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<Case Report>

Pulmonic stenosis with atrial septal defect in a Siamese cat

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Abstract: A 6-month-old mature intact female Siamese cat presented with exertional dyspnea. Diagnostic studies revealed pleural effusion, grade 4/6 left basal systolic murmur, deep S-wave in electrocardiograph leads I, II, and III, cardiomegaly with pleural effusion on radiography, pulmonic systolic (~5.8 m/sec) and tricuspid (3.6 m/sec) regurgitant jets, atrial septal defect, and a hypoplastic right outflow tract. Based on these results, the case was diagnosed as pulmonic stenosis with atrial septal defect. To the best of our knowledge, this is the first case report describing pulmonic stenosis with atrial septal defect in a cat in Korea.

Keywords: right ventricular outflow tract obstruction, pulmonic stenosis, pulmonic valve dysplasia, congenital heart defect, cat

Pulmonic stenosis (PS) is a congenital heart defect in which the right ventricular outflow tract (RVOT) is obstructed by either/both pulmonic valvular deformities or/and narrowing of RVOT (*i.e.*, hypoplastic RVOT) [7, 9]. Although the most PS cases in human and veterinary literatures are isolated defect, the some PS cases are often occurred with other defects such as atrial septal defect (ASD), ventricular septal defect and tetralogy of Fallot [7]. The PS is largely divided into subvalvular, valvular or supravalvular, depending on the location of obstruction [4, 7]. Clinical consequences of symptomatic PS include left basal systolic murmur and right side heart failure accompanied with marked tricuspid insufficiency [9]. One recent retrospective study found the prevalence of PS was ~10% (16/162) in feline congenital heart defects [10]. ASDs are abnormal communications between right and left atria and are generally occurred from congenital heart defects in which the atrial septum formed incompletely. The secondary ASDs can be also occurred from secondary to severe pulmonary or left atrial (LA) dilation [7]. This case report describes a rare case of PS by hypoplastic RVOT with secondary ASD in a Siamese cat.

A 6-month-old intact female Siamese cat (2.3 kg of body weight) was presented with primary complaints of loud heart murmur, exertional dyspnea and exercise intolerance. In physical examination, the cat had grade IV/VI left basal systolic murmur. The mucosa in lips was pink and moist. Systolic blood pressure measured by a Doppler detector (811B; Parks Medical Electronics, USA) was 100 mmHg. Electrocardiography (ECG) studies revealed normal sinus rhythm with right ventricular enlargement (presence deep S-wave in lead I, II and III) and sinus bradycardia (80–95 beats per min; Fig. 1). Complete blood cell count and serum chemistry profiles have no significant abnormalities.

Thoracic radiography revealed marked pleural effusion with loss of cardiac silhouette and retracted caudal lung lobes. Thoracocentesis was performed at right side of chest and removed 350 mL blood tinged fluid. Cytological examination found red blood cells and mononuclear cells, suggesting modified transudate. Further thoracic radiography after removal of pleural effusion revealed cardiomegaly (vetebral heart scale 11.2) with right cardiac deviation and under-circulated lung fields on ventrodorsal projection and cardiomegaly (4 intercostal spaces) with increased cardiac contact to the sternum and dorsal displacement of trachea on right lateral projection, suggesting right cardiac enlargement pattern (Fig. 2A and B). The 2-dimensional (2-D) echocardiography in right parasternal long axis view revealed right atrial (RA) dilation (Fig. 3A), ASD (Fig. 3A) and interventricular septal flattening to left ventricle (LV; Fig. 3A), turbulent jets at tricuspid valvular area and right pulmonary artery (Fig. 3B). Further 2-D and M-mode echocardiography in right parasternal short axis view at papillary muscle level revealed marked interventricular septal flattening to LV (Fig. 3C), turbulent jets at right ventricle (RV; Fig. 3C) and hypertrophied RV and inter-

