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Dilated cardiomyopathy in a cat: Case report

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Abstract
Feline dilated cardiomyopathy (DCM) is a rare disease characterized by dilation of the left ventricle lumen and decreased systolic myocardial function. The primary cause of DCM in cats was found to be taurine deficiency as a result of low taurine levels in conventional pet diets. A 10-year-old Domestic Short Hair cat was referred to the Department of Veterinary Clinical Medicine, Ethics and Jurisprudence with history of anorexia, dyspnea, open mouth breathing, and lethargy. The case was diagnosed with DCM and congestive heart failure based on the results of the radiography and echocardiographic examinations. This case report illustrates the clinical symptoms, cardiac evaluation, and DCM diagnosis in a cat.

Keywords: Cat, dilated cardiomopathy, echocardiography, taurine

Introduction
Feline dilated cardimyopathy (DCM) is an adult onset primary myocardial disease characterized by a substantially dilated left ventricle chamber, reduced systolic myocardial function and hypocontractile myocardium (Ferasin, 2012) [4]. Until it was discovered that many cats had taurine deficiency due to low amounts of taurine in conventional pet food, DCM was thought to be the most frequent type of feline cardiac disease (Pion *et al.*, 1987) [9]. Based on this revelation, food manufacturers improved the amount of taurine in commercial feline diets, which significantly decreased the risk of taurine deficiency in the years that followed. Dilated cardiomyopathy is still seen in a tiny proportion of cats consuming a proper diet, while the cause of these occurrences is unknown. DCM is adult onset disease with an average reported age in one study of 9 years of age (Ferasin *et al.*, 2003) [5]. Presenting complaints include those consistent with heart failure, including dyspnea, tachypnea, and anorexia. Thromboembolism episodes may occur. Soft heart murmur and gallop sounds can be predictable for DCM (Sisson *et al.*, 1991) [10]. Radiographs may show signs of heart disease but they cannot diagnose a particular type of cardiomyopathy. Evidence of left or biventricular heart failure may be observed including pulmonary oedema or pleural effusion. Increased left ventricle internal dimension at end-systole and reduced fractional shortening are unique features of cats with DCM (Ferasin, 2012) [4]. This case illustrates the clinical indicators, cardiac evaluation, and DCM diagnosis in a cat.

Materials and Methods
A 10-year-old neutered female Domestic Short Hair cat named *Khajuri* was presented with the complaints of anorexia, dyspnea, open mouth breathing, and lethargy. Slight mucosal pallor, normothermia (99.8 ºF) and increased capillary refill time (>3 seconds) with respiratory distress were noted in physical examination. Auscultation revealed muffled lung sounds and a soft systolic murmur over the left apex. Femoral arterial pulses were slightly weak.

Results and Discussion
Tables 1 and 2 demonstrate the standard complete blood count, serum biochemistry profile, and serum cardiac troponin I concentration. Radiographs of the thorax revealed cardiomegaly and pleural effusion (Figure 1 and 2).

The left ventricular free wall and interventricular septum were thinned, and regurgitant jets were seen across the mitral valve with Doppler-detected pressure gradients (Figure 3 to 10, Table 3). Based on the history, physical examination results, and diagnostic modalities, an unambiguous diagnosis of DCM was made.

Table 1: Haematological analyses in the case

Parameter	Results	Reference range
Hb (g %)	15.9	9.5-15
PCV (%)	44.5	29-45
TEC (10^6/cmm)	9.7	6-10
TLC (10^3/cmm)	11.4	5.5-19.5
Neutrophils (%)	47	35-75
Eosinophils (%)	2	20-55
Lymphocytes (%)	50	1-4
Monocytes (%)	1	2-12
Basophils (%)	0	0-0
Platelets (10^5/cmm)	2.54	1.5-6.0

