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Echocardiographic Diagnosis of Subaortic Stenosis with Severe Deformation of Mitral Valve Apparatus in a Dog

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Abstract : A 1-year-old castrated male Schnauzer dog was presented with heart murmur. Auscultation revealed systolic murmur located at the left heart base (grade 5/6). There were no remarkable findings on thoracic radiographs. Two-dimensional echocardiography revealed subaortic tunnel-like obstruction at the entrance to the left ventricular outflow tract. Anterior mitral valve leaflet appeared to be tethered to septum with minimal motion. Chordae tendineae was abnormally thickened. Color Doppler analysis revealed turbulent flow starting below the aortic valve. Mitral regurgitation was presented during systole. Spectral Doppler recordings revealed high velocity flow through the aorta and mitral regurgitation. Based on echocardiographic examination, the dog was diagnosed with subaortic stenosis concurrent with mitral dysplasia. The patient was medicated with β-blocker and diuretics. It has been doing well without apparent clinical signs at 2 year after the diagnosis.

Key words **:** congenital heart defects, subaortic stenosis, mitral dysplasia, dog.

Introduction

Subaortic stenosis (SAS) is one of the most common forms of congenital heart disease in dogs. SAS is most common type of aortic stenosis and most frequently described as a single disease. However, in some dogs, it coexists with other congenital cardiac defects (4,12,16). Mitral dysplasia (MD) has been the most common congenital cardiac defect found to be associated with SAS (7).

According to retrospective studies on congenital heart defect, multiple heart defects account for less than 10% of cases (12,22). Associated congenital cardiac defects are challenging to diagnose because typical findings of clinical signs and thoracic radiography may confuse the findings of other lesions. Echocardiography is a noninvasive diagnostic tool and useful for diagnosing these complex cardiac abnormalities in small animals. The aim of this case study is to describe the diagnostic features of a rare case of SAS concurrent with MD in a dog.

Case

A 1 year-old castrated male Schnauzer dog was presented with a heart murmur. Auscultation revealed systolic murmur located at the left heart base (grade 5/6). Its systolic blood pressure was 100 mmHg. Electrocardiography (ECG) revealed tall R wave, depression of ST segment, and left axis deviation (Fig 1). Hematology and biochemical findings were within normal limits except that platelet count at 167 was slightly lower than that of reference range (200 to 500).

Thoracic radiographs demonstrated no particular abnormalities on cardiac silhouette. There was mild interstitial pattern in the caudal lung lobe (Fig 2). Therefore, mild pulmonary edema was suspected.

Two-dimensional echocardiography showed abnormal shape and thickening of anterial mitral valve with hyperechoic areas in enlarged papillary muscles (Fig 4). Chordae tendineae also appeared to be thickened (Fig 4). On real-time image, anterior mitral valve showed abnormal motion with subaortic tunnel-like obstruction. Color Doppler analysis identified turbulent flow starting below the aortic valve during systole (Fig 3). Mitral regurgitation flow into the left atrium was also presented during systole (Fig 4). Spectral Doppler of mitral inflow confirmed that early diastolic inflow velocity was at 1.28 m/s, which was higher than the reference range (0.62 to 0.84 m/s). E:A ratio was 3.52, which was higher than the reference range (1.16 to 2.1). Aortic flow velocity was at 5.2 m/s, which was much higher than the reference range (< 1.7 m/s). Mitral regurgitant maximal flow velocity was at 7.4 m/s. On

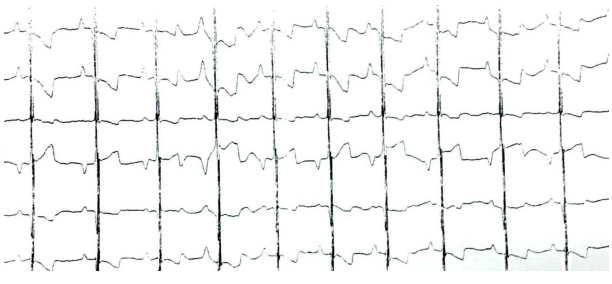

Fig 1. Electrocardiogram of the patient revealing tall R wave, depression of ST segment, and left axis deviation.

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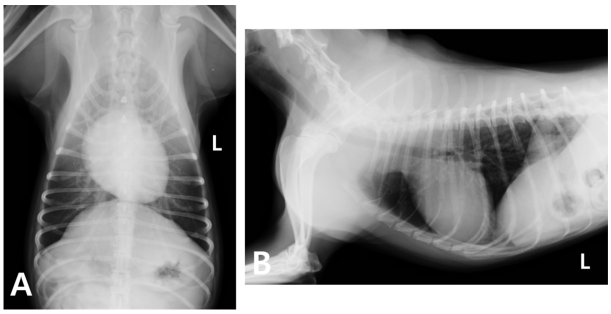

Fig 2. Thoracic radiography of the patient showing mild interstitial pattern in the caudal lung lobe. There was no particular abnormality on cardiac silhouette.

