Human & Veterinary Medicine
International Journal of the Bioflux Society HVM

OPEN ACCESS
Case Report

Nutritional dilated cardiomyopathy in cats – a report of two cases

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Abstract. Taurine deficiency in cats may induce cardiac dilatation and low systolic function of the left ventricle, leading to congestive heart failure. In this paper, we report two cases of taurine deficiency induced dilated cardiomyopathy in cats diagnosed by increased left atrial size and ventricular diameters in both systole and diastole, with low systolic function. Furthermore, this paper describes the reversal of the pathologic changes in the heart after cardiac pharmacotherapy and taurine supplementation.

Key Words: cardiac, feline, systolic dysfunction, taurine deficiency.

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Background

Before 1987, dilated cardiomyopahty (DCM) was the most commonly diagnosed cardiac disease in cats (Mac Donald 2016). Lately, it became an uncommon pathology, the incidence decreasing since the association between taurine-deficiency and DCM was recognized (Novotny et al 1994). Taurine deficiency induced myocardial failure is associated with low plasma, whole blood and tissue taurine concentrations. The underlying mechanism leading to clinical manifestations in cats with DCM is the systolic dysfunction induced by the low contractility of the dilated left ventricle (Mac Donald 2016). At present, DCM in cats is an uncommon finding due to the well-balanced feline diets. Taurine is an amino acid that is only found in animal products. In cat's foods, even with the inclusion of animal ingredients, taurine supplementation is usually required to achieve the minimum requirement for this amino acid. In the case of diets without animal products, taurine supplementation becomes mandatory (Zafalon et al 2020). Cats have limited hepatic production of taurine from amino-acids like methionine and cysteine, which results from the low activity of the enzyme cysteine dioxygenase. This low enzymatic activity limits the production of cysteine sulfate, an intermediate in taurine synthesis. Cats also have limited activity of cysteine sulfinic acid decarboxylase, an enzyme responsible for converting cysteine sulfate to taurine (Morris et al 1994). Conclusions that taurine deficiency results in DCM are based on clinical studies, demonstrating a strong relationship between low plasma taurine concentrations and echocardiographic findings (Novotny et al 1994). However, it has been proven that genetic involvement in development of feline DCM is also present, either by limitations in taurine absorption, metabolism or excretion (Lawler et al 1993). Yet, not all cats with taurine deficiency develop DCM (Novotny et al 1994).

Feline DCM had been described especially in the last two decades of the 20th century. Several studies have reported the prevalence of DCM between 3.4% and 6.29% of the total cardiac diseases in cats (Smith et al 2003; Schober and Maerz 2006; Cote and Jaeger 2008). Taurine-deficiency DCM is one of the few cardiac pathologies reversible once the diet is corrected. One study reported a clinical response and survival rate of 58% at one year after the therapy (Pion et al 1992). The aim of this study is to describe the clinical manifestations and paraclinic findings in two cases of nutritional-induced dilated cardiomyopathy in cats and the reversal of the pathologic changes in the heart after therapy and nutritional correction.

Case presentation

Case no. 1

An eight years old Siamese, female spayed cat, weighting 5.5 kg was presented to our Veterinary Teaching Hospital with signs of breathlessness and panting for 3 days. Owner disclosed that the cat was living with a dog and was eating only dog food for several years. Physical examination revealed normal body condition score (BCS) of 3/5, pink mucosal membranes, increased respiratory rate (75 bpm) with inspiratory effort and normal femoral pulse on both limbs. Chest tapping revealed mate sound and on auscultation, no respiratory sound was heard. Cardiac auscultation revealed the presence of a gallop sound with a heart rate of 215 bpm. Subsequently, the cat was subjected to a complete cardiologic examination. Arterial blood pressure was measured from the tail with an oscyllometric device (Vet HDO) with a C1 cuff and it was 166/78 mmHg (S/D).