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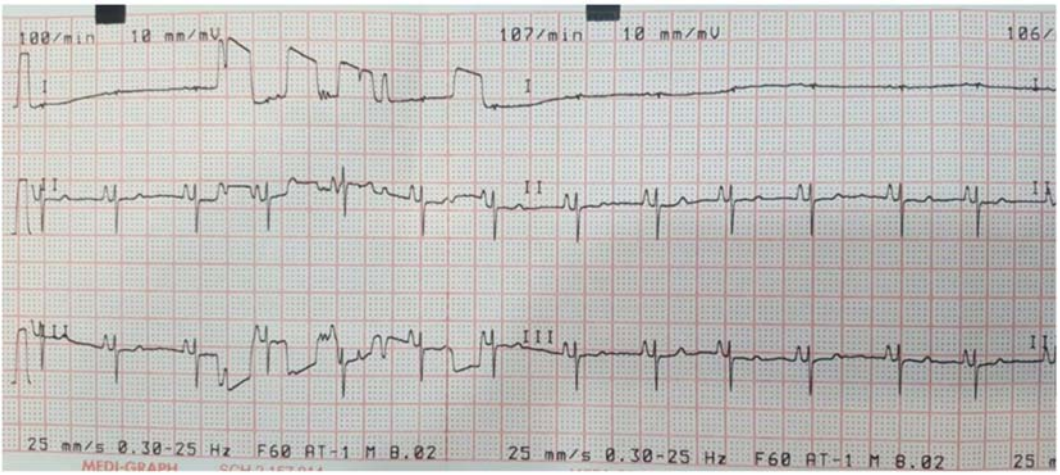

Fig. 1. Electrocardiography studies revealed normal sinus rhythm with right ventricular enlargement (presence deep S-wave in lead I, II and III) and sinus bradycardia (80–95 beats per min).

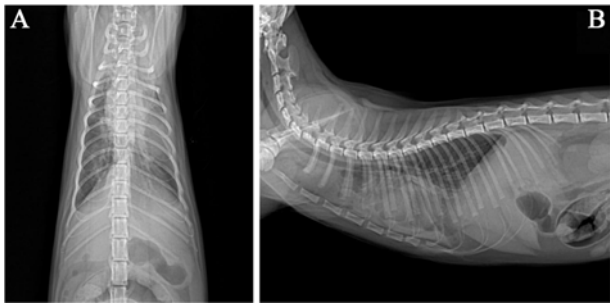

Fig. 2. Thoracic radiography of this case. Thoracic radiography after thoracocentesis revealed cardiomegaly (vetebral heart scale 11.2) with right cardiac deviation and under-circulated lung fields on ventrodorsal projection (A) and cardiomegaly (4 intercostal spaces) with increased cardiac contact to the sternum and dorsal displacement of trachea on right lateral projection (B), suggesting right cardiac enlargement pattern.

ventricular septum (Fig. 3D). There was marked narrowing of RVOT (aortic to pulmonary ratio 2.1), although the pulmonic valve itself was intact (Fig. 4A and B). The RVOT from infundibulum to main pulmonary artery (MPA) in this cat was hypoplastic. Continuous wave Doppler echocardiographic studies revealed turbulent systolic jet flow with peak velocity of 5.8 m/sec (pressure gradient between RV and RVOT 136 mmHg), indicating severe PS (Fig. 4C). Further the 2-D echocardiography in left apical long axis view of 4 chambers found marked RA dilation and tricuspid regurgitation jets with peak velocity of 3.6 m/sec (pressure gradient between RA and RV 52 mmHg; Fig. 4D). Bubble study with agitated saline confirmed mild right-to-left shunt flow at the end of systole. Based on clinical signs and diagnostic findings, the case was diagnosed as PS with right-to-left ASD.

The cat was treated with medications including torsemide (Torsem tablet 0.3 mg/kg, q12h, per orally [PO]; Hanmi Pharm, Korea), atenolol (Tenormin 1 mg/kg, q12h, PO; AstraZeneca, UK), enalapril maleaste (Enalapril tablet 0.5 mg/kg, q24h, PO; Daewon Pharm, Korea) and pimobendan (Vetmedin 0.3 mg/kg, q12h, PO; Boehringer Ingelheim, Germany). Clinical signs were improved after medical treatment. No further fluid retention has found on thoracic radiography after 1, 2 and 4 weeks after medical treatment. Surgical correction was not performed, because of owner's refusal. The cat is currently survived with medical treatment and is regularly monitored. To authors' best knowledge, this is the first case report describing PS with ASD in a cat in Korea.

