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# Primary Dilated Cardiomyopathy in a Miniature Pinscher Dog

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**Abstract :** A 9-month-old, female Miniature Pinscher (MP) dog weighing 1.97kg was presented because of periodic syncopal episode for 5 months. This case was diagnosed as primary dilated cardiomyopathy based on respiratory distress history, weak femoral pulse, generalized cardiomegaly, pulmonary edema, marked dilation of left atrium (LA) and left ventricle (LV), decreased wall thickness of LV and interventricular septum (IVS), increased EPSS in echocardiography, and young age of onset in the absence of other cardiovascular disorders. The patient was stabilized by application of diuretics (Furosemide, 2 mg/kg, SC, q 1 hr) and venodilator (Nitroglycerine patch, 0.5 mg/kg, q 12 hrs). Clinical signs were improved with medical management of positive inotropic vasodilator (Pimobendan, 0.2 mg/kg, PO, q 12 hrs) and angiotensin-converting enzyme (ACE) inhibitor (benazepril, 0.5 mg/kg, PO, q 12 hrs), potassium gluconate gel (2 mEq/dog, PO, q 12 hrs) and, L-carnitine (50 mg/kg, PO, q 12 hrs). The dog still maintains stable clinical status 10 months after the first visit. We report the rare case of DCM in small breed dog, which corresponds to the diagnosis and treatment of typical DCM in large breed dog.

***Key words*** **:** dilated cardiomyopathy, syncope, Miniature Pinscher, pulmonary edema.

## Introduction

Dilated cardiomyopathy (DCM) in dogs is a myocardial disease characterized by ventricular and atrial enlargement, and myocardial (primarily systolic but not soley) dysfunction subsequent with congestive heart failure (CHF) often developing at some stage without other cardiovascular disorders (32). The disease has been recognized in many medium-sized, large and giant breed dogs, especially Doberman Pinschers (5), Great Danes, English (29) and American (15) Cocker Spaniels, Dalmatians (10), Newfoundlands (31), Irish Wolfhounds, Saint Bernards, Old English Sheep dogs, and Scottish Deer hounds (28). However, the diseases are rarely identified in dogs weighing less than 12 kg, and overrepresented in male dogs and/or increase incidence with age (20,28). The age of onset of this progressive disease varies between 3 and 7 years of age, although DCM can be occurred in young age in certain breed dog (e.g., Doberman Pinschers; DP) (21).

The etiology of the primary DCM is usually idiopathic, however antibodies against beta-receptors (18) or the adenine nucleotide translocator (25), mutations in the cardiac actin gene (24) are proved in further study. For etiology of the secondary DCM, a lot of theories have been proposed, such as taurine (15) and/or carnitine (14) deficiency, toxic factors (28), and possibly pregnancy (25). Recent human study also found that DCM was associated with immunologic (17,32) or viral etiology (31). It is fatal if the etiology cannot be eliminated. The most common clinical signs of DCM are respiratory distress, syncope, and sudden death. In a majority of DP, it is presumed that paroxysmal ventricular tachycardia progressing to ventricular fibrillation results in the sudden death (6). Infrequently, however, bradyarrythmias may detected before the onset of sudden death in some DP (7,16).

This case report describes DCM in MP which is the rare breed for DCM.

## Case Report

A 9-month-old, intact female Miniature Pinscher dog weighing 1.97 kg was presented at the veterinary medical teaching hospital because of persistent dyspnea and periodic syncopal episode for 5 months. The dog was responsive to cardiac medication at local animal hospital. At presentation, the dog showed dyspnea and tachypnea with open mouth breathing. Body condition score was 2 of 5 scales, which indicated slightly thin.

On physical examination, weak femoral pulse and tachypnea (52 breathing per minute) were detected. Cardiac auscultation revealed tachycardia (170 bpm) with systolic murmurs of grade 4. The CBC and serum chemistry profiles were not remarkable.

Serial electrocardiographic examination (NIHON KOHDEN Cardiofax GEM) could not detect any arrhythmia. It showed sinus rhythms (normo to tachycardia) with an average heart

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rate of 170 bpm (Fig 1) and tall QRS complexes and deep Q waves indicating left ventricle enlargement.

Radiographic examination revealed the generalized cardiomegaly and increased vertebral heart score (VHS: 14.5). The trachea was elevated dorsally and pulmonary vein was enlarged. Pulmonary edema was obvious with finding of air bronchogram and mixed alveolar infiltration in hilar region and caudal lung lobe (Fig 2).

