大韓獸醫學會誌(2004) 第44卷 第1號
Korea J Vet Res(2004) 44(1) : 125~129

American cocker spaniel dog에서 발생한 삼첨판 이형성 증례

박 철 · 최치봉 · 서정향[1] · 정병현 · 박희명*

건국대학교 수의과대학 내과학 교실
[1]건국대학교 수의과대학 병리학 교실

(게재승인: 2004년 2월 24일)

Tricuspid valve dysplasia(TVD) in an American cocker spaniel dog

Chul Park, Chi-Bong Choi, Jung-Hyang Sur[1], Byung-Hyun Chung, and Hee-Myung Park*

Department of Veterinary Internal Medicine, College of Veterinary Medicine, Konkuk University, Seoul 143-701, Korea
[1]Department of Veterinary Pathology, College of Veterinary Medicine, Konkuk University, Seoul 143-701, Korea

(Accepted: February 24, 2004)

Abstract : A 2-year-old, female, American cocker spaniel dog presented for a 1-year history of severe ascites, exercise intolerance, tachypnea. At that time, she was in an emergency state. First, the dog was stabilized with oxygen therapy. A diagnosis of cardiac problem was made from history, auscultation, radiograph, ECG, and echocardiography. Jugular pulsation was palpated and a harsh, systolic murmur of tricuspid regurgitation was prominent at the right cardiac apex. Tricuspid valve dysplasia (TVD) was confirmed with echocardiography, accompanying enormous myocardial hypertrophy. The clinical signs had been improved for 8 months with careful therapy and periodic abdominocentesis, and ascites was well controlled. The situation, however, became worse quickly in a week because the client did not follow our management schedule. Finally, she died due to dyspnea and shock. After the spontaneous death, necropsy and histopathological examination were performed and when we opened the thorax, a significantly large heart was observed. On histopathological findings, grossly myocardium appeared pale initially, then progressed to yellow and white. Microscopically, there was an extensive hemorrhage along with loss of myocardial striations. Interstitial fibrosis and various degenerative alterations in myocytes were also present.

Key words : tricuspid valve dysplasia (TVD), ascites

Introduction

Congenital malformation of the tricuspid valve complex is seen occasionally in dogs. Since the advent of Doppler echocardiography, TVD has been recognized as a common cardiac problems in dogs and cats [4]. German shepherd dog, Golden retriever, Great Dane, Labrador retriever, Old English sheepdog, and Weimaraner are prone to congenital tricuspid valve malformation, especially in Labrador retrievers. There are no reports in the literature of tricuspid valve insufficiency in Korea. The most common clinical signs are typically a holosystolic murmur, a jugular distension and pulsation, pulmonary edema, ascites, tachypnea, and exercise intolerance. Evidence of reduced exercise tolerance, weakness, or syncope occurs mainly in instances of pulmonary hypertension secondary to chronic mitral valve insufficiency (MVI) or tachyarrhythmia. Affected dogs have a tendency for multiple congenital defects, including pulmonic stenosis, atrial septal defect, and ventricular preexcitation [5]. Valve stenosis can occur and may be severe in some cases [2]. Additionally, dogs with tricuspid dysplasia can have fusion of papillary muscles, patency of foramen

*Corresponding author: Hee-Myung Park
Department of Veterinary Internal Medicine, College of Veterinary Medicine, Konkuk University, Seoul 143-701, Korea
[Tel: +82-02-450-4140, Fax: +82-02-444-4396, E-mail: parkhee@kkucc.konkuk.ac.kr]

ovale, and fibrous epicarditis over the dilated right atrium.

This report describes a case of tricuspid valve dysplasia (TVD) in a dog associated with hypertrophic cardiomyopathy. The diagnosis of TVD was supported by echocardiographical and histopathological evaluations.

Case report

A 2-year-old, intact female, American cocker spaniel dog was presented to the internal medicine department at the University of Konkuk Veterinary Teaching Hospital (UKVTH) for a 1-year history of severe ascites and exercise intolerance. The owner reported that about 1 year prior to presentation, the dog had had ascites, dyspnea, and exercise intolerance. In addition, there had been no specific therapy for ascites until the patient was presented to the UKVTH.

