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Case Report

Association of Mitral Valve Stenosis and Hypertrophic Cardiomyopathy in a Scottish Fold Kitten

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ABSTRACT

Hypertrophic cardiomyopathy (HCM) is a common feline heart disease characterized by left ventricular hypertrophy. Mitral valve stenosis is an extremely rare heart disease in small animals that causes partial obstruction of blood flow from the left atrium (LA) to the left ventricle during diastole. An eight-month-old Scottish Fold cat was brought to Selcuk University Animal Hospital with a complaint of dyspnea. Physical examination revealed tachypnea, dyspnea, and a grade 3/6 systolic murmur. Transthoracic echocardiography showed increased left ventricular wall thickness and LA dilation. Two-dimensional echocardiography revealed incompletely separated mitral valve leaflets, a narrowed mitral valve orifice area, and marked diastolic turbulence at the mitral valve level on color Doppler. Spectral Doppler demonstrated a high mean pressure gradient across the mitral valve inflow. These findings suggest that mitral valve stenosis and hypertrophic cardiomyopathy can coexist in cats and that echocardiography is a valuable diagnostic and evaluative tool for this condition.

Keywords: *Cat, Hypertrophic cardiomyopathy, Mitral valve stenosis*

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Scottish Fold Irkı Yavru Bir Kedide Mitral Kapak Stenozu ve Hipertrofik Kardiyomiyopati İlişkisi

ÖZET

Hipertrofik kardiyomiyopati (HCM), sol ventrikül hipertrofisi ile karakterize, kedilerde yaygın görülen bir kalp hastalığıdır. Mitral kapak stenozu ise diyastol sırasında sol atriyumdan (LA) sol ventriküle kan akışının kısmen engellenmesiyle karakterize, küçük hayvanlarda oldukça nadir görülen bir durumdur. Sekiz aylık Scottish Fold cinsi bir kedi, dispne şikâyeti ile Selçuk Üniversitesi Hayvan Hastanesi'ne getirildi. Fizik muayenede taşipne, dispne ve 3/6 şiddetinde sistolik üfürüm tespit edildi. Transtorasik ekokardiyografide sol ventrikül duvar kalınlığında artış ve LA dilatasyonu saptandı. İki boyutlu ekokardiyografik incelemede mitral kapakçıklarının tam olarak ayrılmadığı, mitral kapak orifis alanının daraldığı ve renkli Doppler incelemede mitral kapak seviyesinde belirgin diyastolik türbülans izlendi. Spektral Doppler analizi, mitral girişinde yüksek ortalama basınç gradiyenti ölçüldü. Bu bulgular, HCM ve mitral kapak stenozunun kedilerde bir arada bulunabileceğini ve ekokardiyografinin bu hastalıkların tanı ve değerlendirilmesinde yararlı bir araç olduğunu ortaya koymaktadır.

Anahtar kelimeler: *Hipertrofik Kardiyomiyopati, Kedi, Mitral Kapak Stenozu*

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INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most common heart disease in cats. It remains an important cause of cardiac morbidity and mortality and is associated with a risk of sudden death (5.9%) (Trehiou-Sechi et al., 2012; Novo Matos et al., 2022). Although the phenotype of HCM is well known, a definitive cause for the development of HCM in cats is still lacking (Grzeczka et al., 2024). Previous studies have demonstrated that genetic variants can lead to the HCM especially in Maine Coon, Ragdolls, and domestic shorthairs (Meurs et al., 2005; Meurs et al., 2007; Schipper et al., 2019). HCM is characterized by concentric left ventricular hypertrophy in the absence of other cardiac or systemic disease. Affected cats may show signs of congestive heart failure (CHF) or arterial thromboembolism (ATE). Similar to humans with HCM, many cats are able to live a normal life without showing any signs of heart disease, indicating the presence of subclinical disease (Kittleson and Côté, 2021). The best diagnostic tool for establishing and monitoring HCM is echocardiography. Significant regional or global left ventricular (LV) wall thickening, severely enlarged papillary muscles, systolic anterior motion (SAM), obliteration of the end-systolic cavity, and moderate to severe left atrial (LA) enlargement are among the hallmark findings (Kittleson and Côté, 2021). Mitral stenosis (MS) is a valvular disease characterized by a narrowing of the mitral valve opening (Shah and Sharma, 2023). Feline MSis an extremely rare heart disease that is usually congenital or very rarely acquired (Matsuu et al., 2007). The abnormalities such as shortened, fused, or excessively elongated chordae tendineae, direct adhesion of the valve to a papillary muscle, thickened, split, or shortened valves, valve prolapse, abnormal positioning or shape of the papillary muscle, and excessive dilatation of the valve annulus can lead to mitral valve stenosis (Campbell and Thomas, 2012; Ware and Ward, 2020). Although there are some reports of mitral valve stenosis in cats (Stamoulis and Fox, 1993; Takemura et al., 2003; Campbell and Thomas, 2012; Lu et al., 2016), there are limited data on Doppler assessment of the stenosis. Therefore, the aim of this case report was to perform spectral Doppler evaluation of the mitral valve stenosis in conjunction with HCM in a Scottish Fold cat using 2D, M-mode and Doppler echocardiography