M-mode echocardiography, left ventricular wall and interventricular septum were thickened during both systole and diastole (LVPWd: 11.3 mm, reference range: 5.3-6.5 mm; LVPWs: 15.9 mm, reference range: 8.8-10.4 mm; IVSd: 10.4 mm, reference range: 6.7-8.1 mm; IVSs: 13 mm, reference range: 10.0-11.7 mm). LV diameter was under normal range (21.6 mm in diastole, reference range: 23.-27.4 mm; 9.9 mm in systole, reference range: 13.3-16.3 mm). Fractional shortening (47%) was within normal range (30-50%). The thickened anterial mitral valve leaflet showed mild systolic anterior motion (SAM) (Fig 3). The ratio of LA to AO was 1.26 (reference range: < 1.3).

Based on these findings, the dog was diagnosed with subaortic stenosis of both fixed and dynamic forms concurrent with severe deformation of mitral valve apparatus. The patient was prescribed with β-blocker (Carvedilol, 0.2 mg/kg, bid, PO) and diuretics (furosemide, 1 mg/kg, bid, PO). At one year follow-up, the patient is in good clinical condition.

Discussion

SAS has been reported most commonly in large breed dogs such as Newfoundland, Golden retriever, Rottweiler, and German Shepherd (7). Most owners of SAS dogs report normal and apparently healthy individuals when presenting dogs for routine examination (7,8). Physical examination findings in dogs with SAS are often unremarkable except that the dogs present systolic ejection murmur that is usually the loudest in the left basilar region (14,6). The murmur intensity is roughly correlated with lesion severity. Aortic regurgitation can produce a diastolic murmur at the left base. The murmur may be inaudible (8,23). More severely affected animals may present exertional fatigue, syncope, or rarely signs referable to congestive left heart failure. Sudden death without premonitory signs is common (3).

MD is particularly common in cats. It is a rare congenital heart disease in dogs. Breeds predisposed for MD include Great Danes, German Shepherds, Bull Terriers, Rottweilers, and Golden Retrievers (11,20). Dogs and cats with MD most commonly present respiratory abnormalities that occur secondary to left heart failure (6,14). The hallmark of mitral insufficiency is a holosystolic murmur heard best over the valve area (23). On previous case reports of dog with SAS and MD, loud systolic ejection murmur has been noted in all dogs. The murmur is heard the loudest at the left heart base (16,18). Clinical signs of dog with SAS and MD vary widely in severity. One dog with loud systolic ejection murmur can be alert and playful, while another dog may present a history of fatigue and exercise intolerance. The present case did not show clinical signs except the presence of heart murmur. The systolic ejection murmur was the loudest at the left heart base and the intensity of the murmur was of 5/6 grade.

