Ankara Univ Vet Fak Derg, **68**, 301-306, 2021
DOI: 10.33988/auvfd.772831

Case Report / *Olgu Sunumu*

Non-taurine responsive dilated cardiomyopathy in 2 cats

Kadir SEVİM[1,a], Ekrem Çağatay ÇOLAKOĞLU[1,b,✉]

[1]Ankara University, Faculty of Veterinary Medicine, Department of Internal Medicine, Ankara, Turkey
[a]ORCID: 0000-0002-1959-1010; [b]ORCID: 0000-0003-2789-035X

[✉]Corresponding author: colakoglu@ankara.edu.tr
Received date: 23.07.2020 - Accepted date: 13.10.2020

Abstract: Feline dilated cardiomyopathy (DCM) is a rare disease characterized by myocardial failure and systolic dysfunction. Taurine deficiency is the most important cause of DCM phenotype in cats. A 2 year-old Scottish fold and a 8 month-old domestic shorthair cat were referred to hospital with anorexia, weakness and increased respiratory effort. Based on electrocardiographic, radiographic and echocardiographic findings, the both cases were diagnosed with DCM. The cases were managed with pimobendan, furosemide, acetylsalicylic acid and taurine. The cats were more active and clinically healthy during 15 days follow-up. No remarkable changes were observed in follow up echocardiographies. The cases presented here reflect the clinical signs, cardiological examination findings, diagnosis and management of idiopathic DCMin 2 cats.
Keywords: Cat, dilated cardiomyopathy, taurine.

İki kedide taurine yanıt vermeyen dilate kardiyomiyopati

Özet: Kedilerin dilate kardiyomiyopatisi (DCM) myokardiyal yetmezlik ve sistolik disfonksiyonla karakterize nadir bir hastalıktır. Taurin yetmezliği kedilerde dilate kardiyomiyopati fenotipinin en önemli nedenidir. İki yaşında Scottish Fold ırkı ve 8 aylık yaştaki Melez ırk kedi; anoreksi, halsizlik ve solunum güçlüğü şikayetleri ile hastaneye getirildi. Elektrokardiyografik, radyografik ve ekokardiyografik bulgular ile her iki olguda da dilate kardiyomiyopati teşhisi koyuldu. Olgular; pimobendan, furosemid, asetilsalisilik asit ve taurin ile sağaltıldı. Kediler on beş gün süresinceki takiplerde daha aktif ve klinik olarak sağlıklıydı. Kontrol ekokardiyografilerinde önemli bir değişiklik belirlenmedi. Burada sunulan olgular; idiyopatik dilate kardiyomiyopatili iki kedideki klinik bulgular, kardiyolojik muayene sonuçları, tanı ve tedaviyi yansıtmaktadır.
Anahtar sözcükler: Dilate kardiyomiyopati, kedi, taurin.

Feline dilated cardiomyopathy (DCM) is a disease characterized by primary myocardial failure. Severe enlarged left ventricle and hypocontractile myocardium are the main features of DCM phenotype in cats (8). It was proved in 1987 that taurine deficiency was the most important cause of DCM phenotype (17). After this scientific data, the incidence of the disease has decreased considerably by adding taurin to commercial cat food (3, 8, 13). However, DCM cases were also recognized in small number of cats feeding with an appropriate diet. Signs of respiratory distress, tachycardia, hypotension, collapse and lethargy indicating congestive heart failure and, thromboembolism may seen in cats with DCM (9, 13, 19). Gallop sounds and systolic murmur can also be predictable for DCM cats (13). Increased left ventricle internal dimension at end-systole and reduced fractional shortening as typical echocardiographic features of cats with DCM (8, 9, 13). The cases presented here reflect the process of clinical signs, cardiological examination findings, diagnosis and management of non-taurine responsive DCM in 2 cats.

A 2-year-old intact female Scottish Fold cat feeding with commercially available food referred to Small Animal Veterinary Teaching Hospital with complaints of anorexia and weakness. Mucosal pallor, normothermia (38^0 C) and increased capillary refill time (>3s) with increased respiratory effort were noticed in physical examination. The cat was tachycardic (Heart rate > 250 bpm). Femoral arterial pulses were slightly weak. The cat had also bilaterally auscultated systolic heart murmurs and precordial thrill. Routine complete blood count, serum profiles and serum cardiac troponin-I concentration were showed in Table 1. Thoracic radiographs indicated severe cardiomegaly with pulmonary edema (Figure 1). 6-lead