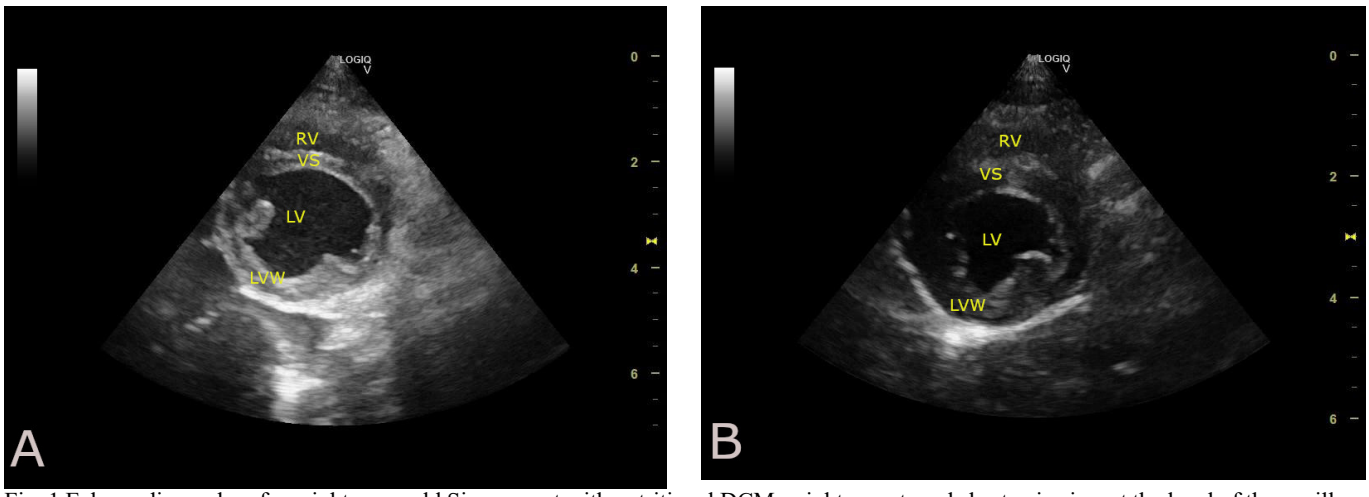

Fig. 1 Echocardiography of an eight years old Siamese cat with nutritional DCM – right parasternal short axis view at the level of the papillary muscles: A. at the first examination – notice the increase diameter of the left ventricle cavity in diastole and the thinning of the interventricular septum and the free wall; B. three months later – notice the reduced LV diameter in diastole and the thickening of the interventricular septum and the free wall; RV – right ventricle, VS – ventricular septum, LV – left ventricle, LVW – left ventricle free wall

Table 1: Echocardiographic measurement values on the first examination and three months later (second examination); normal values are given according to the literature.

Measurement	First examination	Second examination	Normal values
LAD (cm)	2.17	1.45	1.3-1.59 (Karsten et al 2017)
LA/Ao	2.27	1.21	1.06-1.23 (Haggstrom et al 2016)
IVSd (cm)	0.39	0.52	0.36-0.68 (Karsten et al 2017)
LVIDd (cm)	2.08	1.44	1.15-1.9 (Karsten et al 2017)
LVWd(cm)	0.52	0.52	0.36-0.56 (Karsten et al 2017)
IVSs(cm)	0.41	0.68	0.47-0.91 (Haggstrom et al 2016)
LVDs(cm)	1.87	0.82	0.6-1.37 (Haggstrom et al 2016)
LVWs(cm)	0.61	0.9	0.5-0.92 (Haggstrom et al 2016)
SF%	10.11	43	28-62 (Haggstrom et al 2016)
E-wave vel. (m/s)	0.76	0.84	0.6-0.8 (Chetboul et al 2006)
A-wave vel. (m/s)	0.5	N/A	0.4-0.6 (Chetboul et al 2006)
E/A	1.53	N/A	1.2-1.8 (Chetboul et al 2006)
Ao. Vel. (m/s)	0.73	1.13	0.9-1.3 (Chetboul et al 2006)
Pulm. Vel. (m/s)	N/A	0.78	0.7-1.1 (Chetboul et al 2006)
M. reg. vel. (m/s)	3.45	-	N/A

LAD - left atrial diameter in long axis; LA/Ao – left atrium to aorta ratio; IVSd – thickness of the interventricular septum in diastole; LVIDd – left ventricle internal diameter in diastole; LVWd – left ventricle free wall thickness in diastole; IVSs – thickness of the interventricular septum in systole; LVIDs – left ventricle internal diameter in systole; LVWs – left ventricle free wall thickness in systole; SF% - shortening fraction; E-wave vel. – peak velocity of the early transmitral diastolic wave; A-wave vel. - peak velocity of the active transmitral diastolic wave; E/A – E-wave to A-wave ratio; Ao. Vel. - peak velocity of the aortic systolic flow; Pulm. Vel. - peak velocity of the pulmonary systolic flow; M. reg. vel. - peak velocity of the mitral systolic regurgitating jet; N/A –not available

