

Acquired Mitral Stenosis in a Cat with Hypertrophic Cardiomyopathy

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ABSTRACT. A seven-year-old castrated male domestic shorthair cat was diagnosed with hypertrophic cardiomyopathy (HCM) and suspected mitral stenosis (MS) based on electrocardiography, thoracic radiographs and echocardiographic findings. Post-mortem examination of the heart revealed morphological features consistent with HCM. In addition, there was marked fibrous deposition on the surfaces of the chordae tendineae extending to both mitral valve leaflets, which caused total chordal fusion into pillars of fibrous tissue and fusion of the commissures. The present case indicates that acquired MS can occur in association with HCM in the cat.

KEY WORDS: feline, hypertrophic cardiomyopathy, mitral stenosis.

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Hypertrophic cardiomyopathy (HCM) is the most common feline cardiac disease. Cardiac abnormalities associated with HCM include narrowing of the left ventricular diameter, left ventricular outflow obstruction, and mitral regurgitation due to systolic anterior motion [4]. To our knowledge, however, other hemodynamic disorders have not been documented in association with HCM.

Mitral stenosis (MS) commonly occurs in humans [12, 13], however, this valvular disorder rarely develops in animals [5, 7, 8]. Although MS may be a congenital or acquired condition, this disorder most commonly occurs secondary to rheumatic fever in humans [6, 9, 10]. In contrast, only four cats with MS have been reported to date. Of these, two cats had congenital supra-ventricular MS [3, 11]. The others had congenital dysplasia of the mitral valve complex, with the MS considered to be an acquired lesion related to the congenital disorder [11]. The present paper describes acquired MS without dysplasia of the mitral valve complex in a cat with HCM.

A seven-year-old castrated male domestic shorthair cat, weighing 4.9 kg, was referred to the Veterinary Medical Teaching Hospital of the Nippon Veterinary and Animal Science University for evaluation of progressive respiratory distress. The cat had been diagnosed with HCM, and oral therapy consisting of diltiazem (7.5 mg/cat, bid), enalapril (2.5 mg/cat, sid), propranolol (10 mg/cat, sid) and furosemide (5 mg/cat, bid) was initiated by the referring veterinarian. On presentation, the cat was mildly dehydrated, lethargic and in respiratory distress. Femoral pulses were irregular, and pulse quality fluctuated. Capillary refill time was within normal limit. Cardiac auscultation revealed tachycardia with an irregular rhythm, and muffled heart sounds that varied in intensity. Increased bronchovesicular sounds were detected over all lung fields. Oxygen and furosemide (1 mg/kg, iv) therapy was initiated, and a complete blood count (CBC), serum chemistry, electrocardiograph

(ECG), thoracic radiographs and echocardiograph were performed.

CBC and serum chemistry values were within reference ranges, except for a mild increase in blood urea nitrogen (42.4 mg/dl). The ECG showed atrial fibrillation and occasional ventricular premature contractions. The QRS complex was of normal duration and configuration, and the ventricular rate was approximately 220 beats per minute. Thoracic radiographs detected a small amount of pleural effusion and atelectasis of the left pulmonary cardiac lobe. Although film detail were obscured by the pleural effusion, the trachea was elevated, suggesting the presence of an enlarged left ventricle. Left atrial enlargement was also suspected.

B-mode echocardiography (Fig. 1) revealed significant enlargement of the left atrium. A thrombus was not observed in any of the cardiac chambers. The mitral valve leaflets were thickened, and incomplete separation of the leaflets during diastole was observed. In addition, diastolic doming of the anterior mitral valve leaflet into the left ventricle was also observed. The left ventricular posterior wall and interventricular wall thickness during diastole were 9.8 and 7.4 mm, respectively. Mitral regurgitation was detected with color flow Doppler echocardiography. Based on these findings, a clinical diagnosis of HCM was made, and MS was strongly suspected. However, EF slope (diastolic descent rate of mitral valve) and transmitral flow velocity, which are essential to the diagnosis of MS [7], could not be adequately determined because the cat experienced cardiopulmonary arrest during the examination. Although cardiopulmonary resuscitation was intensively performed, the cat could not be resuscitated.