Initial diagnostic evaluation included a complete blood count (CBC), serum chemistry profile, radiographs, electrocardiogram (ECG), echocardiography, and an heartworm test. On presentation to the UKVTH, the dog was dyspneic and cyanotic. A systolic murmur was checked during physical examination on the tricuspid valve area. The dog's abdomen was severely distended. Severe dental tartar and gingivitis were evident. Results of the CBC revealed polycythemia and serum biochemical abnormalities included hypernatremia(150 mmol/L; reference range, 138 to 148 mmol/L), an elevated ALT(63 U/L; reference range, 3 to 50 U/L) and ALP(182 U/L; reference range, 20 to 122 U/L). A lateral radiograph revealed a severely distended abdomen and narrowed thoracic cavity with a marked cardiomegaly, showing a vertebral heart scale of 15.5 vertebrae (Fig. 1A). The heart had an appearance of a ball, occupying the entire thoracic cavity on ventrodorsal projection (Fig. 1B). There was also severe enlargement of caudal vena cava. Result of the heartworm test was negative. A routine ECG confirmed deep S waves in lead II and right axis deviation of the mean electrical axis(172^{o}). P waves were typical P mitralae(0.08 seconds) and P pulmonalae(0.6 mV) in lead II, suggesting left and right atrial enlargement (Fig. 2). An echocardiogram was performed and showed severe right atrial dilatation and right ventricular hypertrophy with fusion of papillary muscles (Fig. 3). Tricuspid valve was also underdeveloped. The dog was treated with furosemide(4 mg/kg, PO, BID) and enalapril (0.5 mg/kg, PO, BID). Repeated thoracic radiographs were performed periodically, and cardiac size and ascites were checked. Although basically we had difficulty in controlling the patient because the dog already was accustomed to high sodium and protein diet and the client did not follow our management program with favor, the patient had been well controlled with careful medication and periodic abdominocentesis. The patient survived 7 more months after medical therapy and routine abdominocentesis. In the end, the dog died because of dyspnea and shock. At the time of necropsy, we confirmed abnormally big heart, fusion of papillary muscles and chordae tendineae, and an enormous cardiac hypertrophy (Fig. 4). Histopathology of myocardium revealed that myocardium has severely disarrayed and disorganized of myocytes. In addition, the yellow-brown granular pigment seen in the myocytes here is lipofusin

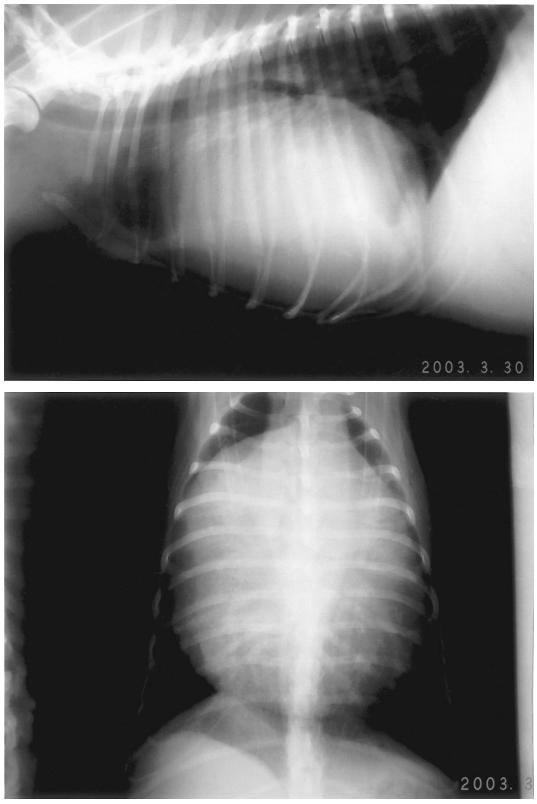

Fig. 1. Lateral radiograph obtained from a dog with TVD accompanying Congestive heart failure(CHF). The cardiac silhouette is tremendously enlarged(A). On the ventrodorsal projection, the heart is enormously enlarged and the caudal vena cave is prominent (B).

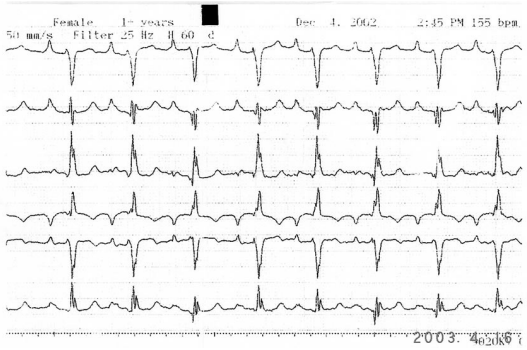

Fig. 2. The ECG record is quite abnormal, showing P pulmonale, P mitrale and deep Q waves and S waves on lead II. This ECG check confirmed right axis deviation of the mean electrical axis(172°), left and right atrial enlargement and right ventricular hypertrophy.

