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<Case Report>

Hypertrophic obstructive cardiomyopathy in a Yorkshire Terrier

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Abstract: An 11-year-old, castrated male dog presented with a 3-month history of cough and depression. Auscultation revealed systolic murmur and thoracic radiographs showing enlargement of both the atrium and left ventricle. Echocardiography showed thickened mitral valve and moderate-to-severe left atrial enlargement. Additionally, M-mode echocardiography showed symmetric left ventricular wall thickening and systolic anterior motion of the mitral valve, while Doppler imaging revealed high velocity turbulent flow through the left ventricular outflow tract. Based on echocardiography, this case was diagnosed with hypertrophic obstructive cardiomyopathy. After 5 months, the dog was clinically static in radiography and echocardiography.

Keywords: dogs, echocardiography, hypertrophic cardiomyopathy, mitral valve

Hypertrophic cardiomyopathy (HCM) refers to a myocardial disease characterized by a hypertrophied non-dilated left ventricle [2]. In a few HCM cases, the hypertrophic basal interventricular septum (IVS) protruded into the left ventricular outflow tract (LVOT) and/or systolic anterior motion (SAM), which is an abnormal motion of the mitral valve towards the LVOT during systole resulting in left ventricular outflow tract obstruction, which defines hypertrophic obstructive cardiomyopathy (HOCM) [11, 12].

HCM is classified into primary and secondary forms based on etiopathogenesis. Primary HCM is triggered by a genetic mutation associated with myocardial contraction. Familial occurrence and genes associated with feline HCM were reported [6]. Secondary HCM is associated with endocrine disease, which induces an increase in left ventricular wall hypertrophy (*e.g.*, hyperthyroidism), pressure overload disorders predisposing to systemic hypertension (*e.g.*, chronic kidney disease, and hyperadrenocorticism) and congenital cardiac disease (*e.g.*, subvalvular aortic stenosis) [3, 12].

HCM is a common hereditary disease in cats and humans; however, it is rare in dogs. Most dogs diagnosed with HCM are large breeds, male, and identified before attaining 3 years of age [5, 15], suggesting a possible hereditary cause [5]. Therefore, HCM in a small-sized and aged canine breed is an extremely rare entity [15].

Retrospective studies of canine HCM suggest that physical, electrocardiographic, radiographic and echocardiographic examinations were used for the diagnosis [8]. This study describes radiographic and echocardiographic features of a dog with HOCM.

An 11-year-old castrated male, Yorkshire terrier presenting with cough and depression was referred to Gyeongsang National University veterinary medical teaching hospital. The dog was treated previously with inotropes, angiotensin-converting-enzyme inhibitors, and diuretics. During physical examination, auscultation revealed systolic murmur located at the base of the left heart. Systolic blood pressure was 148 mmHg. Serum chemistry profiles showed mild elevation in blood nitrogen urea. Electrocardiogram revealed a tall R wave and left-axis deviation suggesting left ventricular enlargement.

Thoracic radiographs revealed mild dilation of left and right atrium, and severe left ventricular enlargement (Fig. 1). Two-dimensional echocardiography showed concentric hypertrophy of left ventricle and multiple hyperechoic foci in the

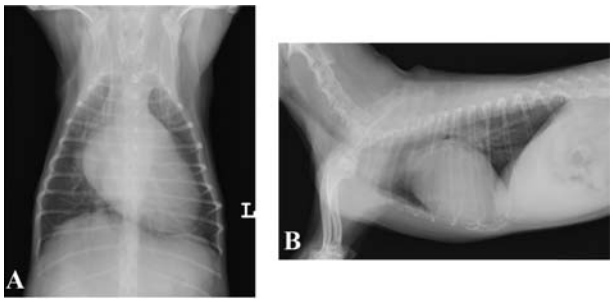

Fig. 1. Ventrodorsal (A) and right lateral (B) thoracic radiographs of the dog showing a severely enlarged left ventricle. Mild dilation of left and right atrium is also detected.