At necropsy, approximately 18 ml of serosanguineous fluid was present in the thoracic cavity, and macroscopical lesions were found in the lung and heart. The lungs were diffusely congested, edematous and firm on palpation, and

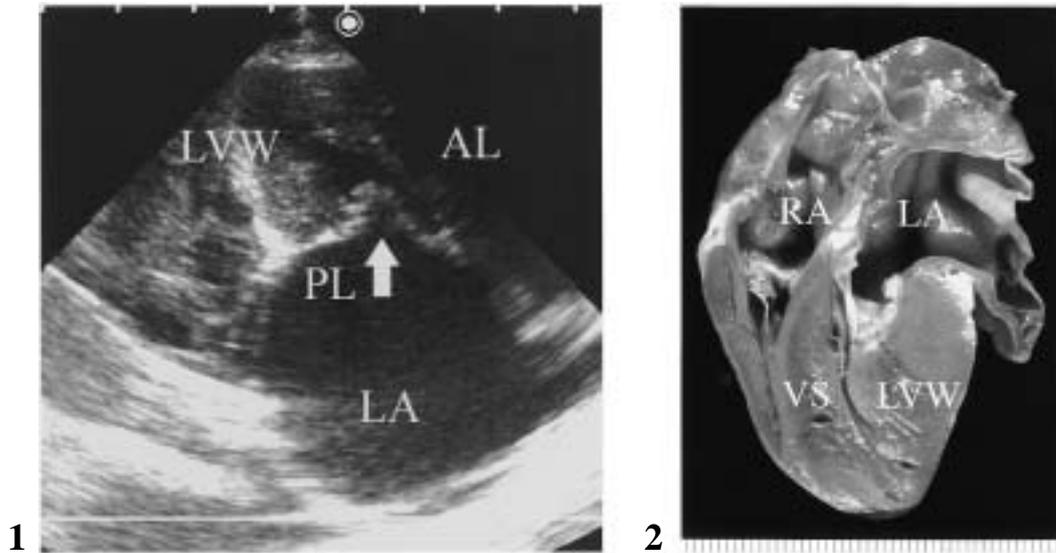


Fig. 1. Two-dimensional echocardiogram of a cat with acquired mitral stenosis and hypertrophic cardiomyopathy. The image is a right-side parasternal long axis view. The left atrium (LA) is severely enlarged. The anterior (AL) and posterior leaflets (PL) of the mitral valve are thickened, and incomplete separation of AL and PL during diastole is observed. An arrow indicates diastolic doming of AL. Eccentric hypertrophy of the left ventricular free wall (LVW) and ventricular septum is also noted.

Fig. 2. Longitudinal section through the entire heart, showing left ventricular hypertrophy, a narrow left ventricular cavity, and left atrial dilatation. LA, left atrium; RA, right atrium; LVW, left ventricular wall; VS, ventricular septum. Scale, 1 mm.

the trachea and bronchi contained a frothy fluid. The heart was enlarged. The left ventricular free wall, left ventricular papillary muscles and ventricular septum were hypertrophied, the left ventricular cavity was decreased, and there was marked dilatation of the left atrium (Fig. 2). The hypertrophied papillary muscles in the left ventricle were positioned closely together. The atrial surface of the mitral valve showed fusion of adjacent surfaces of both mitral valve leaflets in the commissural area, producing an oval, stenosed orifice that was funnel-shaped (Fig. 3). The valve leaflets were thickened, opaque, and grayish-white, but the surface was smooth and glistening. Areas of hemorrhage were observed within the valve stroma of the posterior leaflet. There was an adhesion between the posterior leaflet and the left ventricular wall and fusion of the chordae tendineae.

Histological examination of the ventricular myocardium revealed morphological features characteristic of HCM, such as marked disorganization of cardiac muscle cells, interstitial myocardial fibrosis (plexiform fibrosis), and arteriosclerosis of small intramural coronary arteries with thickened walls and narrowed lumina. In the mitral valve apparatus, there was marked fibrous deposition on the surfaces of the chordae tendineae extending to involve the distal portions of both mitral valve leaflets, which caused total chordal fusion into pillars of fibrous tissue and fusion of the commissures (Fig. 4). The fibrotic changes eradicated the originally layered leaflet architecture, while the collagenous core of chordae embedded in the fibrous lesions remained almost intact. Inflammatory infiltrates were absent. A

mural friction lesion (fibrous thickening) of the left ventricular endocardium was present under the thickened posterior mitral valve leaflet, causing adhesion of the distal portion of the leaflet to the mural endocardium (Fig. 4).