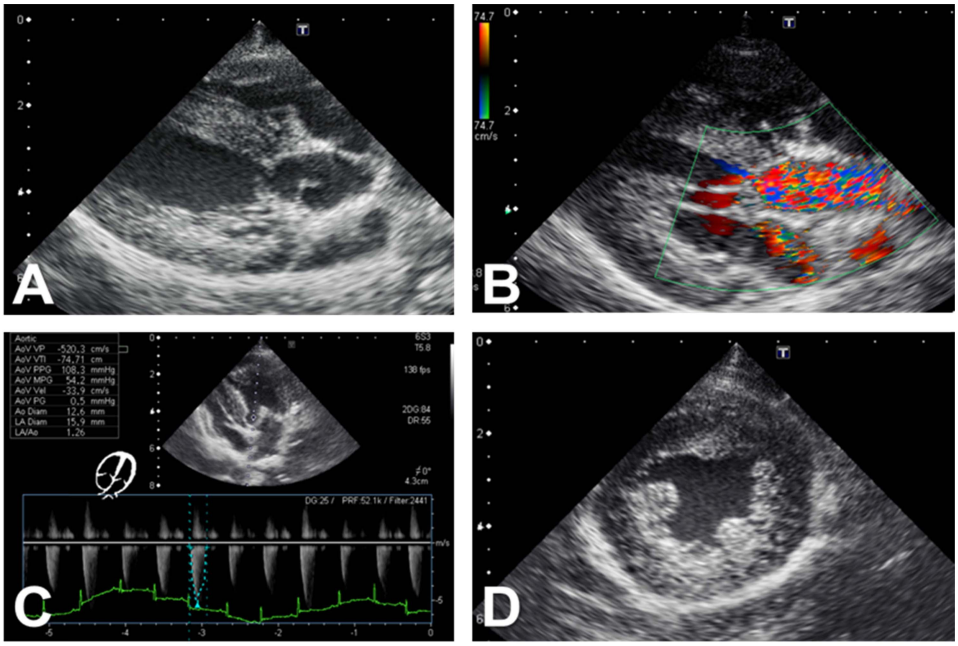

Fig 3. Echocardiographic images of the patient. A, Subaortic tunnel-like obstruction was identified and anterial mitral valve appeared to be protruding into LVOT in systole. B, turbulent flow starting below the aortic valve was shown on Color Doppler mode. C, high velocity of aortic flow (5.2 m/s) was confirmed. D, right parasternal short axis view at the papillary muscle level showing hyperechoic area within thickened papillary muscles.

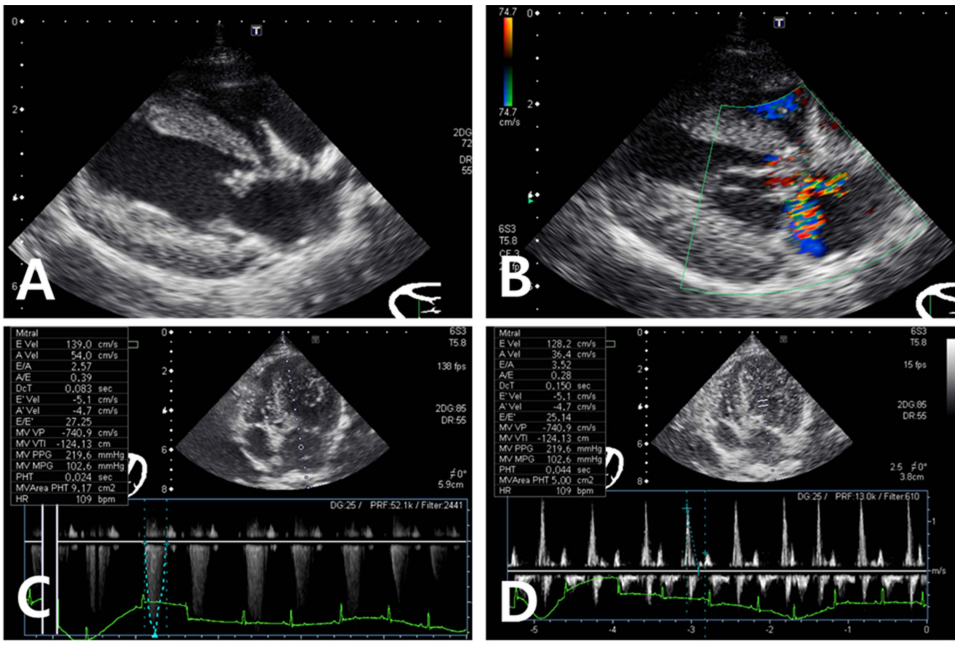

Fig 4. Echocardiographic images of the patient. A, anterial mitral valve appeared to be very thick and chordae tendineae was also thickened. B, mitral regurgitation flow into the left atrium during systole. C, high mitral inflow velocity (7.4 m/s) was identified. D, increased early diastolic inflow velocity was confirmed (1.28 m/s) with E:A ratio of 3.52.

In dogs with SAS, ECG is often normal, although evidence of LV hypertrophy (left axis deviation) or enlargement (tall complexes) can be present (6,23). Depression of the ST segment in lead 2 and aVF can occur from secondary myocardial ischemia. ECG finding in dogs with MD include prolonged P waves and a tall R wave in lead 2 (11,13). Sinus tachycardia, supraventricular premature contractions, supraventricular tachycardia, and atrial fibrillation are commonly observed in dogs with MD (8). In this case, tall R wave, depression of ST segment, and left axis deviation were observed. These findings are suggestive of LV enlargement.

In patients with mild SAS, thoracic radiography finding is typically normal. Only apparent mild cardiomegaly may be found (6,23). Poststenotic dilation in the ascending aorta can cause enlargement of the aortic root and cranial mediastinal widening in severely affected dogs (6,23). In dogs affected by MD, the extent of changes noted on thoracic radiographs depends on the severity of the mitral valve lesion (8). Animals with severe disease have severe left atrial enlargement and moderate-to-severe left ventricular enlargement. On case studies of dogs with SAS and MD, thoracic radiographic features include LA and LV enlargement and pulmonary edema (16,18). In one dog, aortic bulging has been revealed (18). In the present study, thoracic radiographs demonstrated no particular abnormalities on the cardiac silhouette. Aortic bulging was not observed and the LA was not considered to be enlarged, indicating that severe cardiac change did not develop.