The two dimensional echocardiography (LOGIQ400, GE, conneticut, USA) taken from the right parasternal view showed a marked dilation of left atrium (LA) and left ventricle (LV), increased E point septal separation (EPSS) , reduced interventricular septal (IVS) and left ventricular wall (LVW) thickness from the right parasternal short-axis view (Fig 3, Fig 4 and Table 1).

Calculated fractional shortening (Table 1) was lower limit of reference range. Hyperechoic mass suspected thromboembolism was observed in LA (Fig 5). Mitral blood turbulence with intact mitral valve leaflet was detected on the long axis view echocardiography (Fig 6).

Based on the clinical findings including physical examination, auscultation, ECG, thoracic radiography, and echocardiography, this patient was diagnosed as DCM. Oxygen (oxygen cage, 100 ml/kg/min) was supplied for the stabilization of respiratory distress. We administrated the diuretics (Furosemide: Lasix®, Handok pharma Co, Chungbuk, Korea, 2mg/kg, SC, q 1 hour) till the respiration was stabilized. And we also applied the venodilator (Nitroglycerine: Angiderm® patch, Samyangsa, Daejeon, Korea, 0.5 mg/kg) to reduce venous return to LA. After respiration stabilized, we offered water and food (a/d, Hill's Pet Products, Topeka, KS, USA). Furosemide (2 mg/kg, PO, q 12 hrs), positive inotropic vasodilator (Pimobendan: Vetmedin®, Boehringe Ingelheim, North Ryde NSW, 0.2 mg/kg, PO, q 12 hrs), ACE inhibitor (benazepril: Cibacen 10®, Novartis pharma AG, Switzerland, 0.5 mg/kg, PO, q 12 hrs), and potassium gluconate gel (Renal K+™ Vet solutions, USA, 2mEq/dog q 12 hrs) were prescribed to manage the CHF caused by DCM.

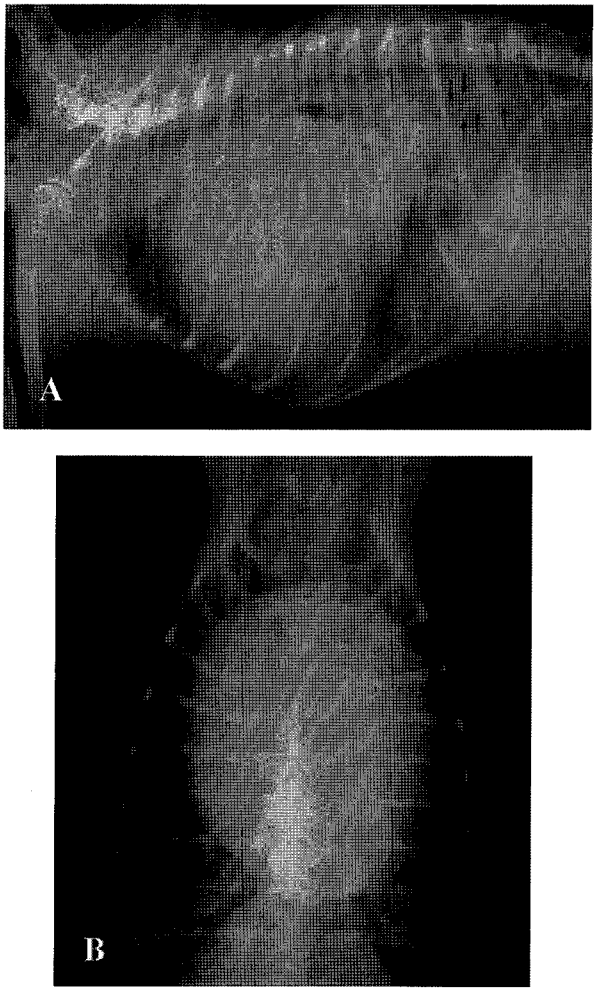


**Fig 2.** Right lateral (A) and ventrodorsal (B) thoracic radiographs of the patient. **A**, The caudal waist of heart are largely obscured by pulmonary edema in the perihilar and middle-lung regions. The air bronchogram is obvious in the lung field (arrows). The trachea is dorsally displaced due to generalized cardiomegaly. **B**, Severe cardiomegaly (especially left atrial and ventricular enlargement) was seen.