MATERIAL AND METHODS

An 8-month-old male Scottish Fold weighing 3 kg was admitted to the Selcuk University Animal Hospital with a history of tachypnea. According to the owner, the cat became anorexic and depressed a few days earlier. On physical examination, tachypnea and dyspnea were observed. On thoracic auscultation, crackle sounds were heard especially in the caudo-dorsal regions. Cardiac auscultation revealed tachycardia with a regular rhythm and a 3/6 grade systolic murmur in the left apical region. Thoracic radiography and echocardiography were recommended based on physical examination findings and medical history. Cardiomegaly, left atrial enlargement, and pulmonary infiltrates were noted. There was no evidence of pleural effusion.
Transthoracic echocardiography (TTE) was performed with an echocardiographic unit and a 4.0 to 7.0 MHz sector transducer (Esaote MyLab 7X Vet, Italy) in right parasternal long- and short-axis views and in left apical views. Two-dimensional (2D), M-mode, and Doppler echocardiography were used (Thomas et al., 1993). No anesthetic protocol was used during the examination. The position attempted was a sitting because of tachypnea and dyspnea.

RESULTS AND DISCUSSION

At the initial examination, 2D echocardiography showed hypertrophic walls and end-systolic left ventricular chamber obliteration. End-diastolic left ventricular wall thickness was measured by 2D echocardiography using the right parasternal short axis view, and increased LV free wall and interventricular septal wall thicknesses were found (7.7 and 9.4 mm, respectively; reference range <6 mm). The left atrium to aorta ratio (LA/Ao) was significantly increased (3.20; reference interval LA:Ao <1.6). Spontaneous echo contrast was observed in the left atrium. Left ventricular ejection fraction and fractional shortening were normal (87% and 53%, respectively).

The 2D examination performed on the right parasternal long-axis and left apical views of the heart showed that the mitral valves were not fully open (Figure 1).

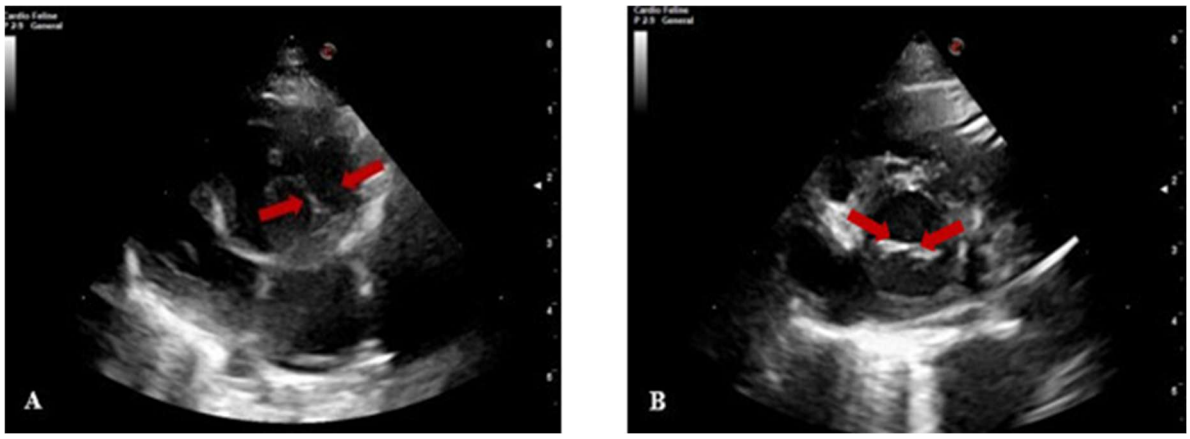

Figure 1. *A. Mitral stenosis seen in the left apical view. B. Mitral stenosis shown in the right parasternal short axis view (Anterior and posterior mitral valve leaflets are indicated by arrows).*

The parasternal short-axis view was also used to assess the mitral valve orifice area by planimetry of the mitral leaflets at the level of the tips and showed a narrowed area (Figure 2). In addition, movement of the anterior mitral valve leaflet toward the left ventricular outflow tract (systolic anterior motion, SAM) was detected.

