogram demonstrates an improved, yet enlarged left ventricle (LV) and normal sized left atrium (LA). The mitral valve leaflet (MV), remains mildly thickened. Color flow Doppler demonstrates significant improvement in mitral regurgitation compared to Figure #2.