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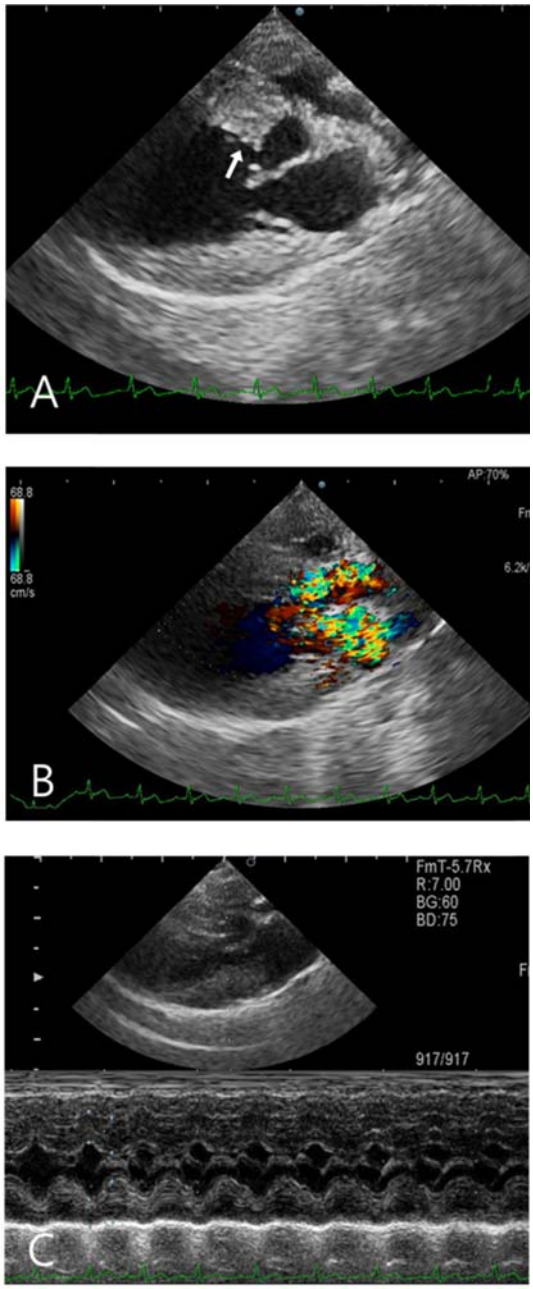

Fig. 2. Right parasternal long-axis: a four-chamber view. (A) B-mode shows concentric hypertrophy of left ventricle and multiple hyperechoic foci in the left ventricular wall. The infundibular hypertrophy creating the outflow obstruction is also clearly visible (arrow). (B) Turbulent flow in the left ventricle extending into the left ventricular outflow tract was identified on the Color Doppler echocardiogram. Mitral regurgitation was found as well. (C) M-mode echocardiography in the mitral valve aspect showing a mild thickening of the mitral valve and mild systolic anterior motion.

left ventricular wall, and remarkably bulging basal septal wall associated with the hypertrophy (Fig. 2). M-mode echocardiography showed a symmetrical thickening of the left ventricular wall during diastole (left ventricular posterior wall diameter: 9.5 mm; reference range, 4.2–5.8 mm; ventricular septal diastolic thickness: 10.9 mm; reference range, 5.3–7.3 mm). Fractional shortening (49.4%) was within normal range (30–50%). Anterior leaflet of mitral valve showed a mild thickening, mild SAM and stiffness (Fig. 2C). The left ventricular outflow tract to aortic valve diameter ratio was 0.49 (reference range, 0.509–0.737) and the left atrial to aortic root diameter ratio was 2.02 (reference range, <1.3). Color-flow Doppler imaging showed a turbulent flow starting in a left ventricle and extending into the LVOT, and eccentric mitral regurgitation. Spectral Doppler recordings revealed high LVOT flow velocity (4.17 m/sec: reference range, <1.7 m/sec). Mitral regurgitation velocity was 5.96 m/sec. Early diastolic mitral inflow velocity was 1.3 m/sec, which was higher than the reference range (0.62 to 0.84 m/sec), and the ratio of the early (E) to late (A) ventricular filling velocities (E/A ratio) was 1.53. Tissue Doppler imaging (TDI) showed reversal of E`/A` in a left ventricular wall and an E peak velocity of 3.7 cm/sec (Fig. 3). Tricuspid regurgitation velocity was 3.39 m/sec. Additionally abdominal ultrasonography was performed to detect other causes of systemic hypertension, and the results showed a mild enlargement of bilateral adrenal gland.

