



Imaging Diagnosis of Dilated Cardiomyopathy in a Maltese Dog

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Abstract A 6-year-old, spayed female, Maltese dog with tachypnea and dry cough was presented to Gyeongsang National University Veterinary Medical Teaching hospital. On physical examination, its respiration rate was 132 per minute. Decreased partial pressure of oxygen, partial pressure of carbon dioxide, and hyperlactatemia were found on arterial blood gas analysis. Its diastolic blood pressure was 80 mmHg. Auscultation revealed arrhythmia. Electrocardiogram revealed P pulmonale, P mitrale, and ventricular premature complexes. Thoracic radiographs revealed mild enlargement of both atrium and moderate enlargement of the left ventricular. There was also a moderate alveolar pattern in the right and caudal part of the left cranial lung lobe. Two-dimensional echocardiography showed enlargement of generalized four chambers without remarkable findings of valvular degeneration. M-mode echocardiography showed decreased left ventricular fractional shortening and enlarged left ventricular internal diameter at both end-systolic and end-diastolic. Color-flow Doppler imaging revealed eccentric turbulent flow starting below the left ventricular outflow tract and extending into the left atrium during systole. Spectral Doppler recordings revealed a high velocity flow through the mitral, tricuspid, aorta, and pulmonic regurgitation. Restrictive transmitral flow revealed high E-wave velocity, short E-wave deceleration time, and reduced A-wave velocity. There was also low ejection velocity thorough left ventricular out tract flow. Based on echocardiographic examination, dilated cardiomyopathy was the tentative diagnosis. The dog was medicated with inotropes, angiotensin converting enzyme inhibitor, and diuretics. At the 10-day following-up, the dog died suddenly. This report describes echocardiographic diagnosis and prognosis of dilated cardiomyopathy rarely reported in small breed dogs.

Key words dilated cardiomyopathy, echocardiography, small breed, dog.

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Introduction

Dilated cardiomyopathy (DCM) is characterized by impaired contraction and dilation of both ventricles or the left ventricle (19). It is the most common myocardial disease in dogs (5,19,17,18,26). Causes of DCM in humans are mostly genetic factors. Myocardial ischemia, hypertension, toxins, infections, and metabolic disorders can also be causes of DCM (1,3,9,15,22,24). Feline DCM is not common. It is usually caused by taurine deficiency (9,17,18,26). Primary DCM in dogs is an idiopathic myocardial disease. It is not related to any secondary disorder such as taurine or L-carnitine deficiency (7,9,17,18,22,26). The diagnosis of DCM requires an active exclusion of other congenital diseases, acquired cardiac diseases, systemic disease, and pulmonary diseases that might secondarily induce a similar phenotype (6).

Although electrocardiogram (ECG) is essential for the evaluation of arrhythmias, the presence of ECG abnormalities has limited value in the diagnosis of DCM (8). Findings on thoracic radiography for DCM include cardiomegaly, often with enlargement of the left atrium, various degrees of pulmonary edema, venous congestion, and sometimes ascites and pleural effusion (8). However, these findings are not specific for DCM (8). Echocardiography is the standard imaging technique for the diagnosis of DCM (8). Diagnosing the presence of specific chamber enlargement and myocardial failure requires M-mode and two-dimensional (2D) echocardiography (8). Echocardiographic findings include increased left ventricular end-diastolic dimension, end-systolic dimension, increased E-point septal separation (EPSS), and decreased fractional shortening (FS) (8). It has been reported that echocardiography is useful for prognostic evaluation of DCM in dogs (4). The restrictive transmitral flow (TMF) pattern correlates well with high filling pressure and poor prognosis of dogs with DCM (4).

DCM usually affects large breed of dogs. It rarely affects dogs weighing less than 12-15 kilograms (5,17,26). The purpose of this report was to describe radiographic findings, echocardiographic features, and prognosis of a small breed of Maltese dog with DCM.

Case Report

A 6-year-old spayed female Maltese dog weighting 4.95 kg with dry cough and tachypnea was referred to Gyeongsang National University Veterinary Medical Teaching Hospital. She had dry cough for one month and severe tachypnea for the previous two days. In physical examination, auscultation revealed arrhythmia. Its respiratory rate was 132 per min and its diastolic blood pressure was 80 mmHg. Electrocardiogram revealed tall and wide P wave that described atriums enlargement. Ventricular premature complexes were detected (Fig. 1). Complete blood cell count showed mild elevation of neutrophil and low levels of tT4 and fT4. Arterial Blood Gas Analysis revealed a low partial pressure of oxygen and a partial pressure of carbon dioxide.