clinic electrocardiography displayed sinus tachycardia and ST-segment elevation (Figure 2). Echocardiography revealed thinning of interventricular septum and left ventricular free wall and, regurgitant jet detected over the mitral and tricuspid valve with doppler-detected pressure gradients (Figure 3, Table 2). Spontaneous echogenic contrast (smoke) was also visible in dilated left atrium and left ventricle (Figure 3). A definitive diagnosis of DCM was made based on the history, examination findings and diagnostic applications and the following medications assigned as: pimobendan (0.2 mg/kg BID, PO), furosemide (2 mg/kg BID, PO), acetylsalicylic acid (25 mg/cat q72h PO) and taurine (250 mg, BID, PO). The owner reported that the cat was more active and were able to breath easily during 15 days follow-up. No remarkable changes observed in follow up echocardiographies.

Table 1. Blood analyses in cases.

CBC	Results (Case 1)	Results (Case 1)	Reference ranges	Serum Profiles	Results (Case 1)	Results (Case 2)	Reference ranges
WBC (10^9/l)	15	14.8	5.5-19.5	**Glucose** (g/dl)	87	109	70-110
LYM (10^9/l)	2.4	1.9	1.1-7	**Urea** (mg/dl)	42.9	49.1	42.8-64.2
MON (10^9/l)	0.9	0.8	0.2-1.5	**Creatinin** (mg/dl)	0.9	0.86	0.8-1.8
NEUT (10^9/l)	11.2	12	2.8-13	**T. Protein** (g/dl)	5.8	6.8	5.4-7.8
EOS (10^9/l)	0.5	0.1	0.1-2.5	**Albumin** (g/dl)	2.9	3.1	2.4-3.8
RBC (10^{12}/l)	9.12	8.5	5-11	**T. Bilirubin** (mg/dl)	0.1	0.13	0.1-0.2
HGB (g/dl)	13.7	11	8-15	**ALP** (IU/L)	46	57	<70
HCT (%)	33.2	29	25-45	**ALT** (IU/L)	25	35	<50
MCV(fl)	36.3	48.4	39-50	**AST** (IU/L)	23	21	<40
MCH (pg)	15.1	13.2	12.5-17.5	**GGT**(IU/L)	7	8	6-28
MCHC (g/dl)	37.5	35.3	31-38.5	**Na** (mmol/l)	149	151	147-156
RDW(%)	14.8	14.9	14-18.5	**K** (mmol/l)	3.8	3.9	3.6-5.6
PLT (10^9/l)	250	350	200-500	**CK** (IU/L)	323	296	<200
MPV(fl)	8.8	8.2	8-12	**tT4** (µg/dl)	2.3	3.8	0.8-4.7
				cTnI (ng/ml)	0.1	-	0.03-0.1 (2)

Table 2. Echocardiographic measurements in cases.

Variables	Case 1	Case 2
IVSd (mm)	6.9	2.51
LVIDd (mm)*	22.5	27
LVWd (mm)	6.9	5
IVSs (mm)	8.1	3.04
LVIDs (mm)*	11.3	13.8
LVWs (mm)	6.6	5.9
LA (mm)	22.2	26.9
Ao (mm)	12.2	7.72
LA/Ao (mm)	1.81	3.48
FS (%)	14.3	28.9
EF (%)	40.7	58.7
High MV E:A Ratio	Restrictive pattern	Restrictive pattern
MR Jet velocity	6.85 m/sec	6.70 m/sec
MR Max PG	187,69 mmHg	179,56 mmHg
Aortic flow velocity	1.75 m/sec	1.45 m/sec

IVSd, interventricular septum diameter during diastole; LVIDd, left ventricular internal dimension during diastole; LVWd, left ventricular wall diameter during diastole; IVSs, interventricular septum diameter during systole; LVIDs, left ventricular internal dimension during systole; LVWs, left ventricular wall diameter during systole (M mode Teich, 2D right parasternal short-axis view); LA, left atrial diameter; Ao, aortic diameter (2D right parasternal short-axis of the heart base); MV, mitral valve; MR, Mitral regurgitation.

* DCM diagnosis based on the measurements of LVIDd > 16 mm and LVIDs > 11 mm (7).