Table 2: Serum biochemistry profile in the case

Parameter	Results	Reference range
Total Bilirubin (mg/dl)	0.3	0-0.4
Direct Bilirubin (mg/dl)	0.2	0-0.1
Indirect Bilirubin (mg/dl)	0.1	0-0.3
Alanine Transaminase (ALT, IU/L)	54	28-76
Aspartate Transaminase (AST, IU/L)	58	5-55
Alkaline Phosphatase (ALP, IU/L)	41	0-62
Total Protein (gm/dl)	5.1	5.9-8.5
Albumin (gm/dl)	3.2	2.4-4.1
Globulin (gm/dl)	1.9	3.4-5.2
Blood Urea Nitrogen (mg/dl)	54.1	15-34
Creatinine (mg/dl)	3	0.8-2.3
Sodium (mEq/L)	148	147-156
Potassium (mEq/L)	4	3.9-5.3
Chloride (mEq/L)	113	111-125
Cardiac Troponin I (ng/ml)	0.98	<0.03-0.16

Table 3: Echocardiographic measurements in the case

Parameter	Results
RVIDd (mm)	2.57
IVSd (mm)	2.14
IVSs (mm)	2.57
LVIDd (mm)*	22.1
LVIDs (mm)*	20.24
LVPWd (mm)	1.71
LVPWs (mm)	2.71
EF (%)	19.89
FS (%)*	8.39
EPSS (mm)	10.12
LA (cm)	1.74
AO (cm)	0.64
LA/AO	2.71
Aortic flow velocity (m/s)	0.20
MV E/A ratio	2.20 (Restrictive pattern)
MR jet velocity (m/s)	2.52
MR Max PG (mmHg)	25.45
TV E/A ratio	2.34 (Restrictive pattern)

RVIDd, Right ventricular internal diameter at diastole; IVSd, Interventricular septum diameter at diastole; IVSs, Interventricular septum diameter at systole; LVIDd, Left ventricle internal diameter at diastole; LVIDs, Left ventricle internal diameter at systole; LVPWd, Left ventricular posterior wall at diastole; LVPWs, Left ventricular posterior wall at systole; EF, Ejection fraction; FS, Fractional shortening; EPSS, E point to septal separation; LA, Left atrial diameter; Ao, Aortic root diameter; MV, Mitral valve; MR, Mitral regurgitation; TV, Tricuspid valve.

*DCM diagnosis based on the measurements of LVIDd > 16 mm, LVIDs > 11 mm, and systolic function is decreased with a left ventricular fractional shortening of < 20% (Cote *et al.*, 2011) [1].

Fig 1: Left lateral radiograph of cat suffering from DCM with elevated VHS and pleural effusion

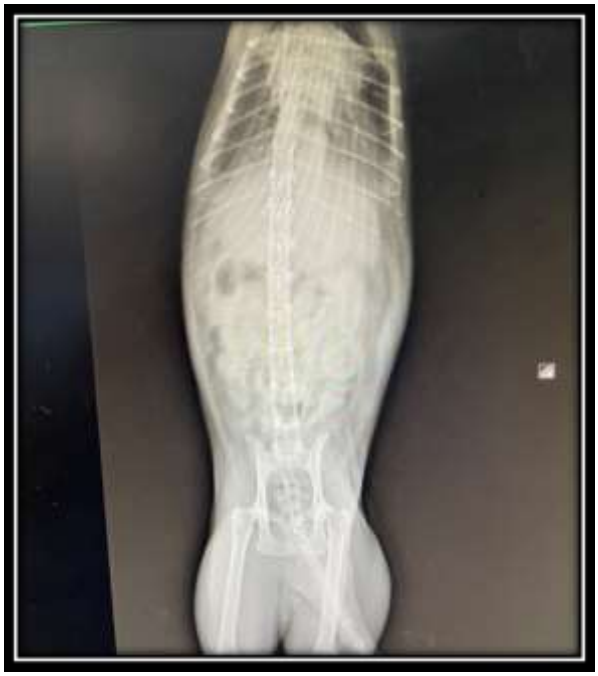

Fig 2: Ventrodorsal radiograph of cat suffering from DCM

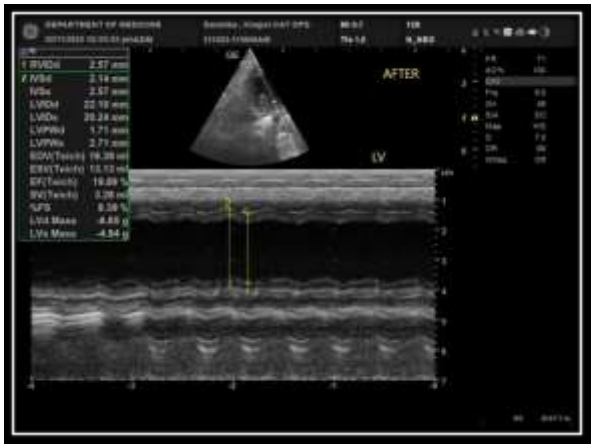

Fig 3 M-mode right PSAX view at papillary muscle level in cat with DCM indicating thinning of interventricular septum and left ventricular free wall