A five minutes six lead electrocardiography with the PolySpectrum 8E/X device was performed as previously described by the literature (Tilley and Smith Jr 2016). Electrocardiography revealed sinus rhythm, with a mean electrical axis of +900. No arrhythmias were present on the 5-minute long recording. The morphology was within normal limits: P wave amplitude – 0.06 mV; P-wave length – 46 msec; PQ interval – 70 msec; R-wave amplitude – 0.32 mV; QRS length – 47 msec; QT interval – 180 msec and T-wave amplitude – 0.08 mV. Biochemical analyses and complete blood count (CBC) were normal.

Echocardiographic examination was performed as previously described by Thomas et al. (1993) using Logiq V5 Expert, General Electric machine equipped with a phased array probe 4-8 MHz. Echocardiography revealed significant amount of pleural fluid. The left atrial cavity scaled to aortic root was above the normal reference ranges, along with the left ventricular diameter in both systole and diastole. A thinning of the inter-ventricular septum and left ventricular free wall were observed (Fig. 1A). Also, a low systolic function of the left ventricle expressed by a decreased shortening and ejection fraction was found.

A significantly hemodynamic regurgitating jet was present over the mitral valve. No smoke or clots were found in the left atrium. The values of the echocardiographic examination along with the normal reference ranges are presented in Table 1.

Based on the history and cardiologic examination, the suspected diagnosis was feline nutritional dilated cardiomyopathy and the following therapy was assigned: pimobendan (0.25 mg/kg BID), benazepril (0.5 mg/kg QD), clopidogrel (18.75 mg QD), furosemide (2 mg/kg BID) and taurine (250 mg BID) and a commercially available food for cats was recommended. Three months later the cat was scheduled for reexamination.

At recheck, the owners reported that the cat was feeling well, the respiratory distress disappeared and the patient was more active. On physical examination the BCS was 4/5, the respiratory rate was 28 bpm with normal breathing patter, pink mucosal membranes and normal femoral pulse on both limbs. On auscultation, the heart rate was 180 bpm and the gallop sound was absent. Pulmonary auscultation was unremakable. The blood pressure values were 166/78 mmHg (S/D).

Electrocardiography showed sinus rhythm, with a mean electrical axis of +600. No arrhythmias were found on a 5-minute recording trace and the morphology was within normal ranges. Echocardiography showed normal aspect of the left ventricle myocardial wall and normal size of the left atrium (Fig. 1B). The contractility was significantly improved and no pleural fluid was visible. No mitral regurgitating jet was present. A final diagnosis of nutritional dilated cardiomyopathy was established and the medication was withdrawn within one week after the second examination. Six months after the first presentation the owners reported by telephone that the cat was feeling well and no clinical signs were present.

Case no. 2

A sixteen years domestic shorthair (DSH) intact male cat weighting 3.8 kg was referred to the Cardiology service of the Veterinary Teaching Hospital with weakness, weight loss, dyspnea and tachypnea. The cat fell from the balcony 35 days ago and returned home 2 days prior to the medical investigations. On physical examination, the BCS was 2/5, the cat was tachypneic (a respiratory rate of 60 bpm), with increased respiratory effort, pink mucosal membranes, and the femoral pulse was normal on both limbs. Auscultation of the lungs revealed absence of the respiratory sounds on the lower part of the thorax and normal sounds over the upper half of the thorax. The cardiac auscultation revealed a gallop sound with a heart rate of 200 bpm. Electrocardiography showed sinus rhythm with a mean electrical axis of +600. No arrhythmias were present on the 5-minute recording. The morphology of the ECG was within normal limits: P wave amplitude – 0.15 mV, P-wave length – 47 msec, PQ interval – 70 msec, R-wave amplitude – 0.24 mV, QRS length – 46 msec, QT interval – 180 msec and T-wave amplitude – 0.05 mV. Echocardiography revealed moderate amount of pleural fluid. The cardiac chambers were enlarged, with thinning of the left ventricle walls (Fig. 2A).

