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Congenital Aortic Valvular Insufficiency Caused by Abnormal Valvular Structures in a Labrador Retriever Dog

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Abstract : A 10-month-old intact male Labrador Retriever dog was referred with the primary complaint of exercise intolerance, especially after vigorous exercise. Physical examination revealed split S1 and grade III/VI diastolic regurgitant murmur at the left apex and base, respectively. ECG finding was normal sinus rhythm at rest, but supraventricular tachycardia with bundle branch blocks after exercise. Thoracic radiography revealed dilated ascending aorta with normal range of cardiac silhouette (VHS 10.2). Echocardiography revealed abnormal valvular structures just above the aortic valvular cusps causing aortic regurgitation with a reduction of left ventricular ejection fraction (LVEF). Based on those findings, the case was diagnosed as congenital aortic regurgitation caused by abnormal valvular structures. The dog was managed with diltiazem and exercise restriction. This is a rare case of aortic deformity in dogs.

Key words **:** Labrador Retriever, dog, aortic valvular deformity, aortic insufficiency.

Introduction

Aortic valvular insufficiency is characterized by diastolic reflux through the aortic valve (aortic regurgitation) leading to a leakage of blood from the aorta into the left ventricle, which causes a subsequent left ventricular volume overload (left ventricular eccentric hypertrophy). The severity of the aortic valvular insufficiency (regurgitation) is dependent on the diastolic valve area, the diastolic pressure gradient between the aorta and left ventricle, and the duration of diastole (10). The common congenital causes of aortic insufficiency (AI) are aortic valvular deformities (i.e. bicuspid or quadricuspid aortic valve) (7,16), while the common acquired causes of AI are bacterial endocarditis, ruptured sinus of valsalva or laceration of aorta in dogs (2,11).

The AI often occurs with subvalvular aortic stenosis (SAS). It has been also observed with ventricular septal defects and tetralogy of Fallot and after balloon catheter dilatation for SAS (10). Although AI is a rare cardiac disease, especially AI with congenital-origin, it is recognized with increasing frequency as a complication of other cardiac malformations, thanks to technology of Doppler echocardiography (8,9,12). In AI, the degree of left ventricular eccentric hypertrophy entirely depends on the severity of the aortic valvular insufficiency. Although dogs with mild case of AI can survive for an entire life without accompanying severe clinical signs, dogs with a severe AI commonly dies of complications associated with left-sided congestive heart failure. Cardiac bypass surgery and valve replacement are definitive methods for repair in human medicine, although they are rarely applied in veterinary fields. Medical management for severe AI is directed to lessen clinical signs associated with left ventricular eccentric hypertrophy. The aims of this case study are to provide clinical and diagnostic features of AI in a dog and to enrich our resources for rare congenital heart defects.

Case

A 10-month-old intact male Labrador Retriever dog (25 kg of body weight) was referred at Veterinary Teaching Hospital of Kangwon National University with the primary complaint of severe exercise intolerance. Clinically the dog developed ananastasia after running 400 m track, suggesting a forward heart failure associated with exercise. In physical examination, split S1 and grade III/VI diastolic decrescendo murmur were audible loudly at the left apex and base, respectively (Fig 1A). However, no sensible abnormality was detected in the femoral pulsation. Complete blood cell count (CBC) and serum chemistry profiles have no significant abnormalities. On the day of presentation, electrocardiographic (ECG) studies revealed normal sinus rhythm, while ECG studies after exercise (running 400 m track) revealed runs of left bundle branch blocks (Fig 1B). Furthermore, the blood pressure was not increased after exercise, rather it was decreased (Table 1).

Thoracic radiography revealed marked aortic dilation, especially in aortic root and ascending aorta (Fig 2). In the 2-dimensional echocardiography in right parasternal short axis view, abnormal accessory valvular structures moving independently from the aortic valvular excursion were observed at just above the aortic

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Table 1. Echocardiographic dimensions of this case

Method [a]	A		B	
Pressure	Systolic pressure	Diastolic pressure	Systolic pressure	Diastolic pressure
Before exercise	145 ± 3.2 [b]	95 ± 2.9	143 ± 5.1	101 ± 6.2
After exercise	126 ± 6.8	70 ± 2.4	123 ± 6.8	67 ± 5.2

a) Blood pressures were measured by two different methods (Method A: Doppler method using Doppler flow detector 811B, Parks medical electronics; Method B: Volume plethysmographic method using VSM7, Vetspecs®)
b) Blood pressures were measured 5 times and shown as mean ± SD.