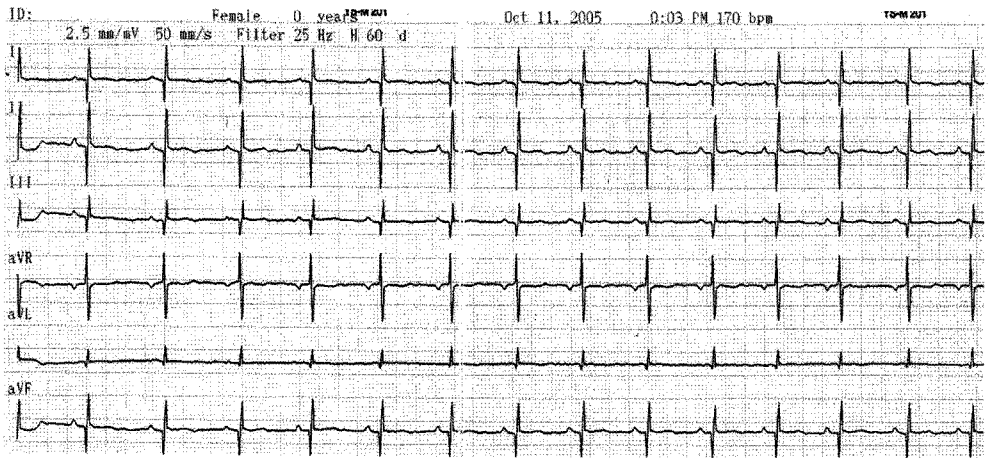


**Fig 1.** The 6 lead ECG tracing showed tall QRS complex and deep Q wave indicating LV enlargement (2.5 mm/mV, 50 mm/sec).

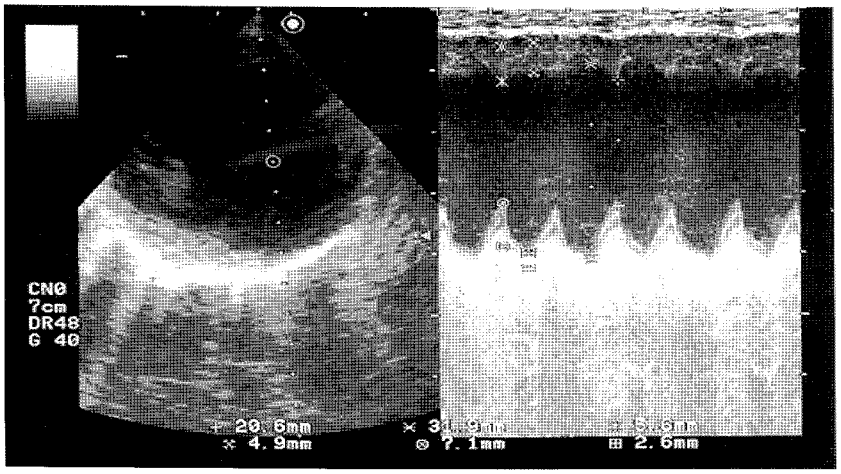


**Fig 3.** The 2D and M-mode echocardiography taken from right parasternal short axis view of papillary muscle level showed increased LV dimension in systole and diastole.

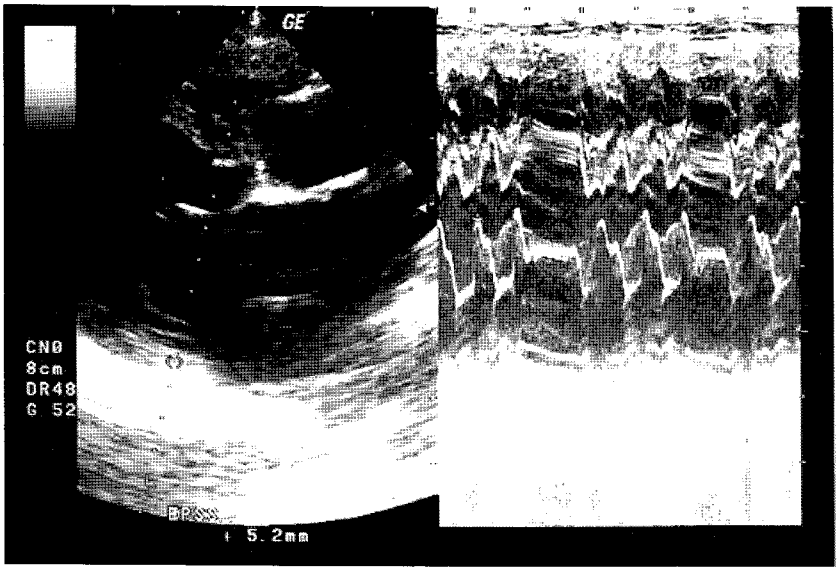


**Fig 4.** The 2D and M-mode echocardiography taken from right parasternal long axis view of four chamber showed increased EPSS.