In cats with HCM, one of the most common hemodynamic alternations is the obstruction of the left ventricular outflow tract, impairing passage of blood into the aorta. In addition, systolic anterior motion of the mitral valve and left ventricular diastolic dysfunction also develop, leading to elevated left atrial pressure and pulmonary edema. However, the authors believe that other hemodynamic alterations have not been described in the veterinary literature.

MS is an extremely rare cardiac disease in small animals, and only four cats with MS have been reported to our knowledge. Two cats had congenital supravalvular MS [3, 11], whereas the others had mitral valve dysplasia, and the commissures of the mitral valve leaflet fused causing MS. The latter type of MS was described to be a terminal acquired lesion of the mitral valve dysplasia [11]. Thus, the present paper would be the first report of acquired MS without mitral valve dysplasia in a cat.

The etiology and pathogenesis of acquired MS have not been ascertained in cats. In humans, the most frequent etiology of acquired MS is chronic rheumatic heart disease [1, 6, 9, 10], but there have been no reports of rheumatic disease in cats. Other causes of acquired MS in humans are uncommon, and include active infective endocarditis and systemic lupus erythematosus [1, 6, 9, 10]. However, there was no evi-

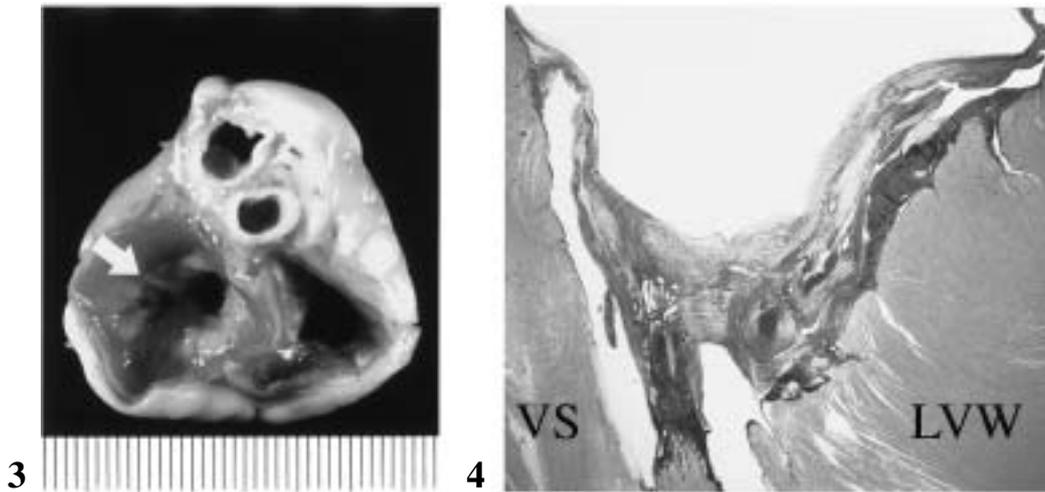


Fig. 3. Atrial aspect of the mitral valve (an arrow), showing a markedly stenotic valve orifice with fusion of the valve commissures. Scale, 1 mm.

Fig. 4. Microscopic section of the mitral valve, showing chordal thickening with fusion and fibrotic valve leaflets causing commissural fusion. LVW, left ventricular wall; VS, ventricular septum. Elastica van Gieson $\times 10$.

dence to support the view that prior bacterial infection or immune-mediated phenomena might play a role in the disease process in our case. In the present case, the basis of the mitral valve lesion was fibrous thickening with fusion of the chordae tendineae and leaflet. Considering the gross and histopathologic nature of the valvular lesion, it is reasonable to speculate that the fibrotic changes were due to abnormal contact (mechanical friction) between the chordae tendineae [2]. Namely, the papillary muscles in the left ventricle were hypertrophied and positioned closely together by a pathological process of HCM. As a result, the interchordal spaces were narrowed and, eventually, obliterated by a fibrous proliferation associated with mechanical friction during systole, leading to chordal and commissural fusion.

In conclusion, the present case demonstrates that acquired MS can occur in association with feline HCM.

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