Ventricular septal defect in an Abyssinian cat

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Abstract: A 2-month-old female Abyssinian cat was presented with a severe ascites, cyanosis, and exercise intolerance. Diagnostic studies revealed V/VI holosystolic murmur, sinus tachycardia, generalized cardiomegaly with marked left atrial enlargement and shunt flow between left and right ventricles. Doppler study showed bi-directional shunts in rest and right-to-left shunt after exercise. Based on clinical signs and diagnostic findings, the cat was diagnosed as a reversed ventricular septal defect. The cat was treated with furosemide, nitroglycerine, dobutamine and oxygen supplement. Despite initial improvement of clinical signs after initiation of medical treatment, the cat died of sudden cardiac arrest. Necropsy revealed a perimembranous ventricular septal defect.

Keywords: Abyssinian cat, congenital heart disease, ventricular septal defect

Ventricular septal defect (VSD) is an orifice in the inter-ventricular septum that allows blood to flow from one ventricle to the other [1, 2]. If the defect is small, there is no hemodynamic consequence, since the pulmonary flow is increased minimally. However, if the defect is large, it causes a significant hemodynamic change. Initially, systemic vascular resistance exceeds the pulmonary vascular resistance (PVR), so that left-to-right shunting is more common. However, over time, PVR may increase due to pulmonary over-circulation and pulmonary vascular changes, resulting in pulmonary hypertension. This may then result in right-to-left (R-L) shunting across the defect (Eisenmenger's physiology) [2]. Clinically, VSDs are classified based on the anatomical location of the defect (e.g., perimembranous, supracristal, muscular or posterior). VSDs are also classified based on where they enter the right ventricle (e.g., sub-pulmonic, into RV inflow, etc.). More than two-thirds of VSDs have perimembranous intraventricular defects [8]. Similar to atrial septal defects (ASDs), most VSDs are either sporadic or isolated, although they do occur as a part of other defects (e.g., tetralogy of Fallot, and atrioventricular canal defect). Mutations in transcription factors associated with cardiomyogenesis (e.g. NKX2.5, TBX20, GATA4) have been found in isolated VSDs [4, 6, 7, 10].

In dogs, the frequency of VSD is much lower (1.1 per 1,000 dogs examined) than that in humans [6]. But in cats, VSD is the first or second most common congenital heart disease, although the actual prevalence rate has not been clearly documented [12]. Most VSDs in these animals are sporadic, although one familial VSD has been identified in English Springer Spaniels [3]. In this dog breed, autosomal dominant inheritance with incomplete penetrance or polygenic trait was suggested [3]. However, no breed or familial predisposition has yet been found in cats [8]. This case report described a rare case of reversed VSD in an Abyssinian cat.

Case Description

A 2-month-old female Abyssinian cat (700 g of body weight) was presented at the Veterinary Teaching Hospital, Kangwon National University with clinical signs of exertional dyspnea, orthopnea, exercise intolerance, and cyanosis. On thoracic auscultation, a grade V/VI holosystolic and mild diastolic regurgitant murmur were detected over the left and right apex area with a precordial thrill (Fig. 1). Femoral pulse was weak and fast. Electrocardiographic studies showed sinus tachycardia with left ventricular enlargement (QRS in lead II: 1.3 mV; Fig. 2). No remarkable abnormalities were observed in routine hematology (Hematocrit, 29%; total red blood cell, 6.9 × 10¹²/µl) and blood chemistry.

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except mild hypoxia ($P_{aO_2}$: 72 mm Hg; reference range, 80 to 104 mm Hg) measured by i-STAT (Abbott, USA) and increased hepatic enzymes (alkaline phosphatase 114 IU/L, alanine transaminase 1,000 IU/L).

Thoracic radiography revealed generalized cardiomegaly (VHS 11.6, reference range: 6.7-8.1) with a marked right atrial enlargement, pulmonary undercirculation, pleural and abdominal effusions and hepatomegaly (Fig. 3). Two-dimensional echocardiography taken at right parasternal long-axis view of the left ventricle (LV) and aorta revealed a region of discontinuity between the upper portion of the interventricular septum and the base of the aorta (Fig. 4). Further echocardiographic studies found pericardial effusion, marked LV dilation, moderate RV hypertrophy and bi-atrial enlargement. Doppler echocardiography showed bi-directional turbulent flows in the septal defect (Fig. 5). Continuous Doppler echocardiography revealed the velocity of shunt flow 3-4 m/s in both directions, suggesting the size of VSD might be medium to large. Based on these findings, the case was diagnosed as reversed VSD complicated with pericardial, pleural and abdominal effusions.

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Fig. 1. Phonocardiogram of this case. The heart sound was recorded at the left apex. Because the shunt direction was the right-to-left, the holosystolic murmur was more clearly detected at the left apex although the right apical holosystolic murmur was generally detected in the left-to-right ventricular septal defect. The cat also had mild diastolic regurgitant murmur.

Fig. 2. Electrocardiogram of this case. The heart rhythm was tachycardic but was originated from the sinus node. There was a marked increase of QRS amplitude (1.3 mV), suggesting left ventricular enlargement. No remarkable abnormalities were observed in 1 h-event recordings in this cat.