Thoracic radiographs revealed moderate enlargement of the left ventricle, mild bulging of atrium, and moderate alveolar pattern in the right lung lobe and the cranial part of left caudal lung lobe (Fig. 2). Two-dimensional echocardiography showed a severely dilated left-side heart and a mildly enlarged right-side heart. The ratio of LA: Ao was 2.45 (reference range: <1.3). Color-flow Doppler echocardiogram showed eccentric mitral regurgitation during systole (Fig. 3). Transventricular time-motion echocardiography showed an enlarged left ventricular wall during both diastole and systole (LVIDd: 42 mm, reference range: 20-30 mm; LVIDs: 34 mm, reference range: 11-21 mm). FS (19.9%) was lower than the normal range (30-50%). Spectral Doppler recordings revealed a high mitral regurgitation velocity (5.96 m/s). Early diastolic mitral inflow velocity (0.88 m/s) was increased with an E:A ratio of 5.5. Reduced A-wave velocity (0.16 m/s) and short E-wave deceleration time (48 ms) were also identified. Tissue Doppler imaging (TDI) in LVW showed reversal E'A' and E peak velocity (5.7 cm/s) (Fig. 4). TDI showed no reversal E'A' in RVW with E peak velocity of 3.7 cm/s. Tricuspid regurgitation velocity was 3.39 m/s and pulmonary regurgitation was 2.37 m/s (Fig. 5).

Based on radiographic and echocardiographic examinations, the tentative diagnosis was DCM accompanying severe pulmonary hypertension. The dog was medicated with pimobendan (0.25 mg/kg orally twice daily), furosemide (2

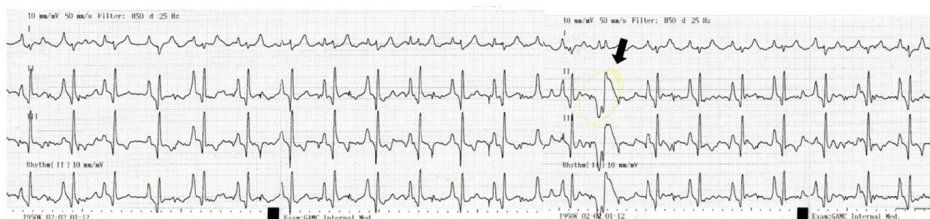


Fig. 1. Electrocardiogram of the patient showing tall and wide P wave with ventricular premature complexes (arrow).

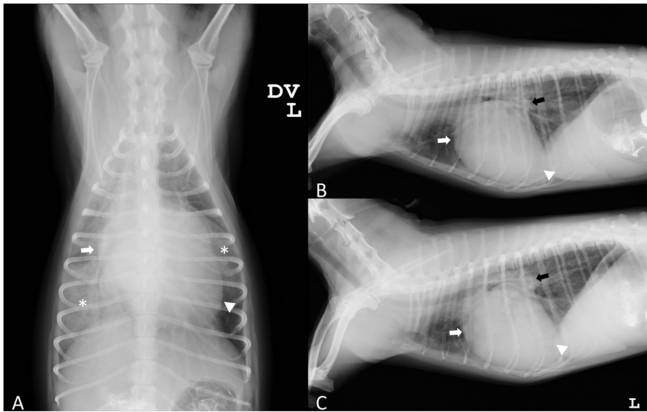


Fig. 2. Dorsoventral (A), right lateral (B), and left lateral (C) thoracic radiographs of the patient revealing moderate enlargement of the left ventricle (arrowhead) with mild bulging of the right (white arrow) and left (black arrow) atria. Moderate alveolar pattern (asterisk) in the right lung lobe and cranial part of the left caudal lung lobe are also detected.

mg/kg orally twice daily), ramipril (0.125 mg/kg orally once daily), and taurine (500 mg/dog orally twice daily). After 10 days of following-up, the dog had a sudden death.