The contractility of the left ventricle was at the lower limit. The left ventricle internal diameter in diastole was above the upper limit. Left atrium to aorta ratio was above the normal limit but not smoke or clots was found inside. The values of the echocardiographic examination are presented in Table 2.

Blood analyses were proposed but refused by the owners. The cat was released home with the following therapy: pimobendan (0.25 mg/kg BID), furosemide (2 mg/kg BID), clopidogrel (18.75 mg QD) and taurine (250 mg BID). Owners were instructed to feed the cat with commercially available food designed for cats only. One month later the cat was brought for recheck. Owners reported that the cat improved clinically and no respiratory distress was present. The BCS was 3/5, the body-weight was 4.1 kg, the mucosal membranes were pink, the respiratory rate was 36 bpm and the femoral pulse was normal on both limbs. On auscultation, the respiratory sounds were normal. A gallop sound was still present, and the heart rate was 200 bpm. The blood pressure was 161/87 mmHg (S/D). Electrocardiography revealed sinus rhythm, with a mean electrical axis of +600 and no arrhythmias were present over the 5 minutes recording. The morphology of

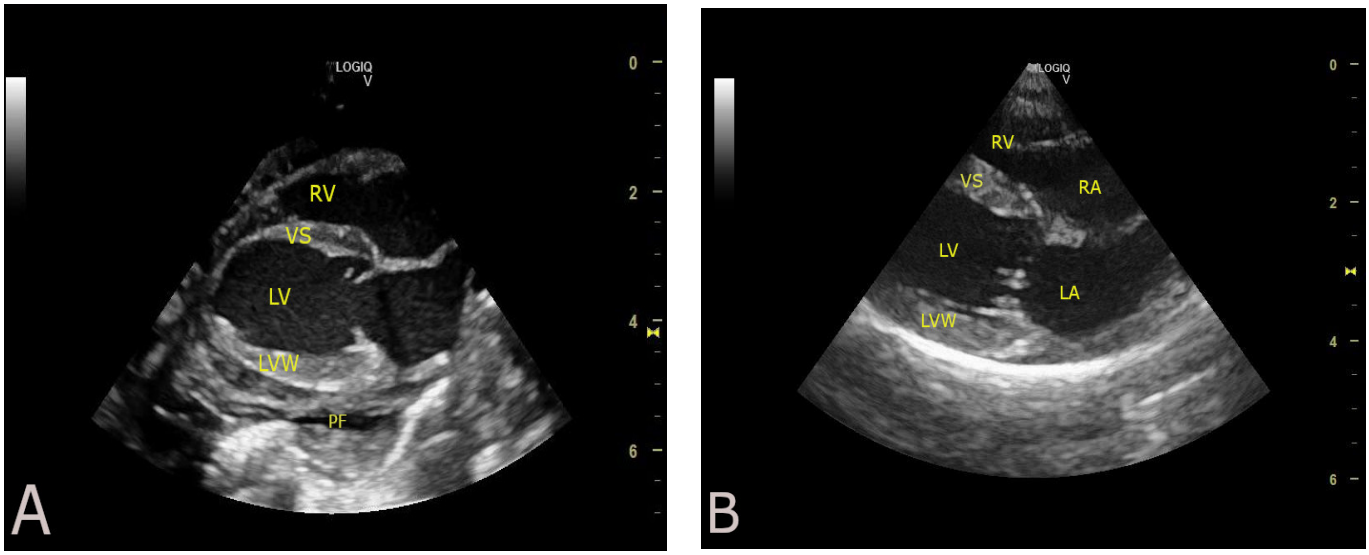

Fig. 2. Echocardiography of a sixteen years old DSH cat with nutritional DCM - right parasternal long axis four chamber view: A. at first examination – notice the general dilatation of the cardiac cavities during diastole with the thinning of the interventricular septum and the free wall. A small amount of pleural fluid is visible in this view posterior to the left ventricle free wall; B. one month after the first examination – notice the reduction in the diameters of the cardiac cavities in diastole and the thickening of the interventricular septum and left ventricular free wall; the pleural fluid is not visible in this view; RV – right ventricle, VS- ventricular septum, LV – left ventricle, LVW – left ventricle free wall, PF – pleural fluid

Table 2. Echocardiographic values form the first and second examination (one month later); normal values are given according to the literature