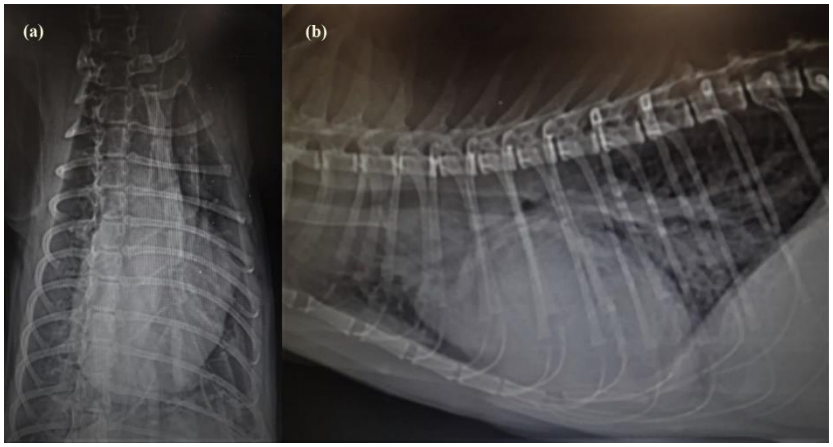

Figure 1. Cardiomegaly in ventrodorsal view (a). Marked pulmonary edema on caudal lung fields. Dorsoventral view (b).

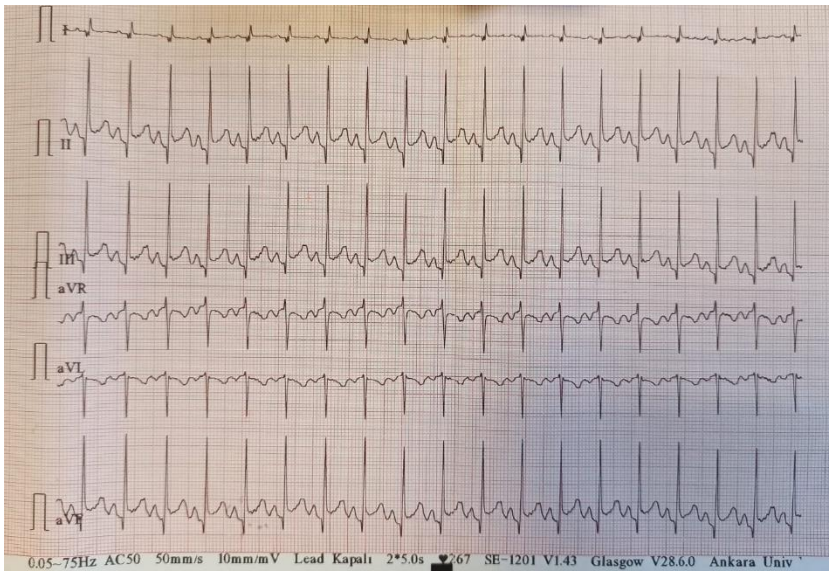

Figure 2. Sinus tachycardia with interatrial conduction delay (P mitrale and P pulmonale) indicating biatrial dilatation.
P wave amplitude 0.3 mV (reference range < 0.2 mV) and P duration 64 ms (reference range < 35ms). Increased R wave amplitude and QRS duration revealed left ventricular enlargement. QRS amplitude 2.2 mV (reference range < 0.9 mV), QRS duration 60 ms (reference range < 40 ms). ST-segment elevation associated with hypoxia is also remarkable in all leads.