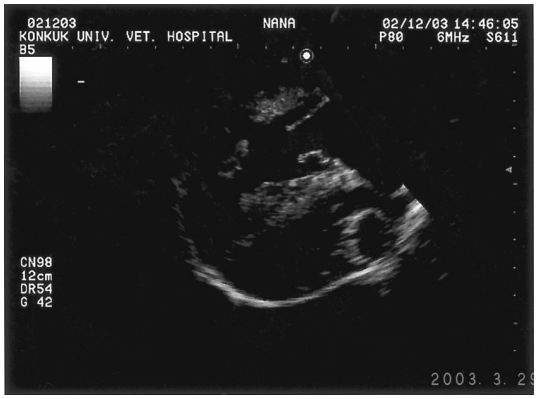

Fig. 3. Right parasternal long-axis images of the echocardiography may show the tricuspid valve tightly adhered to the ventricular septum and anterior tricuspid valve leaflet is elongated.

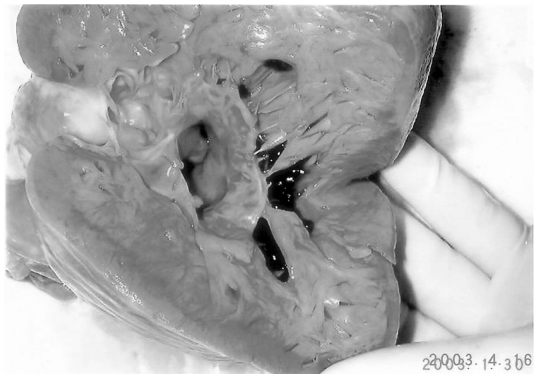

Fig. 4. On autopsy findings, fusion of chordae tendinae and papillary muscles is clear. In addition, papillary muscles are fused with each other.

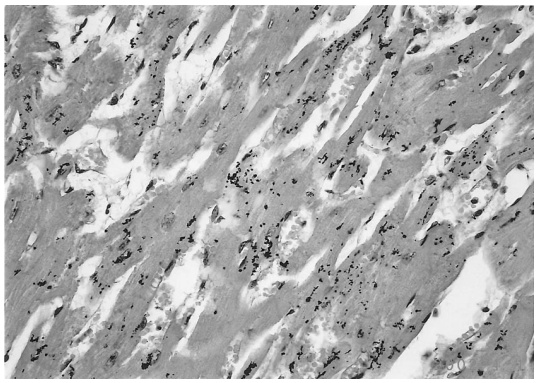

Fig. 5. Histopathologicaly, the myocardium have severely disarrayed and disorganized of myocytes. In addition, the yellow-brown granular pigment seen in the myocytes here is lipofusin suspected which accumulates over time in the myocytes as a result of “wear and tear” with overburden (hard-working muscles).

suspected which accumulates over time in myocytes as a result of “wear and tear” with overburden (hard-working muscles) (Fig. 5).

Discussion

The dog described in this report clearly showed echocardiographic findings consistent with TVD. On autopsy findings, the primary defect was a malformation of tricuspid valves and fusion of papillary muscles and chordae tendinae.

TVD, in the absence of concurrent obstruction of the pulmonary valve or pulmonary artery hypertension, is comparably well tolerated [6]. Generally, the lesions are tolerated for many years; however, in other cases, signs of progressive valve dysfunction, cardiomegaly, atrial arrhythmias and heart failure and death ensue [2, 9, 10, 11, 12].

Tricuspid dysplasia results in tricuspid valve insufficiency and systolic regurgitation of blood into the right atrium. In Ebstein’s anomaly, a recognized variant of tricuspid dysplasia, the tricuspid valve is displaced toward the cardiac apex. Because the right ventricle is designed to contract against a low-pressure artery, it is vulnerable to increases in pressure [1]. It responds poorly to the increased work; even relatively small acute increases in pulmonary artery pressure cause sharp decreases in the right ventricular stroke volume [7].

In this case, ascites was the most significant complaint following primary congenital TVD, exacerbating clinical signs.

In right-sided congestive heart failure, the increase in mean capillary hydraulic pressure increases the rate of fluid transfer from capillaries to the peritoneal space [1]. Increased pre- and postcapillary resistance, decreased renin and aldosterone clearance, decreased oncotic pressure gradient, and decreased lymphatic flow in the thoracic duct contribute to the formation of effusion [1]. That effusion is a modified transudate.