Discussion

Idiopathic DCM is commonly found in large and medium sized breed dogs weighing more than 12-15 kilograms (5,9,17,21,26) such as Doberman Pinscher, Irish Wolfhound, Portuguese water dogs, and Newfoundland (1,3,5,9,17,18,21,22,26). Small breed canine patients with DCM are very rare, making it difficult to determine its etiology. This is a very rare case of a small Maltese dog weighting 4.95 kg with DCM.

Clinical signs of DCM include weakness, lethargy, tachypnea or dyspnea, exercise intolerance, cough, anorexia, abdominal distension, ascites, and syncope (9,17,21,22,24,26). However, clinical signs may not be noticed until DCM is progressed (9,17,21,22,24,26). The present case also had severe cough and tachypnea for a month. Auscultation revealed arrhythmia.

Electrocardiography (ECG) findings for DCM include QRS complex widening, P wave widening and notching, uniform or multiform ventricular premature complex (VPCs), and paroxysmal ventricular tachycardia (26). In the present case, ECG revealed tall and wide P wave and VPCs.

Secondary DCM might be associated with toxic change, infectious agents, endocrinopathies, and nutritional disorder (3,5,9,17,18,22,24). Previous reports have shown that lower levels of taurine and carnitine (5,9-11,17,18,20,26)

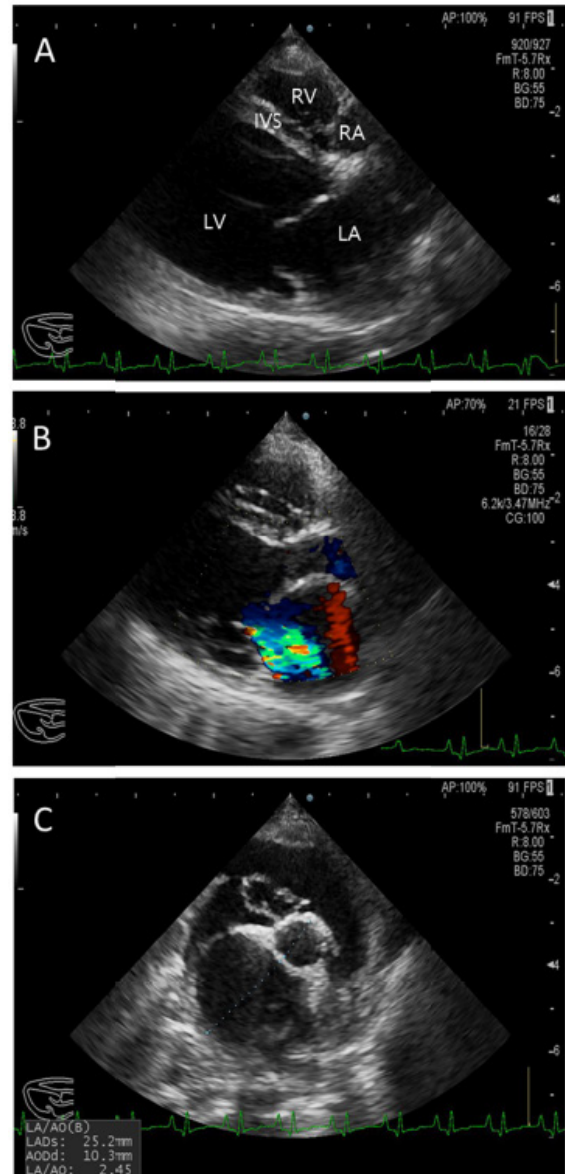


Fig. 3. Echocardiography of the patient. Right parasternal long-axis (A, B) and short axis view (C) showing severe dilated left atrium (LA) and left ventricle (LV). Color-flow Doppler echocardiogram (B) showing eccentric mitral regurgitation during systole (arrow). The left atrial-aortic root ratio (LA/Ao) was 2.45 (C).

could affect breeds such as Standard schnauzer (13) and Portuguese water dogs, especially Cocker Spaniels (3,9,10-12,14,17,18,20,22,24,26). In addition, DCM may occur if a normal dog is supplied with less taurine and carnitine. Providing taurine supplementation can improve their survival time (7,10,11). In the present case, although the dog was treated with 500 mg taurine and L-carnitine BID for one day, the dog died suddenly.