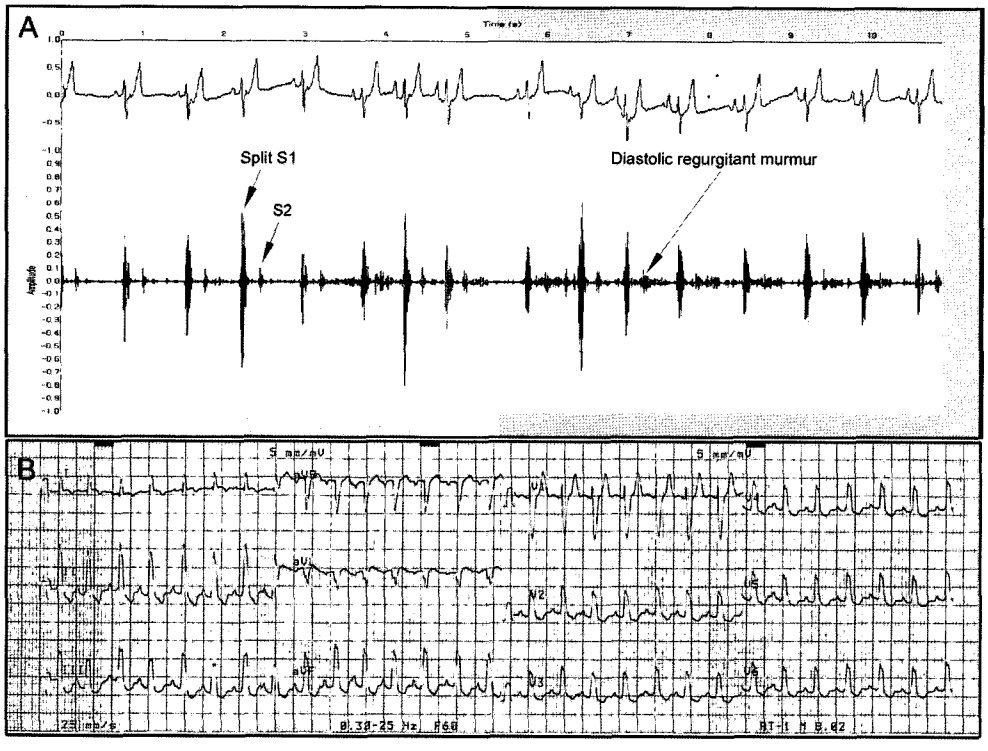

Fig 1. Phonocardiography revealed split S1 suggesting bundle branch block and diastolic decrescendo murmur suggesting aortic valvular regurgitation (A). Electrocardiogram (ECG) revealed abnormally widened and notched QRS complexes indicating left bundle branch block (B).

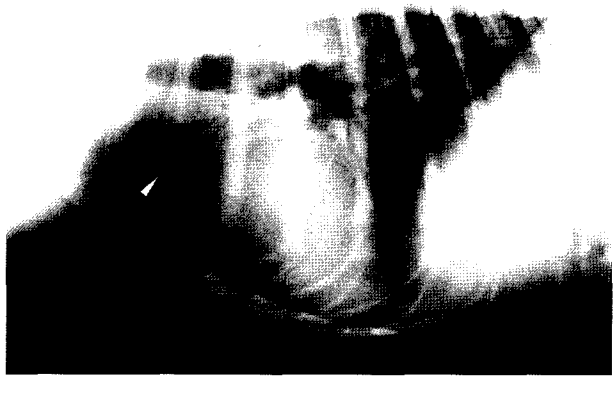

Fig 2. Thoracic radiography showed a marked aortic dilation (arrow head) and a loss of cranial cardiac waist. However the overall dimension of cardiac silhouette was within a normal range (VHS = 10.2).