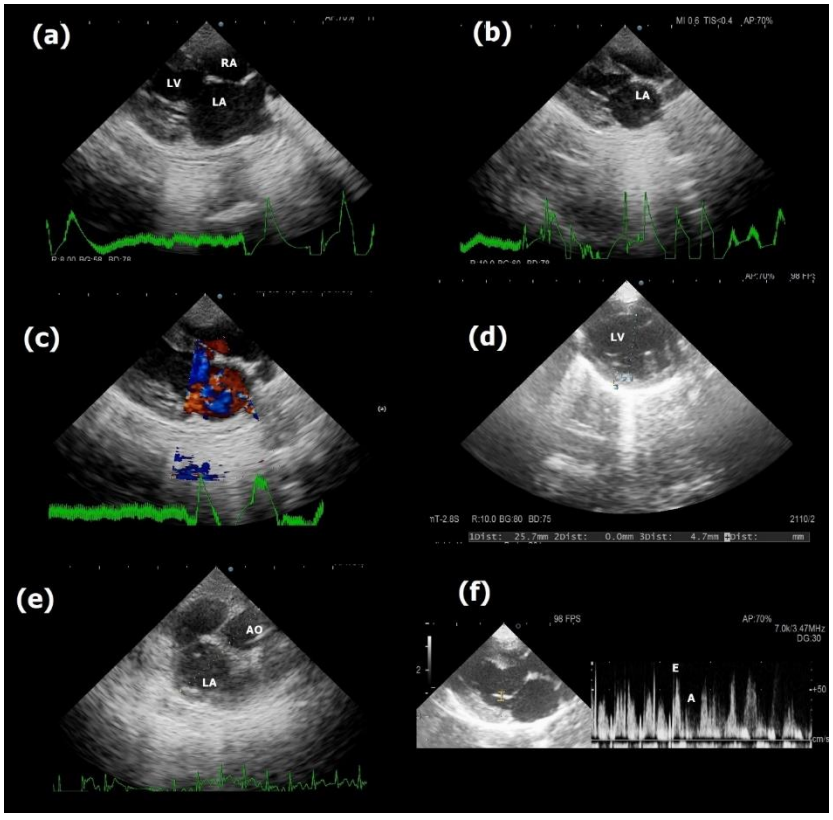

Figure 3. Marked LA dilatation compared to RA and, pulmonary vein dilatation.
Visible smoke in LA. Right PLAX (a,b). Mitral regurgitant jet detected over mitral valve leaflets. Right PLAX (c). Thinning of interventricular septum and left ventricular free wall. Right PSAX papillary muscle level (d). LA (22.2 mm)/AO (12.8) rate: 1.73. Right PSAX heart base (e). Restrictive pattern PW E > A (f). LA: Left atrium, LV: Left ventricle, RA: Right atrium, AO: Aort, PLAX: Parasternal long axis view, PSAX: Parasternal short axis view.

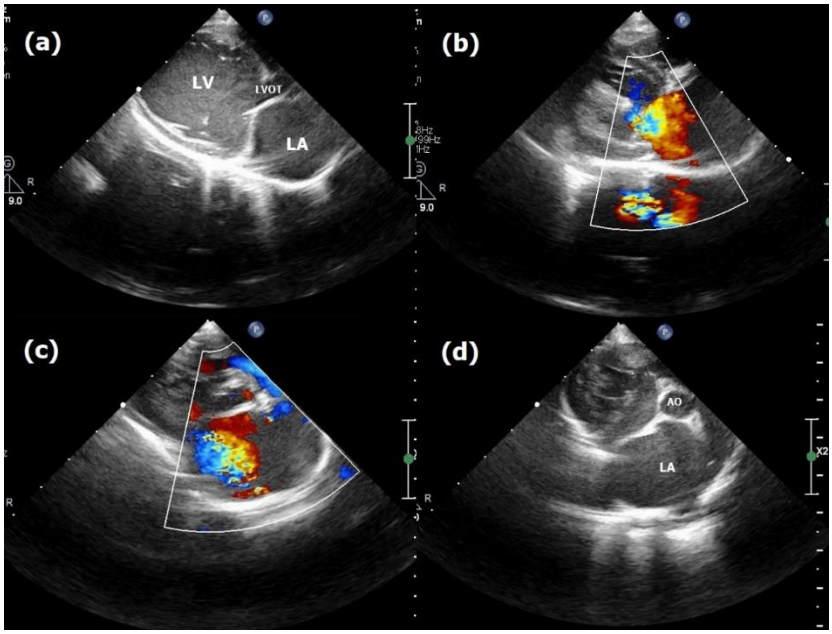

Figure 4. Severe LA and LV dilatation with thinning of interventricular septum and left ventricular free wall.
Visible smoke in LA and LV. Right PLAX (a). Mitral regurgitant jet over mitral valve. Right PLAX (b,c). Increased LA/Ao rate. Right PSAX heart base (d). LA: Left atrium, LV: Left ventricle, AO: Aort, LVOT: Left ventricle outflow tract, PLAX: Parasternal long axis view, PSAX: Parasternal short axis view.

A 8-month-old domestic shorthair cat referred to hospital with anorexia and intermittent open-mouth breathing. Physical examination revealed normothermia (38.3 ^{0}C), hypokinetic femoral pulses, tachycardia (> 240 bpm), gallop rhythm with precordial thrill and increased respiratory effort. Routine complete blood count and serum profile analysis were also performed as showed in Table 1. Thoracic radiographs showed cardiomegaly with increased sternal contact surface. Echocardiography confirmed DCM with smoke formation (Figure 4, Table 2). The cat was initiated the following medications: pimobendan (0.2 mg/kg BID, PO), furosemide (1 mg/kg BID, PO), acetylsalicylic acid (25 mg/cat q72h PO), taurine supplementation (250 mg, BID, PO) and oxygen support. The cat was alive and clinically active during 15 days follow up. No remarkable changes observed in follow up echocardiographies.