**Table 1.** Echocardiogaraphic measurement of this patient. (1.97 kg dog)*

| | Diastole | Systole | Reference range (mm) | |
|---|---|---|---|---|
| **IVS** | 4.9 | 5.6 | 5.0~7.1 | 7.6~10.0 |
| **LVW** | 2.1 | 7.1 | 4.0~5.7 | 6.9~9.0 |
| **ESD** | 20.6 | | 8.4~12.6 | |
| **EDD** | 31.9 | | 16.1~21.9 | |
| **FS (%)** | 35 | | 33~46 | |
| **EPSS (cm)** | 0.52 | | 0-0.1 | |

IVS, interventricular septum; LVW, left ventricular wall; ESD, end-systolic dimension; EDD, end-diastolic dimension; FS, fractional shortening; E point septal seperation; EPSS
*June A. Boon. Appendix. In: Manual of veterinary echocardiography. 1st ed. Baltimore: Lippincott Williams & Wilkins. 1998.

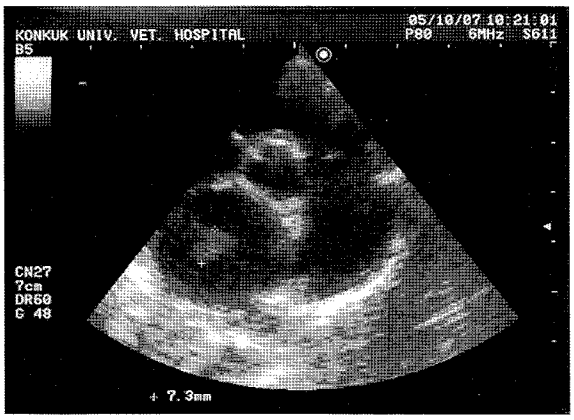


**Fig 5.** The 2D-ehocardiography taken from the right parasternal short axis view. The hyperechoic mass of 7.3 mm diameter was obsereved in left atrium

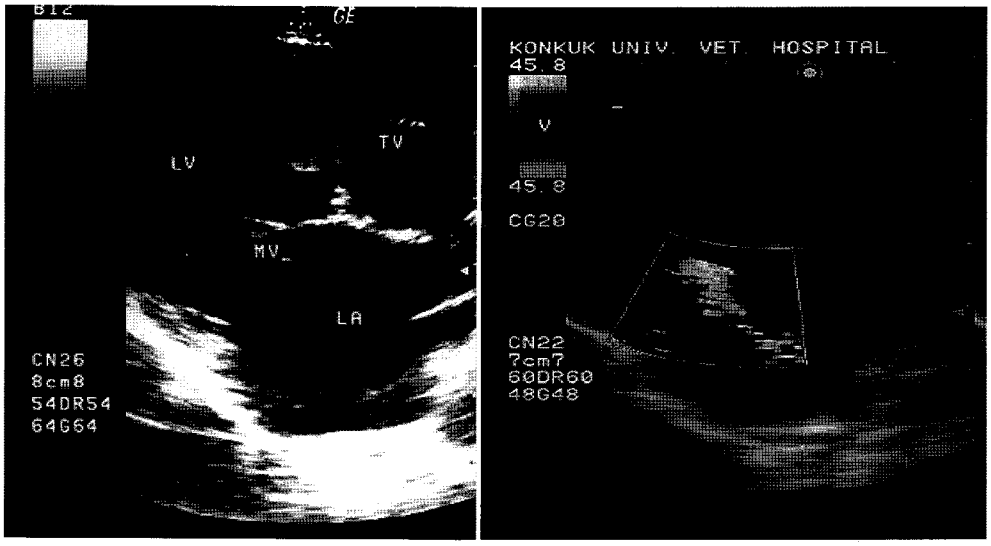


**Fig 6.** Color image echocardiography taken from the right parasternal long axis view of left ventricle outflow level showed mild turbulent flow to LA implying interatrial septum protrusion into RA suggested severely increased LA pressure.