Hypothyroidism with hypercholesterolemia can occur in

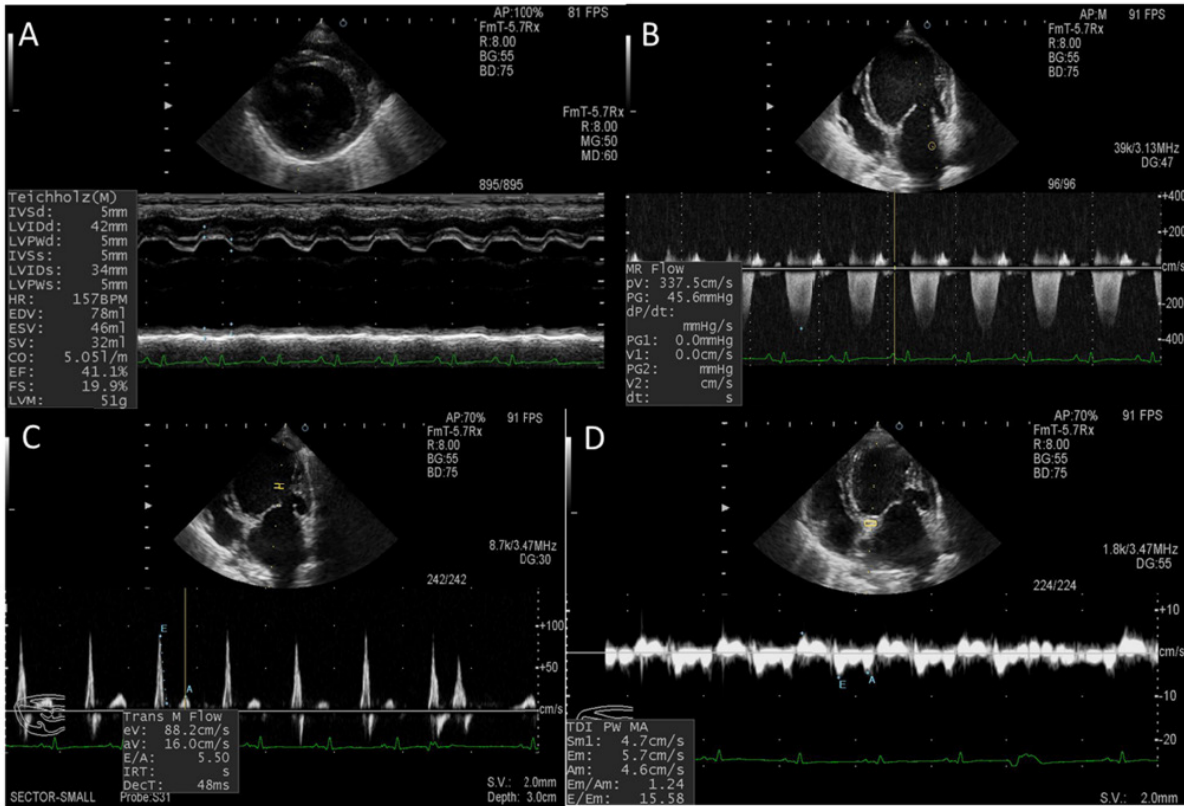


Fig. 4. Transventricular time-motion echocardiogram (A), spectral Doppler (B, C), and tissue Doppler imaging (D) of the patient. Doppler imaging was taken from the left apical four-chamber view. (A, B) Low shortening fraction (19.9%) and mitral regurgitation velocity (5.96 m/s) were identified. (C) Early diastolic mitral inflow velocity (0.88 m/s) was increased with an E:A ratio of 5.5 and a short E-wave deceleration time (48 ms). (D) Tissue Doppler imaging in LVW showing no reversal E'A'.