Measurement	First examination	Second examination	Normal values
LAD (cm)	2.09	1.52	1.3-1.59 (Karsten et al 2017)
LA/Ao	1.86	1.71	1.06-1.23 (Haggstrom et al 2016)
IVSd (cm)	0.37	0.46	0.35-0.64 (Karsten et al 2017)
LVIDd (cm)	2.03	1.77	1.09-1.8 (Karsten et al 2017)
LVWd(cm)	0.32	0.56	0.34-0.53 (Karsten et al 2017)
IVSs(cm)	0.32	0.7	0.43-0.84 (Haggstrom et al 2016)
LVDs(cm)	1.43	0.96	0.55-1.26 (Haggstrom et al 2016)
LVWs(cm)	0.41	0.74	0.46-0.85 (Haggstrom et al 2016)
SF%	29.5	45.5	28-62 (Haggstrom et al 2016)
E-wave vel. (m/s)	N/A	0.93	0.6-0.8 (Chetboul et al 2006)
Ao. vel. (m/s)	N/A	0.78	0.9-1.3 (Chetboul et al 2006)
Pulm. vel. (m/s)	N/A	0.75	0.7-1.1 (Chetboul et al 2006)

LAD - left atrial diameter in long axis; LA/Ao – left atrium to aorta ratio; IVSd – thickness of the interventricular septum in diastole; LVIDd – left ventricle internal diameter in diastole; LVWd – left ventricle free wall thickness in diastole; IVSs – thickness of the interventricular septum in systole; LVIDs – left ventricle internal diameter in systole; LVWs – left ventricle free wall thickness in systole; SF% - shortening fraction; E-wave vel. – peak velocity of the early transmitral diastolic wave; Ao. Vel. - peak velocity of the aortic systolic flow; Pulm. Vel. - peak velocity of the pulmonary systolic flow; M. reg.vel. - peak velocity of the mitral systolic regurgitating jet; N/A –not available

the ECG was within normal ranges. Echocardiography showed normal left ventricular diameter in diastole, but the left atrial cavity was still increased in size. The systolic function improved significantly and the inter-ventricular septum and left ventricle free wall were thicker (Fig. 2B). A small amount of free fluid was observed in the pleural cavity. A final diagnosis of nutritional dilated cardiomyopathy was established. Owners were instructed to continue the therapy only with furosemide dose decreased to 1 mg/kg BID. Two weeks later the owners reported by telephone that the cat was free of clinical signs and a recheck was scheduled.

Discussions

The cases of cats presented in this paper had a history of incorrect diet, one eating dog food and the other being missing outside for 35 days with unknown nutritional status. Based on the history and echocardiography, nutritional DCM was suspected in both cats. Following the positive response to taurine supplementation a final diagnosis of DCM was accepted. Interestingly, the taurine-deficiency period was significantly different (35 days vs. several years). To the author's best knowledge, there is no data in the literature specifying a minimum period for developing DCM secondary to taurine deficiency in cats. One study found that cats with taurine deficiency developed gallop sound and cardiac murmurs between five to eight months post low-taurine food administration and one cat developed cardiomegaly and pulmonary edema by six months (Novotny et al 1994). On the basis of echocardiographic findings, the most likely differential diagnoses were dilated cardiomyopathy and end-stage hypertrophic cardiomyopathy. However, a complete history may help orientating towards a correct diagnosis. A final diagnosis of nutritional DCM may be acquired only after the positive response and clinical improvement to taurine supplementation and nutritional correction. The therapeutic management is recommended for improving the hemodynamic status and should be withdrawn following the resilience of cardiac function.

Conclusions

This paper presents two cases of cardiac enlargement and systolic dysfunction associated with inappropriate diet in cats. Following the cardiac therapy for maintaining the hemodynamic status and nutritional correction, the cardiac dysfunction and morphological changes have been restored. Considering the positive response to therapy, a diagnosis of taurine-deficiency dilated cardiomyopathy was set. Clinicians should be aware of the nutritional deficiencies in cats and the possible association with dilated cardiomyopathy which is one of the few reversible cardiac diseases.

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Citation	Baisan RA, Turcu CA, Vulpe V. Nutritional dilated cardiomyopathy in cats – a report of two cases. HVM Bioflux 2020;12(1):28-32.
Editor	Antonia Macarie
Received	23 January 2020
Accepted	19 February 2020
Published Online	16 March 2020
Funding	None reported
Conflicts/ Competing Interests	None reported