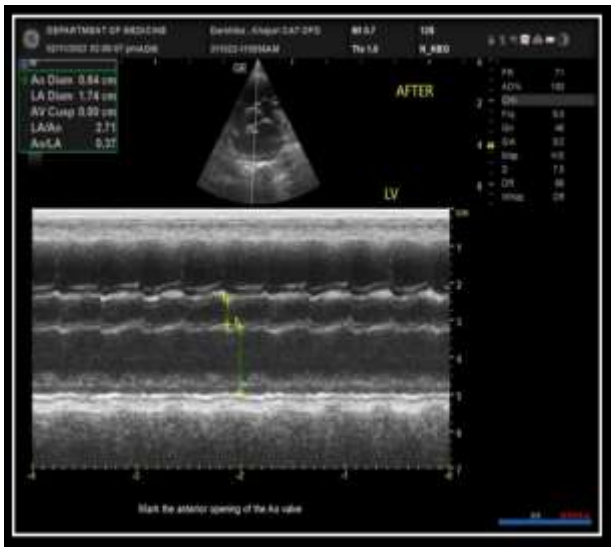

Fig 4: M-mode right PSAX view at the level of aortic root in cat with DCM indicating severe left atrial dilation with elevated LA/Ao

Fig 5: M-mode right PSAX view at the level of mitral valve showing increased value of E point to septal separation in cat with DCM

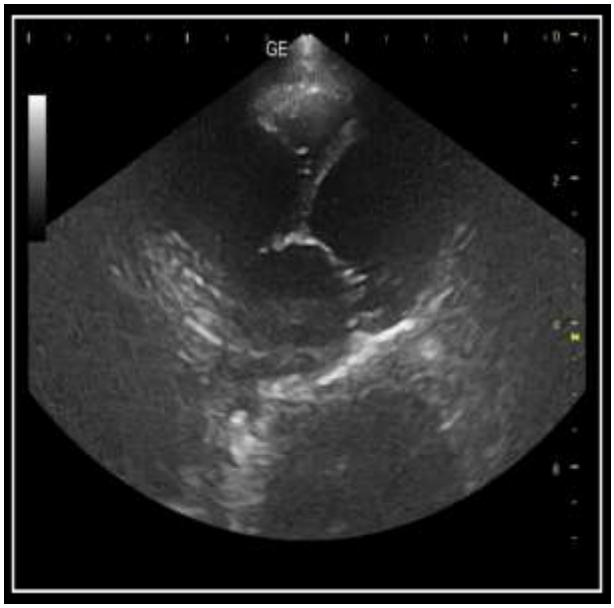

Fig 6: Right PLAX view in cat with DCM showing thinning of interventricular septum and free wall, knobbing of mitral valve and left atrial dilation

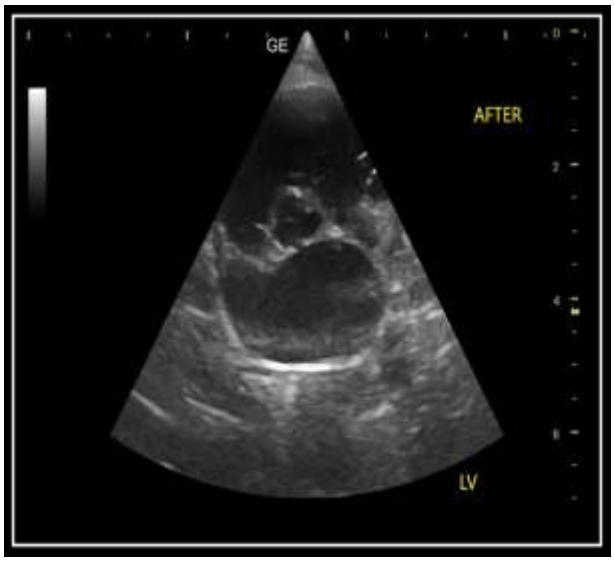

Fig 7: Right PSAX view in cat with DCM at the level of aortic root showing severe left atrial dilation