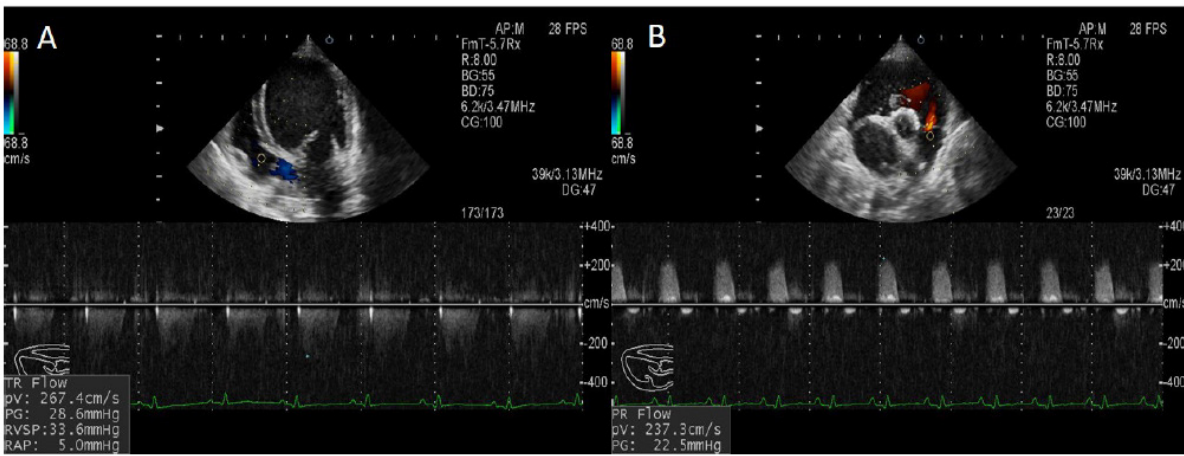


Fig. 5. Continuous wave Doppler echocardiogram. Note tricuspid regurgitation (3.39 m/s) on the left long-axis of a four-chamber view (A) and pulmonary regurgitation (2.37 m/s) on the left short axis view (B).

some dogs with DCM. However, most dogs with DCM have normal thyrotropin stimulating hormone and free T4 concentrations (2,9,17,24-26). Note that human is affected by hyperthyroidism (27). In the present case, blood test revealed mild-

ly low levels of tT4 and ft4. Therefore, it was unclear whether its DCM was caused by taurine deficiency or hypothyroidism.

DCM refers to a myocardial disease characterized by a cardiac chamber dilation causing myocardial systolic and

diastolic dysfunction, especially in the left side of the heart (3,5,8,9,16-18,21-23,24,26). Previously studies have reported some methods for diagnosis DCM, such as left-sided or four chamber dilatation and impaired left ventricular systolic function (3,5,8,9,16-18,21-23,24,26). Thoracic radiographs can reveal left atrial and left ventricular enlargement (may right atrial and right ventricle), pulmonary vein dilation, remarkable pulmonary vasculature, and pulmonary edema due to left sided congestive heart failure (21,23). Echocardiography is the most useful assessment for diagnosing DCM. 2D and M-mode evaluation in echocardiography showed dilated left atrial and ventricle, normal to thin left ventricular wall and intraventricular septal, poor FS, and large EPSS. Color Doppler evaluation show mitral insufficiency secondary to dilation of mitral annulus. Systolic dysfunction is significant. Low aortic velocity is a consequence of reduced cardiac output. DCM may show diastolic dysfunction and increased E wave in mitral inflow studies (3,5,16,21,23,26).

Many radiographic and echocardiographic studies on DCM are limited to large breed dogs. The present case shows typical findings in thoracic radiography and echocardiography such as left-side heart enlargement, pulmonary edema, poor FS, high mitral regurgitation, and high E peak.

Restrictive TMF pattern is one of indicators for predicting poor prognosis of dogs with DCM. This restrictive pattern is characterized by a high E-wave velocity, a short E-wave deceleration time (rapid filling in early diastole), and a reduced A-wave velocity (minimal filling during atrial systole) (4). The present patient also showed high E-wave velocity, short E-wave deceleration time, and reduced A-wave velocity as features of a restrictive TMF pattern. Thus, its prognosis was expected to be poor.

Severe DCM can proceed to pulmonary artery hypertension secondary to left-sided heart failure (3,9,16-18,21,24,26). Previous reports have shown that the presence of pulmonary hypertension concomitant with poor right ventricular function is associated with a worse prognosis for both human and dogs with DCM (3,15-18,22,24,26). If symptoms indicate congestion heart failure, the prognosis is often poor (3,9,17,18,21,22,26). This dog also had severe pulmonary hypertension with a sudden death.

Conclusions

This case describes echocardiographic diagnosis of a dilated cardiomyopathy rarely reported in small breed dogs. We should consider a poor prognosis if restrictive TMF pattern and pulmonary artery hypertension are found in dogs with DCM.

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