Based on the radiographic and echocardiographic examination, hypertrophic obstructive cardiomyopathy was diagnosed with secondary changes involving myxomatous mitral valve degeneration and mild pulmonary hypertension. The dog was managed with previous drugs and a β-blocker (carvedilol, 0.2 mg/kg, twice a day, orally). Two days later, the dog presented with cyanosis and hypotension. The β-blocker was eliminated and only inotropes, angiotensin-converting-enzyme inhibitor and diuretics were administered. After 5 months of follow-up, no significant radiographic or echocardiographic changes were found.

HCM is a rare disease in dogs, unlike feline patients, occurring in large young males [5, 8, 13, 15] and was impossible to establish the etiology of canine HCM [11, 15]. This report presents an extremely rare case of HCM involving an aged, small canine breed. Previous canine reports were diagnosed based on cardiac wall thickness (IVS: 13–22 mm, mean 19 mm; LVPW: 10–19 mm, mean 15 mm) [9, 10], TDI (PW-TDI E` peak velocity 3.0–7.3 cm/sec) and systolic/diastolic function [11]. We also used same criteria for diagnosing HCM.

HCM with dynamic obstruction of the LVOT (DLVOTO) was classified as HOCM, which is frequently diagnosed in feline and human patients. SAM resulted as the anterior mitral valve is sucked into LVOT [3, 11]. In a recent report, SAM was also responsible for abnormal geometry of the mitral valve abnormalities, such as displacement of the left ventricular papillary muscle [1]. Obstruction of the LVOT in canines was frequently characterized by the fixed form, the so-called aortic stenosis. DLVOTO was found in a majority of canine patients presenting with the mitral valve abnormal-