DCM phenotype is a myocardial disorder characterized by enlargement of the left ventricular lumen and decreased systolic myocardial function. It was the most common myocardial disease in cats until its relationship with taurine deficiency was determined (17). With the addition of taurine to commercial foods, the incidence of the disease decreased in cats (7, 15). Nevertheless, cats with DCM are encountered although they are fed with a balanced diet. Since the factors causing myocardial dysfunction are often undetectable, non-taurine responsive feline DCM is referred to as idiopathic (13). DCM phenotype can also be possible in the end stage valvular diseases (7). In cases presented here, there is no evidence of pre-existing heart murmur or echocardiographic evidence the valvular pathology to suggest the valvular insufficiency resulting in DCM phenotype or systolic dysfunction. It has been also reported the possibility of infectious etiology and genetic predisposition in occurrence of feline DCM (14, 16). The increase in thyroid hormone levels causes an increase in heart rate and a stronger contraction of the heart muscle. Untreated or unmanaged cases of hyperthyroidism, heart functions deteriorate over time and heart failure may occur (11). In cases here, free thyroxine levels were also within reference ranges. Sustained tachycardia and tachyarrhythmias can also damage myocardial function, causing secondary AV insufficiency or heart failure. This pathological sequela is called tachycardia-induced cardiomyopathy. Arrhythmias can occur due to myocardial diseases, such as DCM or vice versa. Sustained tachycardia can cause DCM (10, 12). It is difficult to distinguish between these two situations. Greet et al. (12) have set criteria for diagnosing supraventricular tachycardia including narrow complex tachycardia with a regular R-R interval and a HR value of more than 260 bpm in the ECG recording, a sudden onset or exit from a narrow complex tachycardia, presence of a permanent atrial depolarization at a rate greater than 260 bpm and the determination of a wide complex tachycardia suggesting intraventricular conduction disturbance. No evidence of supraventricular tachycardia was observed in electrocardiographic evaluations. Congestive signs of respiratory distress, tachycardia, hypotension and collapse and, lethargy and thromboembolism may be seen in cats with DCM (9, 13, 19). Gallop sounds and systolic murmur can also accompany to DCM phenotype in cats (7, 15). In the cases here, congestive signs, gallop rhythm with various degrees of systolic murmur were remarkable as well. Although ECGs are nonspecific in cats with DCM,

sinus tachycardia, atrial or ventricular premature beats, ventricular tachyarrhythmias and atrial fibrillation could be present (5-7, 9, 13). In the first case with ECG, the findings were consistent with the reports previously described. Aroch et al. (4) reported that creatine kinase is a reliable marker of myocardial damage in cats. In accordance with this view, the increase in creatine kinase levels in the case confirms myocardial damage. Cardiac troponins are used as the gold standard in humans to determine myocardial damage (12). Wells et al. (20) reported that reference ranges in healthy cats were 0-0.17 ng / ml in their study of cardiac troponin-I. The absence of any study on cardiac troponin levels in cats with DCM suggests that further studies are needed to determine the diagnostic and prognostic significance of the data we obtain. As DCM progresses, thinning of the atrioventricular area causes dynamic valve insufficiency (9). Mitral insufficiency in echocardiography is the result of this condition. Diagnosis can be made on echocardiography when an enlargement of the left ventricle diameter (> 11 mm in systole and or > 16 mm in diastole) (7). The data we obtained are compatible with this information. In cases with DCM, reduction of fractional shortening (FS <20%) is an expected result (7). In these cases, the differences of FS can be explained by the fact that fractional shortening is affected by hemodynamic status (15). Decreasing myocardial contractility may cause increased fractional shortening. Since it was known that the cat was feeding with a balanced diet, that the limitations of the test were used to identify taurine deficiency and that the response to taurine appears the most reliable method in deficiency, taurine supplementation recommended to the owners regardless of the whole blood concentration (1, 18). The prognosis of idiopathic DCM is poor. In one study, the median survival time was reported to be 11 days (9) while in another one it was 49 days in cats treated with pimobendan (13). Cases presented here remains alive at 1 month following the diagnosis.

In conclusion the cases presented here reflect clinical signs, cardiological examination findings, diagnosis and management of idiopathic dilated cardiomyopathy in 2 cats. We hope that this study will contribute to determining the feline idiopathic dilated cardiomyopathy and help veterinary practitioners.

Financial Support

This research received no grant from any funding agency/sector.

Ethical Statement

This study does not present any ethical concerns.

Conflict of Interest

The authors declared that there is no conflict of interest.

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