On the 5th day after therapy, the patent showed marked improvement of clinical signs although the appetite was slight decreased and sinus tachycardia were persisted. L-carnitine (L-CARNITINE®, Rexall, Inc, USA, 50 mg/kg, PO, q 12 hrs) was added to the medication for 7 days.

On the 11th day after therapy, she revisited for mild depression, tachycardia (168 bpm), tachypnea (72 bpm) and anorexia. Oxygen therapy, nitroglycerine patch, furosemide were applied for 12 hrs for intensive care. Additionally we prescribed with adjusting dosage of furosemide to 2.5 mg/kg (PO, q 12 hrs) for 7 days. As the clinical signs were improved, the client refused medication and periodical examinations of the patient. However the patient was stable and still healthy at 10 months after the first visit.

## Discussion

DCM has long been suspected to have a genetic basis in dogs due to the breed predisposition. Furthermore, a noted familial predisposition has been also found in certain dog breeds (11). Therefore a systemic genetic screening for canine DCM is warranted to remove affected dogs from breeding stocks. Most of the natural history of data available concerning DCM has not been studied in most breeds except for the DP. One needs to ask whether the clinical features, natural course, and response to therapy as described for the DP are typical for other dogs. It is presumed that DCM in other breeds is modeled on that of the DP, except that the progression of DCM in the final stage (overt stage) is more rapid in the DP (19). Because MP has genetic relation with DP, presumably clinical presentation of DCM in MP is expected to have some similarities(9). Furthermore juvenile onset type of DCM in DP is supportive the young age of this case.

Clinical signs of DCM in DP are weakness, loss of appetite, weight loss, depression, episodes of collapse, respiratory difficulties, a soft cough (especially at rest), and an enlarged abdomen (ascite). The syncopal episode in this patient were referable to abnormalities of heart rhythm (particularly atrial fibrillation) (21) or generalized heart failure (3).

In physical examination, dogs with DCM have weak femoral pulse as in this case. However the variability in femoral pulse may present.

In diagnosis of DCM, systemic diagnostic tests are required to rule out other cardiac diseases, which may result in a dilated, hypokinetic heart (11). Typical findings include increased left ventricular end-systolic dimension (ESD) and end-diastolic dimension (EDD) and decreased fractional shortening (FS) (1). In this case, ESD and EDD were increased but FS was lower limit of reference range. FS might be lower, because FS was measured in short axis view. Even this case missed, however, EF evaluated by B-mode ultrasonography might represent a more accurate index of global myocardial function compared with FS because increased septal movement cause increased FS.

EPSS is also an important parameter to discriminate mitral valve dysplasia (MVD) from DCM. In MVD, EPSS is generally decreased due to thickened mitral valve, however EPSS is increased in DCM due to increased LV dimension causing increased septal separate from mitral valve excursion. In this case, we proved increased EPSS. Furthermore we excluded the mitral valve insufficiency by detect the intact mitral valve leaflet and absent of mitral valvular prolapse into LA and generalized decreased wall thickeness on the echocardiography.

Radiographic features of DCM included generalized cardiomegaly with LA and LV enlargement and in advanced DCM pulmonary edema is often obvious. In this case, all radiographic features were clearly observed.

Electrocardiographic reatures of DCM ere LA and LV enlargement (e.g., widened P wave, tall QRS complexes, left axis deviation), atrial fibrillation (AF) or ventricular fibrillation (VF) in certain type of DCM (19).

Recently European Society of Veterinary Cardiology provides a diagnostic guideline for canine DCM, which can be appropriately used in most dog breeds (11). In that score system, the major criteria include LV in M-mode systolic or diastolic dimensions exceeding 95% of reference range which is available for that breed, increased sphericity, decreased FS, and /or two-D derived left ventricular ejection fraction <40%. In addition, the minor criteria is that presence of an arrhythmia, atrial fibrillation, increased mitral M-mode end point to septal separation, increased pre-ejection period (PEP): ejection time (ET) ratio, and left or bi-atrial enlargement. The identification of one or more major or minor criteria should prompt regular (six-monthly or annual) re-evaluation.