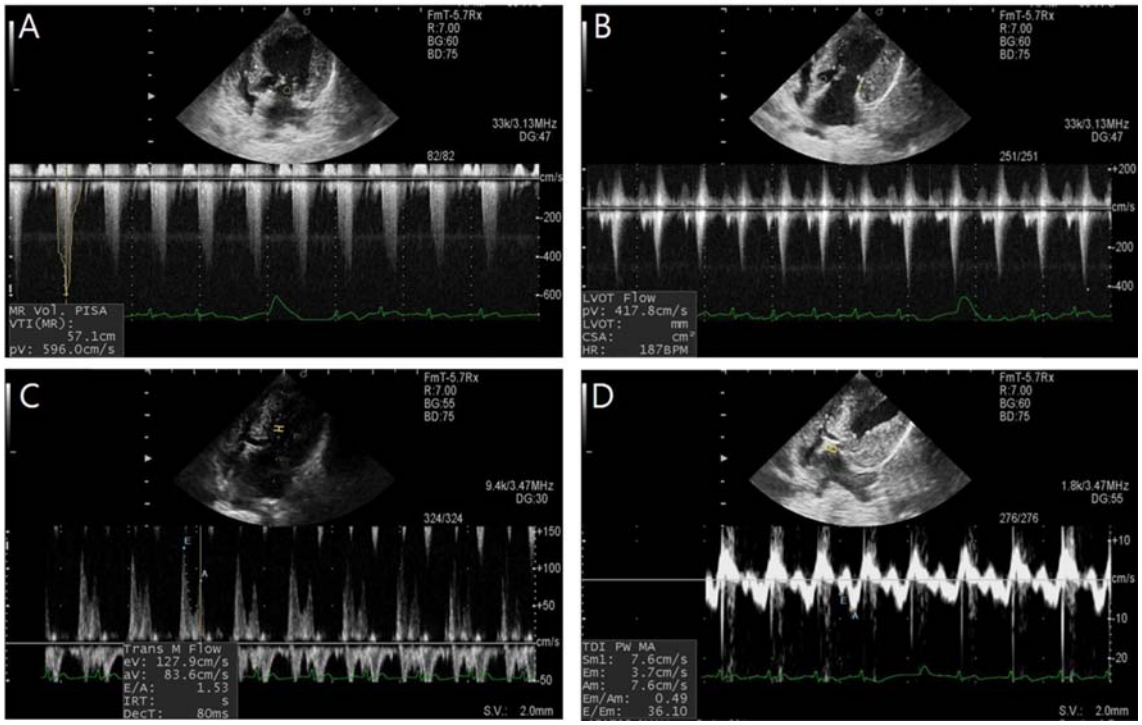

Fig. 3. Spectral Doppler echocardiogram of the dog in the left long-axis view of the four chambers. High left ventricular outflow tract flow velocity (4.17 m/sec) (A) and mitral regurgitation velocity (5.96 m/sec) (B) were identified. Early diastolic mitral inflow velocity (1.3 m/sec) (C) was increased with an E/A ratio of 1.53. Tissue Doppler imaging in left ventricular wall (D) shows reversal E`/A` and E peak velocity (3.7 cm/sec).

ity [11]. In this case, echocardiography revealed a turbulent flow in the LVOT around the hypertrophic basal septal wall, and SAM. SAM was mild, but remarkable morphological changes in basal septal wall bulge were found, resulting in obstructive changes in cardiomyopathy.

It is known that mitral valve degeneration and pulmonary arterial hypertension coexist with secondary changes in HCM patient [2, 4, 5, 7, 15]. In this case, we were able to find similar findings with previous studies.

Studies reporting abdominal findings using ultrasound are unavailable, except for the case involving canine HCM manifesting peritoneal effusion in ultrasonography [8], suggesting that peritoneal effusion was associated with right-sided heart failure [12]. In this case, additional abdominal ultrasonography revealed a mild enlargement of bilateral adrenal gland, diagnostic of benign changes (*e.g.*, nodular hyperplasia), without excluding hyperadrenocorticism. It is known that feline patients with hyperadrenocorticism show secondary hypertension ranging from a possible 60 to 80% [12]. In this case, it is not easy to make clear that the HOCM was related with hyperadrenocorticism due to lack of evidence for hypertension and further test for adrenal diseases.

Canine and feline cardiac cases are classified and treated according to the clinical severity based on the American College of Cardiology (ACC) grading system [12]. Feline HCM cases were managed with calcium-channel blockers, ACE inhibitors, diuretics and beta blockers [1, 2, 5, 12]. Antithrombotic drugs were also used to prevent pulmonary thromboembolism [5, 12]. In a recent report, the use of enalapril with furosemide was considered reasonable to treat congestive heart failure in feline HCM cases [1]. In previous reports, younger canine HCM cases treated with calcium-channel blockers, ACE inhibitors, diuretics and beta-blockers showed improved clinical signs [4, 11]. Older canine cases recovered following treatment with these drugs [8]. However, a few cases improved initially, and subsequently deteriorated clinically [15]. In this case, treatment with beta blockers resulted in side effects of hypotension and cyanosis within 2 days. It is known that hypotension and bradycardia are the most common adverse reactions in elderly patients exposed to beta blockers [14].

This case was well managed with diuretics, ACE inhibitors and inotropes. After 5 months of follow-up, the dog was static in radiography and echocardiography. The study limitation is related to the failure to further investigate bilateral adrenal gland enlargement via ultrasonography, because of absence of significant clinical abnormalities and lack of owner's consent to perform the examination.

In conclusion, this study involved a diagnosis of HOCM via radiography and echocardiography in an aged small breed dog. Despite a minor clue in abdominal ultrasonography suggesting the possibility of systemic hypertension, no further

study was conducted to investigate the causes of bilateral AG enlargement. However, this case suggests the possibility of secondary HOCM in an aged small breed dog.

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