valvular cusps (Fig 3A). Aortic valves were also mildly defected. Spectral and color Doppler echocardiographic studies revealed turbulent backward flow (bi-directional regurgitant flow) (Fig 3C & D). In this color Doppler study, the abnormal valvular structures were more clearly visible, and apparently separated from the aortic valve (Fig 3B). M-mode echocardiography taken after exercise revealed decreased left ventricular systolic and diastolic dimensions and volume and left ventricular ejection fraction (LVEF), fractional shortening (FS) and stroke volume (SV) after exercise (Table 2). Based on those findings, the case was diagnosed as congenital aortic valvular insufficiency caused by abnormal accessory valvular structures, which are complicated with supraventricular tachyarrhythmia.

The dog was released with prescription of 2 mg/kg diltiazem (Diltiazem, Nelson Pharmaceuticals) twice per day orally and recommendation of exercise restriction for reducing the frequency of tachyarrhythmia. Since the dog did not have any complications associated with heart failure (e.g. pulmonary edema),

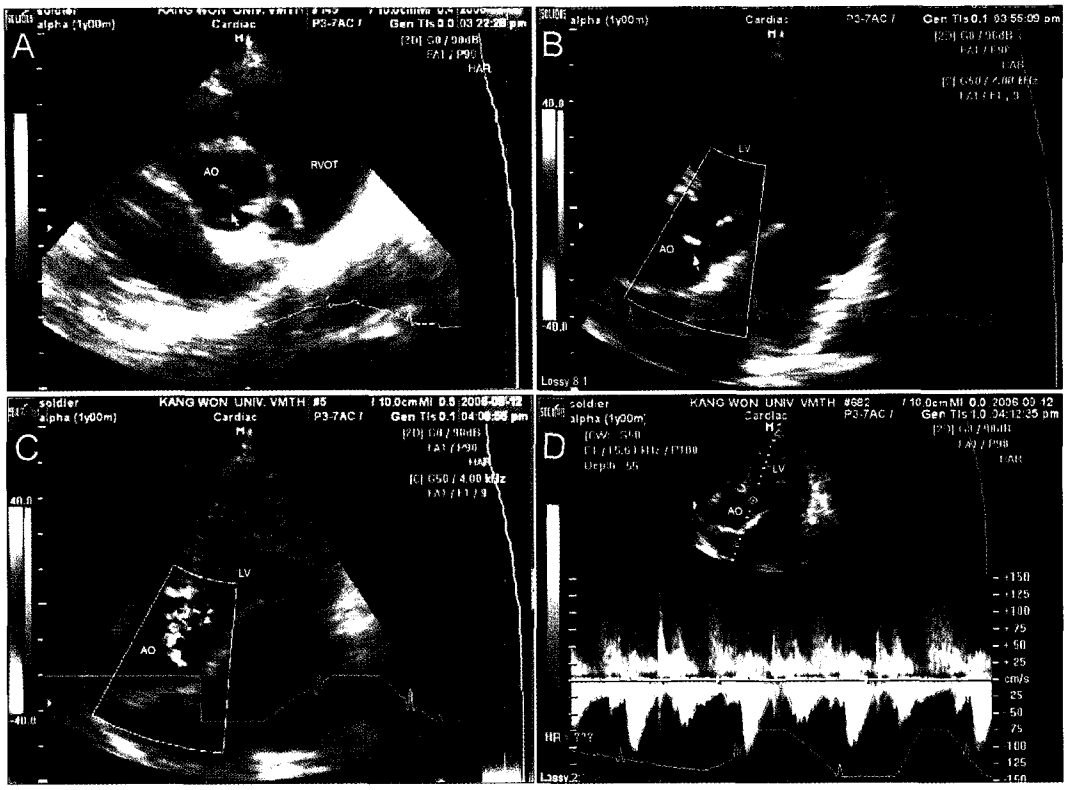

Fig 3. Echocardiography of this case. **A:** The 2-dimensional echocardiogram demonstrated abnormal valvular structures (arrow) moving independently from the aortic valvular excursion just above the aortic valvular cusps. **B:** Color Doppler echocardiogram demonstrated abnormal valvular leaflet (arrow) separated from the aortic valve. **C:** Color Doppler echocardiogram demonstrated a turbulent regurgitant flow at the end of systole. D: Continuous spectral Doppler echocardiogram demonstrated bi-directional regurgitant flow (Maximum velocity at 1.25 m/s).