SAS can be divided into fixed form and dynamic form. In some cases, both forms can occur together (20). Fibrous ridge (completely or partially) can encircle the left ventricular outflow tract (LVOT) below the aortic valve, causing a fixed obstruction (17,19). Dynamic form is caused by the septal leaflet of the mitral valve protruding into the LVOT (7,15,21). Fixed and dynamic obstruction of the LVOT has been seen to coexist with anomalies of the mitral valve complex and concentric hypertrophy of the left ventricle (5,6). In this case, both fibrous ridge and anterial mitral valve protruding into LVOT in systole were identified.

The spectrum of SAS severity can vary widely. There are three grades of SAS lesions based on postmortem studies in Newfoundland puppies (19). The mildest form (grade 1) consists of small, raised nodule of thickened endocardium on the interventricular septum below the aortic valve. Grade 2 lesions consists of a narrow ridge of whitish, thickened endocardium that partially encircles the LVOT below the aortic valve. Dogs with grade 3 have a complete fibrous ring around the LVOT, causing elongated tunnel-like obstruction. In dogs with SAS, the severity of the stenosis determines the degree of left ventricular pressure overload and the resulting concentric hypertrophy (1,6,23). In the present case, the dog had a fibrous ring around the LVOT. High velocity of aortic flow and turbulent flow starting below the aortic valve were identified. In addition, moderate to severe LV concentric hypertrophy was detected. These might be caused by severe SAS.

Many animals with SAS also have aortic or mitral valve regurgitation because of related malformations or secondary changes (23). In previous studies, stenotic jet due to SAS often causes aortic valve thickening and ischemic fibrosis of left ventricular papillary muscles or subendocardium (6). In the dog of this case report, increased echogenicity of the subendocardial region and enlarged papillary muscles suggestive of myocardial fibrosis were found. Mitral regurgitation was also identified. However, in this case, mitral regurgitation was most likely caused by MD rather than by SAS.

MD is defined as an abnormally formed mitral valve resulting in regurgitation (1,2,8,14,23). Malformations of the mitral valve apparatus include shortened or overly elongated

chordae tendineae, direct attachment of the valve cusp to a papillary muscle, thickened or cleft or shortened valve cusps, and prolapse of valve leaflets (11,23). In this case, severe thickening and abnormal shape of anterial mitral valve, thickened chordae tendineae, and abnormal movement of mitral valve were identified.

Mitral insufficiency is a very common echocardiographic abnormality detected with MD (10). Cardiac changes associated with MD are similar to those seen in mitral insufficiencies secondary to acquired valvular lesions (2). For this reason, MD is associated with dilation of LV and/or LA (8). The left ventricular enlargement is characterized by an increase in end-diastolic diameter but normal LV wall thicknesses (8,22). The LA is larger than normal and is larger than the LV. LV shortening fraction ranges from 15% to 45%. In the dog of the present study, high mitral regurgitation flow (peak velocity of 7.4 m/s), reduced diastolic function of LV, and LV hypertrophy were identified. However, LA and LV dilation were not found.

Various survival time of dogs with SAS and MD have been reported. One study reported that the patient with SAS and MD died suddenly in the first 4 months of life (18). In another study, the patient lived longer than 1 year without remarkable finding in thoracic radiographs or echocardiography. Findings indicating left atrial dilation were not observed either (24). One study of 195 untreated dogs with SAS has reported that the prognosis of dogs with SAS is worse as the severity of stenosis is increased (12). Mild malformations of the mitral valve that are common in dogs with SAS are usually not functionally important. However, SAS dogs with moderate to severe MD usually die from left congestive heart failure at an early age (9). In the present case, any finding indicating left atrial dilation was not found in thoracic radiography or echocardiography. In addition, the patient was 17 months old at the time of presentation. It was in good condition without any treatment. Based on these facts, LA dilation might be associated with the prognosis of patient.

In conclusion, this case report described a rare case of complex congenital cardiac anomalies in a dog diagnosed by echocardiography. The dog was diagnosed as severe SAS with MD and secondary changes including subendocardial region and papillary muscles. Although the patient had severe stenosis at the subaortic region, LA dilation was not identified. There is no clinical sign by 2 years.

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