Fig 8: Left apical 4 chambered view in cat with DCM showing mitral regurgitant jet over mitral valve

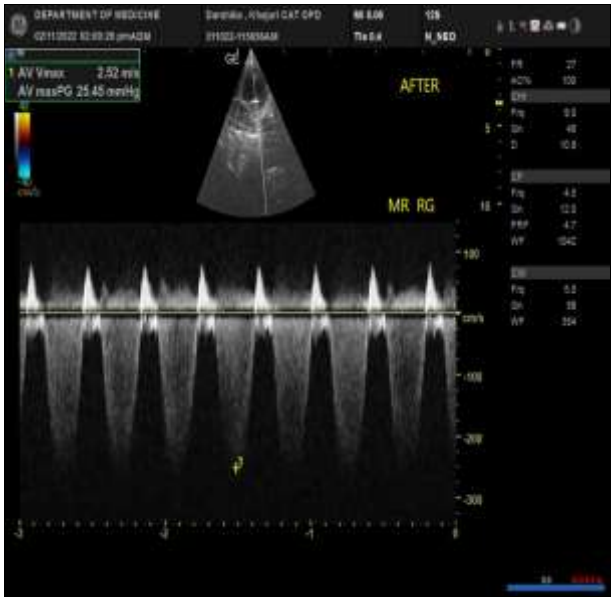

Fig 8 Left apical 4 chambered view showing mitral regurgitant jet velocity and pressure gradient in cat with DCM

Fig 9: Mitral inflow profile in cat with DCM showing restrictive filling pattern

Fig 10: Aortic velocity in cat with DCM

Discussion

DCM is a myocardial condition characterised by expansion of the left ventricular lumen and diminished systolic myocardial performance. Prior to the discovery of its connection to taurine insufficiency, it was the most prevalent cardiac disease in cats (Pion *et al.*, 1987) [9]. The prevalence of the condition declined in cats because to the inclusion of taurine in commercial meals (Madron, 2015) [7]. However, despite being provided a healthy diet, cats with DCM continue to be observed.

DCM is additionally conceivable in advanced valvular disorders (Cote *et al.*, 2011) [1]. Moreover, it has been suggested that feline DCM may have an infectious origin and genetic propensity (Lawler *et al.*, 1993; Meurs *et al.*, 2000) [6]. Cats with DCM may exhibit congestive signs of respiratory distress, open mouth breathing, lethargy, and thromboembolism (Ferasin*et al.*, 2003; Smith *et al.*, 2003) [5, 11]. Gallop sounds and systolic murmur can also accompany to DCM in cats (Cote *et al.*, 2011) [1]. In the situation that is being discussed here, congestive symptoms, a systolic murmur, and muffled lung sounds were also noteworthy.In order to assess myocardial damage in humans cardiac troponins are utilised as the gold standard (Greet *et al.*, 2020) [2]. Hemdon *et al.* (2002) [3] reported that the reference range of cardiac troponin I in healthy cats were <0.03-0.16 ng/ml. Further research is necessary to understand the diagnostic and prognostic implications of the data we collected because there hasn't been any research on the levels of cardiac troponin I in cats with DCM. As DCM progresses, thinning of the atrioventricular area causes dynamic valvular insufficiency which results in mitral insufficiency on echocardiography (Ferasin *et al.*, 2003) [5]. Diagnosis can be made on echocardiography when an enlargement of the left ventricle diameter (> 11 mm in systole and or > 16 mm in diastole and fractional shortening < 20%) (Cote *et al.*, 2011) [1]. This information is consistent with the data that we have collected. The prognosis of DCM is poor. In one study, the median survival time was reported to be 11 days (Ferasin *et al.*, 2003) [5]. The patient in this case survived for three days after the diagnosis.

Conflict of Interest

The authors declare that there is no conflict of interest.

Conclusion

In conclusion, the case described here reflects the clinical symptoms, the results of cardiac examination, and the diagnosis of feline dilated cardiomyopathy. We anticipate that this research will aid practitioners in understanding feline dilated cardiomyopathy.

Acknowledgments

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