Table 2. Echocardiographic dimensions of this case

Measurement	IVSd [a)] (mm)	LVIDd [a)] (mm)	LVPWd [a)] (mm)	IVSs [a)] (mm)	LVIDs [a)] (mm)	LVPWs [a)] (mm)	EF [a)] (%)	FS [a)] (%)	SV (ml)
Reference [b)]	8.7 ± 0.9	44.8 ± 2.9	7.9 ± 1.0	13.0 ± 1.5	31.0 ± 2.5	12.1 ± 1.2	20-70	28-40	
Before exercise	11.25 ± 0.95	46.65 ± 1.15	10.55 ± 0.25	19.45 ± 0.25	31.06 ± 0.70	13.4 ± 0.70	60.25 ± 4.41	32.18 ± 3.17	60.9 ± 7.94
After exercise	11.25 ± 1.45	38.79 ± 1.29	9.6 ± 0.2	14.05 ± 0.45	29.75 ± 0.25	11.39 ± 1.31	48.16 ± 4.12	23.9 ± 2.57	32.4 ± 5.97

a) IVSd: interventricular septal thickness at diastole, LVIDd: left ventricular internal dimension at diastole, LVPWd: left ventricular posterior wall thickness at diastole, IVSs: interventricular septal thickness at systole, LVIDs: left ventricular internal dimension at systole, LVPWs: left ventricular posterior wall thickness at systole, EF:% ejection fraction, FS:% fractional shortening.
b) Reference : Bonagura et al. (1985)

other cardiac medications (e.g. furosemide or enalapril) were not prescribed. We only recommended the owner to use a low salt diet (e.g., Hill's h/d). In the examination performed at one month after the first visit, the dog was clinically normal, but still intolerant to vigorous exercise. Abnormal valvular structures still existed at the same location. In addition, aortic insufficiency is still persisted without further deterioration. Diltiazem was discontinued since the tachyarrhythmia was apparently associated with exercise. The dog is currently alive and is restricted exercise with dietary management with Hill's h/d.

Discussion

AI can occur if aortic valvular leaflets incompletely close the aortic orifice during diastole (3). If a regurgitant area is as small as 20% of the valve, the left ventricular (LV) workload will be doubled. Increased LV workload will cause gradual LV dilation. For minimizing cardiac changes by increased LV workload, the heart will try to reduce the afterload and improve the forward flow by means of reflex peripheral dilation (3,10). Increased LV volume (preload) caused by aortic regurgitant flow can be compensated by increased left ventricular

compliance. However, if the aortic regurgitation is persisted, the left ventricle is gradually dilated by increased LV preload and subsequent LV stroke volume. AI also increases myocardial oxygen consumption, because the myocardial blow flow can be significantly reduced by insufficient blood supply to coronary arteries (3,10). Myocardial oxygen supply is decreased, but the demand is increased leading to ischemia, and failure of compensatory mechanisms eventually leading to LV failure. However, the significant cardiac remodeling associated with AI was not observed in this case. The younger age of the patient and the development of the rate-dependent tachyarrhythmia might be the reason why this dog did not have cardiac remodeling associated with AI.

The ratios of jet area to left ventricular outflow tract (LVOT) and jet height to LVOT measured by Doppler color flow mapping is widely used to determine the severity of aortic regurgitation (12). The ratio of jet area to LVOT is graded into severe regurgitation (> 60%), moderate (25-59%) and mild (< 25%), while that of jet height to LVOT is graded into severe (> 65%), moderate (47-64%) and mild (< 46%). This case had about 60% jet area and 65% jet height to LVOT, suggesting severe aortic regurgitation. With these evidences, increased pulse gradient, clearly audible diastolic murmur at the left base and aortic dilation on the thoracic radiography supported severe aortic valvular insufficiency in this dog.