The most common type of PS in cats was valvular malformation which was characterised by thickening of the valve leaflets causing partial obstruction of RV outflow to the lower pulmonary vasculatures [10]. Due to the increased resistance to blood flow to lower pulmonary vasculatures, RV pressure at systole could elevate and cause to secondary right ventricular hypertrophy and dilation. The RV dilation and hypertrophy could further induce secondary tricuspid valvular insufficiency along with right atrial (RA) dilation, causing right-sided heart failure. Although mild PS in cats is usually asymptomatic even at adulthood, more advanced PS might cause clinical signs associated with right-sided heart failure (*e.g.* ascites, pleural effusion) [8]. In this case of cat, the clinical signs and ECG findings clearly indicated classic PS and right-sided heart diseases. On echocardiographic evaluation, there was no commissural fusion on pulmonic valve. Therefore, the valvular PS was ruled out. The RVOT from the valvular infundibulum to MPA was underdeveloped (much narrower), indicating supravalvular PS.

Balloon dilation is interventional method for widening stenotic valvular orifice and has been successfully applied in dogs and cats with clinical PS [2, 3, 5]. Generally, balloon dilation is indicated for cats with clinical signs of right-sided heart failure or PS with peak velocity of > 4.0 m/sec (pressure gradient > 64 mmHg [8], although this technique is not effective for PS with annular hypoplasia. In this case of cat,

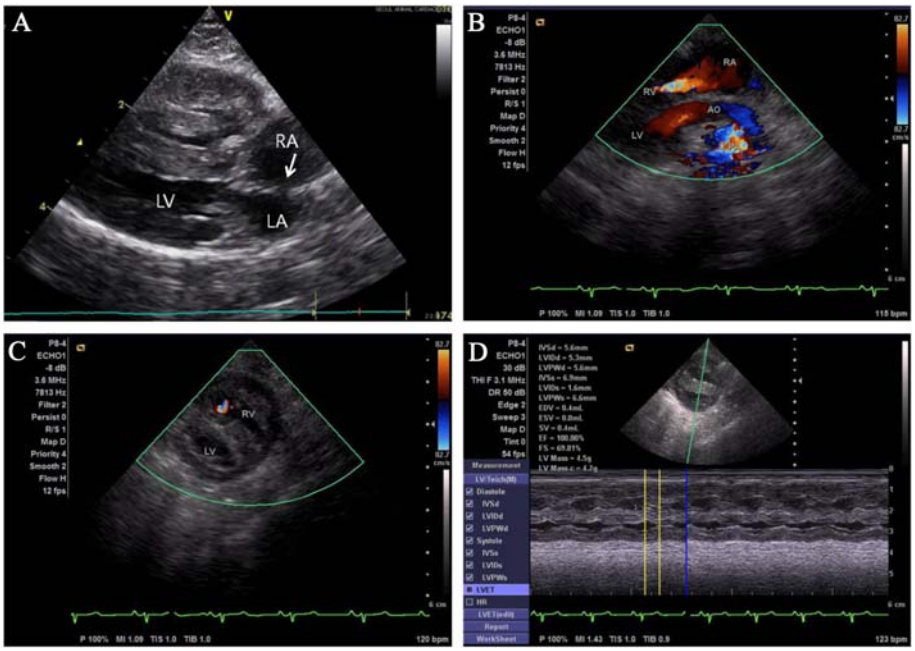

Fig. 3. The echocardiography of this case. (A) The 2-dimensional (2-D) echocardiography in right parasternal long axis view revealed right atrial (RA) dilation, atrial septal defect (arrow) and ventricular septal flattening to left ventricle. (B) Color Doppler echocardiography in right parasternal long axis view revealed turbulent jets at tricuspid valvular area and right pulmonary artery. (C) The 2-D echocardiography in right parasternal short axis view at papillary muscle level revealed marked ventricular septal flattening to left ventricle and turbulent jets at right ventricle (RV). (D) M-mode echocardiography in right parasternal short axis view at papillary muscle level revealed hypertrophied right ventricular and interventricular septum.