For treatment of DCM, the proper drugs are essential to survival. For stabilization of sudden onset of heart failure, we used diuretics to remove excess fluid from pulmonary vasculature, and nitroglycerin to decrease the amount of venous return to LA. In addition, digitalis, enzyme blockers, vasodilators, carnitine and taurine were administrated to improve the clinical sign of DCM (3,12-15,21). Even if laboratory endomyocardial carnitine deficiency has not been proved, oral L-carnitine therapy could be recommended in young dogs with familial DCM. It has been reported that there were significant delay in the time to onset of the overt stage of DCM with the use of ACE inhibitors versus no therapy (18).

To date, no studies have been conducted to address the risk factor of sudden death in canine population. Even though antiarrythmic agents can be used to reduce the risk of sudden death, the human experience in such cases suggests that most antiarryhthmic agents are ineffective in this capacity. In fact, most antiarrhthmic agents probably increase the risk of sudden death in human (19).

## References


1. Borgarelli M, Santilli R.A, Chiavegato D, D'Agnolo G, Zanatta R, Mannelli A, Tarducci A. Prognostic Indicators for Dogs with Dilated Cardiomyopathy. J Vet Intern Med 2006; 20: 104-110.
2. Borgarelli M, Tarducci A, Santilli RA, Priano L. Fattori prognostici ecocardiografici ed eco Doppler in cani conmiocardiopatia dilatativa. Atti S I S Vet 1998; 52: 233.
3. Borgarelli M, Tarducci A, Tidholm A, Häggström J. Canine Idiopathic Dilated Cardiomyopathy. Part II: Pathophysiology and therapy. Vet J 2001; 162: 182-195.
4. Broschk C, Distl O. Dilated cardiomyopathy in dogs: pathological, clinical, diagnosis and genetic aspects. Dtsch Tierarztl Wochenschr 2005; 112: 380-385.
5. Calvert CA, Chapman WL, Total RL. Congestive cardiomyopathy in Doberman Pinscher dogs. J Am Vet Med Assoc 1982; 181: 598-602.
6. Calvert CA, Hall G, Jacobs G, Pickus C. Clinical and pathologic findings in Doberman pinschers with occult cardiomyopathy that died suddenly or developed congestive heart failure 54 cases (1984-1991). J Am Vet Med Assoc 1997; 210: 505-511.
7. Calvert CA, Jacobs GJ, Pickus CW. Bradycardia-associated episodic weakness, syncope, and aborted sudden death in cardiomyopathic Doberman Pinschers. J Vet Intern Med 1996; 10: 88-93.
8. Calvert CA., Pickus CW, Jacobs GJ, brown J. Signalement, survival and prognostic factors in Doberman Pinschers with end-stage cardiomyopathy. J Vet Intern Med 1997; 11: 323-326.
9. Dominique GJ, Josee and Jean PV. The royal canin Dog encyclopedia. Aniwa publishing : 67-68
10. Freeman LM, Michel KE, Brown DJ, Kaplan PM, Stamoulis ME, Posenthal SL, Keene BW, Rush JE. Idiopathic dilated cardiomyopathy in dalmatians: nine cases (1990-1995). J Am Vet Med Assoc 1996; 209: 1592.
11. Joanna Dukes-McEwan. Dilated Cardiomyopathy: European Experience. The 23rd Proceedings of American College of Veterinary Internal Medicine Forum 2005: 101-103.
12. Keene BW, Panciera DP, Atkins CE, Reqitz V, Schmidt MJ, Shug AL. Myocardial carnitine deficiency in a family of dog with dilated cardiomyopathy. J Am Vet Med Assoc. 1991; 198: 647-650.
13. Keene BW. L-carnitine deficiency in canine dilated cardiomyopathy. In Kirk RW, Bonagura JD (Eds). Kirk's Current Veterinary Therapy XI WB Saunders Co Philadelphia 1992: 780.
14. Keene BW, Panciera DP, Atkins CE. Myocardial L-carnitine deficiency in a family of dogs with dilated cardiomyopathy. J Am Vet Med Assoc 1991; 198: 647-650.
15. Kittelson MD, Keene B, Pion PD, Loyer CG. 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: 204-211.
16. Kittleson MD. Primary myocardial disease leading to chronic myocardial failure (dilated cardiomyopathy and realated diseases). In: Kittleson MD, Kienle RD, editors. Small animal cardiovascular medicine. St Louis Mosby; 1998: 319-346.
17. Limas CJ, Goldenberg IF, Limas C. Autoantibodies against beta-adrenoseptors in human idiopathic dilated cardiomyopathy. Cir Res 1989; 64: 97-103.
18. Lazaros A. Nikolaidis, MD. Angiotensin-converting enzyme inhibitors improve coronary flow reserve in dilated cardiomyopathy by a bradykini-mediated, Nitric Oxide-Dependent Mechanism. Circulation 2002: 2785-2790.
19. Michael R. O'Grady, and Lynne O'sullivan M. Dilated cardiomyopthy: an update. Vet Clin Small Anim 2004; 34: 1187-1207.
20. Monnet E, Orton C, Salman M, et al. Idiopathic dilated cardiomyopathy in dogs: survival and prognostic indicators. J Vet Intern Med 1995; 9: 12-17.
21. Morales M, Ynaraja E, Montoya JA. Dilated cardiomyopathy in Presa Canario dogs: ECG findings. J Vet Med Assoc 2001; A 48: 577-580.
22. O'Grady MR, Minors SL. Resting and dobutamine stress echocardiographic factors associated with the development of occult dilated cardiomyopathy in healthy Doberman pinscher dogs. J Vet Intern Med 1998; 12: 369-380.