Abnormal echocardiographic findings in dogs with TVD are well documented on the screen. Therefore, definitive diagnosis requires echocardiography. Abnormal location, shape, motion, or attachment of the valve apparatus is observed by 2-dimentional (2-D) echo. A typical finding is the presence of a large, fused papillary muscles instead of normally small, discrete muscles [2]. Sometimes it is difficult to define where the anterior leaflet of the tricuspid valve is and where the chordae tendinae start [3, 8]. Actually, this case showed very short, underdeveloped chordae tendinae with fusion of papillary muscles on the autopsy findings. Typical right ventricular dilation was not observed on the 2-D imaging. Instead, tremendous right ventricular hypertrophy was remarkable. Although usually ECG has a low sensitivity in detecting right atrial and ventricular enlargement secondary to TVD [2], ECG also supported the evidence of right ventricular hypertrophy.

On radiographical findings, the heart occupied the entire thoracic cavity. Generalized cardiomegaly was quite clear. In general, thoracic radiographs demonstrate right-sided cardiomegaly, particularly right atrial enlargement, often with marked apex shifting to the left at the TVI patients. As the radiographic interpretation can be confusing without concurrent echocardiography, echo should be performed as a following diagnostic tool.

During autopsy, tremendous cardiomegaly was observed. At that time, the patient's heart weighed 0.89 kg - the dog's body weight had changed from 10.2 kg to 7.2 kg when she was alive. Normal percentage of the heart of the total body weight is 0.8~1 percent. Histopathologicaly, myocytes were severely disarranged and disorganized.

Conclusions

We diagnosed a TVD in an American cocker spaniel dog based on history taking, clinical signs, physical examination, blood works, radiography, ECG, and especially echocardiography. According to echocardiography, the dog had a tricuspid valve dysplasia with abnormal valvular motion, ventricular hypertrophy, and short chordae tendinae. The patient had survived for 8 months with cardiac therapy, however, medical management dammed up ability to control systemic dysfunction. In the end, the dog died due to dyspnea and shock. This case was unique as a TVD, because severe myocardial hypertrophy accompanied.

References

1. **Stephen, J. Ettinger and Edward, C.** Feldman, In: Textbook of Veterinary Internal Medicine. pp. 795-796, 5th ed. Saunders, Philadelphia, 2000, **137**.
2. **Philip, R. Fox, David Sisson and N. Sydney Moïse,** In: Textbook of Canine and Feline Cardiology. pp. 524-525, 2nd ed. Saunders, Philadelphia, 1999.
3. **June, A. Boon.** In: Manual of Veterinary echocardiography. pp. 424-431, 1st ed. Lippincott, Baltimore, 1998.
4. **Yuill, C. D. and O'Grady, M. R.** Doppler-derived velocity of blood flow across the cardiac valves in the normal dogs. Can J. Vet. Res. 1991, **55**, 185-192.
5. **Liu, S. K. and Tilley, L. P.** Dysplasia of the tricuspid valve in the dog and cat. J. Am. Vet. Med. Assoc. 1975, **167**, 465.
6. **Barbour, D. and Roberts, W. C.** Valve excision only versus valve excision plus replacement for active infective endocarditis involving the tricuspid valve. Am. J. Cardiol. 1986, **57**, 475-478.
7. **Wiedeman, H.** Cor pulmonale *In* Braunwald E (ed): Heart Disease. pp. 1604-1625, 5th ed. Saunders, Philadelphia, 1997.
8. **Wingfield, W. E. and Boon, J. A.** Echocardiography for the diagnosis of congenital heart defects in the dog. Vet. Clin. North Am. Small Anim. Pract. 1987, **17**, 735-753.
9. **Lehmkuhl, L. B., Ware, W. A. and Bonagura, J. D.** Mitral stenosis in 15 dogs. J. Vet. Intern. Med. 1994, **8**, 2.
10. **Hamlin, R. L., Smetzer, D. L. and Smith, C. R.** Congenital mitral insufficiency in the dog. J. Am. Vet. Med. Assoc. 1965, **146**, 1088.
11. **Weirich, W. E., Blevins, W. E., Conrad, C. R., Ruth,**

G. R. and Gallina, A. M. Congenital tricuspid insufficiency in a dog. J. Am. Vet. Med. Assoc. 1974, **164**, 1025-1028.

12. **Liu, S. K. and Tilley, L. P.** Dysplasia of the tricuspid valve in the dog and cat. J. Am. Vet. Med. Assoc. 1976, **169**, 625.