Congenital causes of AI are most commonly associated with aortic valvular deformities (uni-, bi- quadri- and pentcuspid aortic valves) (4,14). Bi- and quadricuspid valvular deformities have been described in dogs (7,16). In humans, these aortic valvular deformities are often asymptomatic and incidentally identified on postmortem examination (1,5,6). Although in this case the acquired causes (e.g. bacterial/non-bacterial endocarditis) could not be absolutely ruled out, clinical presentation and diagnostic findings of this case were quite different from those of bacterial AI, since the main features of bacterial AI are the aggressive progression and the formation of vegetative lesion on the aortic valve. However, this case of dog had no vegetative lesion on the aortic valve and no medical history of infection. Instead, echocardiographic findings of this case were strongly suggested that the aortic insufficiency might be caused by abnormal valvular structures (congenital etiology).

Abnormal valvular structure has been rarely reported in veterinary and human medical literatures. An accessory mitral valve is probably the most common disease associated with abnormal valvular development (17). The incomplete separation of the mitral valve from the endocardial cushion tissue to the ventricular septum during embryogenesis is suggested for the cause of accessory mitral valve (17). Clinical consequences of accessory mitral valve are aortic stenosis (more common) or aortic insufficiency (3,13). In this case, we carefully examined mitral valvular annulus and valvular excursions to clarify whether the abnormal valvular structures are originated from mitral valve. However, any abnormalities were not found in the mitral annulus. Instead, careful review of echocardiographic images revealed the abnormal valvular structures were originated from the inside of the aortic arc, suggesting a congenital etiology.

The dog had clinical signs of forward heart failure after exercise (no increase in blood pressure after exercise). Because the dog had tachyarrhythmias in supraventricular origin only after exercise, we were not able to clearly define whether the forward failure is resulted from the tachyarrhythmias or the forward flow depletion by AI. Furthermore, we could not detect any ECG abnormalities in one hour digital ECG recordings at rest, even after we discontinued diltiazem administration. Therefore, the tachyarrhythmia seemed to be associated with cardiac changes (increased sympathetic tone) caused by aortic regurgitation. Relationship between AI and blood pressure has been reported previously (18). Acute onset of AI can lower markedly the mean arterial blood pressure, as well as the aortic diastolic pressure (18). No significant elevation of systolic blood pressure after exercise in this dog, suggesting that the cause for exercise intolerance might be due to the loss of forward blood flow by aortic regurgitation. Tachyarrhythmias may arise from the increased chronotropic β-adrenergic activities for accelerating heart rate. Although the dog showed mild aortic regurgitation on the echocardiography taken at rest, the dog showed severer aortic regurgitation after vigorous exercise with significant reduction of stroke volume.

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래브라도 리트리버종 개의 비정상 판막 구조에 의한 선천성 대동맥 판막 부전

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요 약 : 10 개월령 수컷 래브라도 리트리버종 개가 특히 과도한 운동 이후에 나타나는 운동 불내성으로 내원하였다. 신체검사에서 좌측 심첨부와 심저부에서 제1심음의 분열음과 grade III/IV의 이완기 역류성 잡음이 청진되었다. 심전도 검사에서 휴식기에는 정상 동박동을 나타낸 반면, 운동 후에는 각 차단과 함께 심실상성 빈맥이 나타났다. 흉부 방사선에서는 정상 심장크기(VHS 10.2)이나, 확장된 상행 대동맥이 관찰되었다. 심장초음파에서는 대동맥 판막상단부위에서 기시된 비정상적인 판막성 구조물에 의해 대동맥 역류증이 관찰되었고 그 결과 좌심실 박출율(LVEF)이 감소되는 소견을 보였다. 상기의 결과를 토대로 본 증례를 비정상의 판막 구조물에 의한 선천성 대동맥 역류증으로 진단하였다. 환자에게 diltiazem을 처방하였으며, 운동제한을 시켰다. 본 증례는 매우 드물게 보고되는 대동맥 판막 기형이다.

주요어 : 래브라도 리트리버, 개, 대동맥 판막 기형, 판막 부전