23. O'Grady MR, O'Sullivian ML. Dilataed cardiomyoopathy: an update. Vet Clin North Am Samll Anim Prac 2004; 34: 1187-1207.
24. Olson TM, Michels VV, Thibodeau SN, Tai YS, Keating MT. Actin mutations in dilaed cardiomyopathy, a heretable form of heart failure. Science 1998; 280: 750-752.
25. Sandusky G.E Cho DY. Congestive cardiomyopathy in a dog associated with pregnancy. Cornell Vet 1984; 74: 60-64.
26. Schultheiss HP, Bolte HD. Immunological analysis of auto-antibodies against the adenine nucleotide translocator in dilated cardiomyopathy. J Mol Cell Cardiol 1985; 17: 603-617.
27. Sisson D, O'Grady MR, and Calvert CA. Myocardial disease of dogs. In: Fox PR., Sisson D, and Moïse NS, (eds), Canine and Feline Cardilolgy 2nd edn. WB Saunders Philadelphia 1999: 581-619.
28. Sisson DD, Thomas WP, Keene BW. Primary myocardial disease in the dog. In: Ettinger SJ, Felman EC, editors. Textbook of veterinary internal medicine 5th edition Philadelphia. WB Saunders 2000: 874-895.
29. Staaden RV. Cardiomyopathy of English Cocker Spaniels. J Am Vet Med Assoc 1981; 178: 1289-1292.
30. Tidholm A, Häggström J, Borgarelli M, Tarducci A. Canine Idiopathic Dilated Cardiomyopathy. Part I: Aetiology, Clinical Characteristics, Epidemiology and Pathology. Vet J 2001; 162: 92-107.
31. Tidholm A, Jonsson L. Dilated cardiomyopathy in the Newfoundland: a study of 37 cases (1983-1994). J Am Anim Hosp Assoc 1996; 32: 465-470.
32. Wynne J, Braunwald E. The cardiomyopathies and myocarditides. In: Braunwald E, Zipes DP, Libby P, editors. Heart disease: a textbook of cardiovascular medicine. 6th edition. Philadelphia WB Saunders 2001: 1751-1806.

# 미니어쳐 핀셔견에서 발생한 확장성 심근질환

**김정현 · 박 철 · 고기진 · 강병택 · 정동인 · 김주원 · 김하정 · 임채영 · 이소영 · 조수경 · 박희명[1]**

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**요 약** : 5개월간의 일시적인 기절현상(syncope)을 주증상으로 내원한 9개월령의 1.97 kg 미니어쳐 핀셔견에서 호흡곤란, 심초음파에서의 현저한 심실/심방의 확장, 전반적인 심비대, 약한 맥박, 폐수종 등 DCM의 진단 지표를 기준을 통해 확장성 심근병을 진단하였다. 본 증례에서 심초음파 검사는 심장의 확장과 심무력을 유발하는 다른 심장 질환을 배제하고 DCM을 확진 하는데 중요한 역할을 하였다. 치료를 위해 산소공급과, nitroglycerine patch, furosemide, pimobendan, benazepril, potassium gluconate gel을 19일간 처방한 결과 초기 내원시 보였던 대부분의 임상증상이 개선되었다.

**주요어** : 확장성 심근질환 (DCM), 기절현상, 미니어쳐 핀셔, 폐수종.