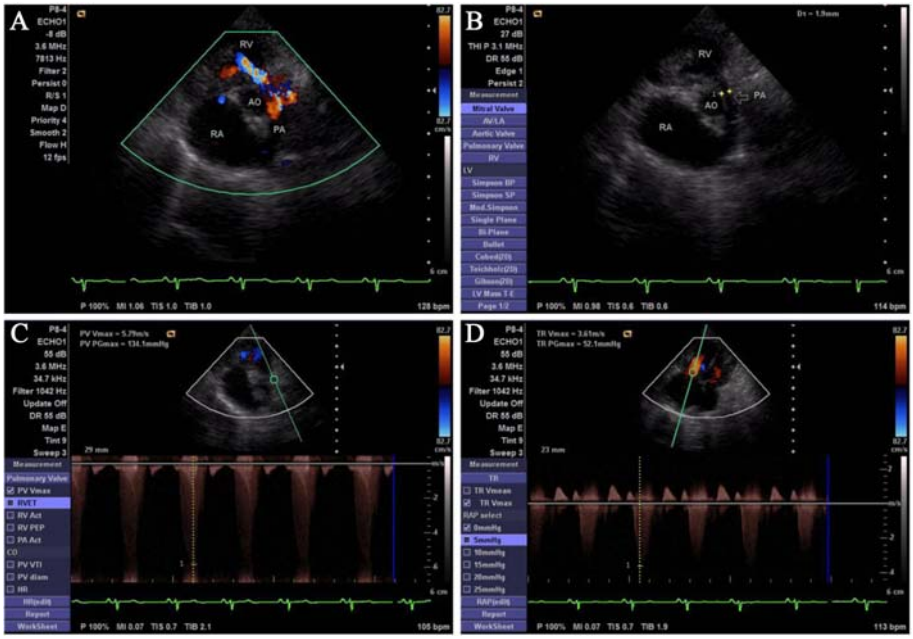

Fig. 4. The echocardiography of this case. (A and B) The 2-D and color Doppler echocardiography in right parasternal short axis of pulmonary artery revealed marked narrowing of right ventricular outflow tract (RVOT; aortic to pulmonary ratio 2.1), although the pulmonic valve itself was intact. The RVOT from infundibulum to main pulmonary artery in this cat was hypoplastic. (C) Color Doppler echocardiography in right parasternal short axis of pulmonary artery revealed turbulent systolic jet flow with peak velocity of 5.8 m/sec (pressure gradient between RV and RVOT 136 mmHg), indicating severe pulmonic stenosis. (D) Continuous wave Doppler echocardiography in left apical long axis view of 4 chambers found marked RA dilation and tricuspid regurgitation jets with peak velocity of 3.6 m/sec (pressure gradient between RA and RV 52 mmHg).

the balloon dilation was not performed due to poor outcome of surgical correction and owners' refusal, since the PS was occurred from annular hypoplasia of RVOT.

The etiology of ASD in this cat was unclear. It might be due to congenital heart defect or secondary to increased RA pressure from PS. The most common type of ASD in cats is ostium primum ASD, characterized by failure to fuse the superior and inferior endocardial cushions. This type of ASD often caused large ASD with cleft of mitral valve leaflets. However, in this case of cat, the mitral valve was intact and the shunt flow across ASD was minimal, suggesting the type of ASD might be patent foramen ovale (PFO) which is benign type of ASD occurred from failure of foramen ovale closure. In this cat, increased RA pressure even after birth (due to severe PS) might cause the foramen ovale not to close. Although the shunt flow was directed from RA to LA, there was no evidence of cyanosis in physical and laboratory examinations, because the shunt flow was minimally entered into LA at the end of systole.

Therapeutic strategies for this case were directed to minimize fluid retention (*i.e.*, torsemide), to prevent neruohormonal responses (*i.e.*, enalapril and atenolol), and to improve ventricular relaxation using a β-blocker (*i.e.*, atenolol) and pimbendan. Unfortunately, no study has yet evaluated the potency of torsemide in cats, although it has more potent diuretic effect in dogs [6]. Anecdotal reports have found the potency of torsemide in cats was similar to that of dogs. However, close monitoring and proper dosing based on diagnostic imaging studies and clinical signs should be retained to minimize the occurrence of azotemia by over-dosing. Lusitropic effect (*i.e.*, improvement of ventricular relaxation) of pimobendan has been recently reported [1]. β-blockers have proven to be effective in left ventricular outflow obstruction in cats with hypertrophic cardiomyopathy. Because the cat of this case has marked right outflow tract obstruction by severe PS, authors added atenolol to improve RV flow to pulmonary circulation.

In conclusion, this case report describes a rare case of supravalvular PS with ADS in a cat. To the best of our knowledge, this is the first case report describing PS of cat in Korea.

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