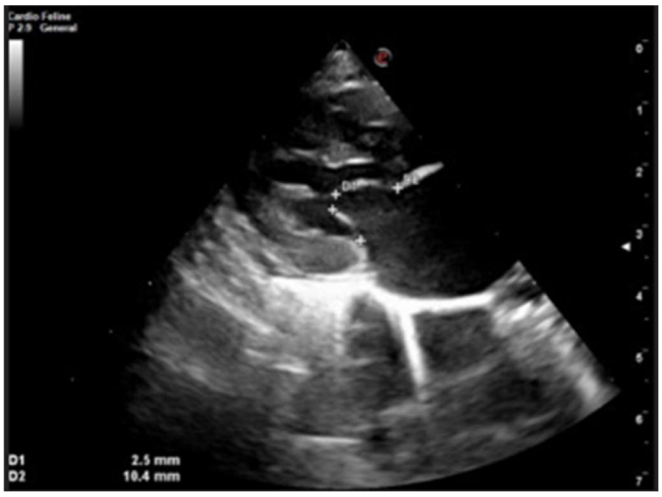

Figure 2. *In the right parasternal long-axis view, dimensions of mitral valve leaflets (2.5 mm) and mitral annulus (10.4 mm) showing mitral stenosis.*

Color Doppler imaging in the left apical 4-chamber view showed marked diastolic turbulence at the level of the mitral valve (Figure 3).

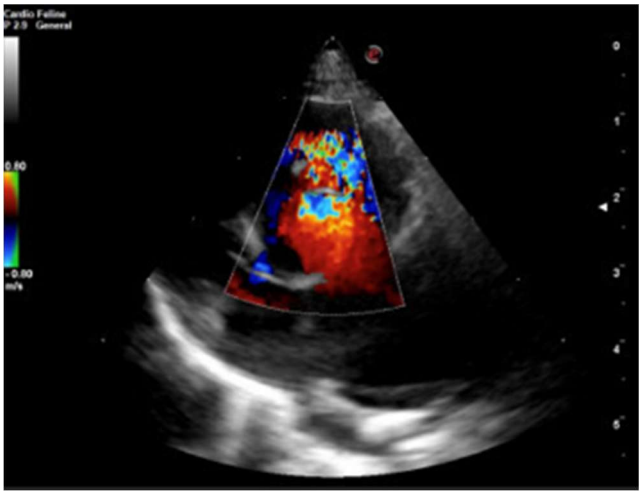

Figure 3. *Color Doppler imaging in the left apical 4-chamber view shows distinct diastolic turbulence at the level of the mitral valve.*

Continuous-wave spectral Doppler flow profile obtained from the left parasternal apical four-chamber view shows increased mitral inflow velocity (>2 m/s) and prolonged mitral inflow deceleration time. The mean pressure gradient (MPG), pressure half-time (PHT) and calculated area of the mitral valve were 10.3 mmHg, 39 ms, and 5.60 cm^2, respectively (Figure 4). Transmitral flow velocities and myocardial motion were measured from the left apical four-chamber view using PW and PW-TDI echocardiography.The ratio of peak velocity of early diastolic to late diastolic transmitral flow (E/A) was 0.74 (normal > 0.8). Early diastolic mitral annular velocity (Em) and E wave deceleration time were 0.06 m/s (normal: > 0.08 m/s) and 52 ms (normal: > 200 ms), respectively. The calculated ratio of peak velocity of early diastolic transmitral flow to peak velocity of early diastolic mitral annular motion (E/Em) was 21.23 (normal: < 8). Based on these findings, diastolic dysfunction (grade I) was diagnosed. Taken together, these findings were interpreted as hypertrophic cardiomyopathy concurrent with mitral valve stenosis.

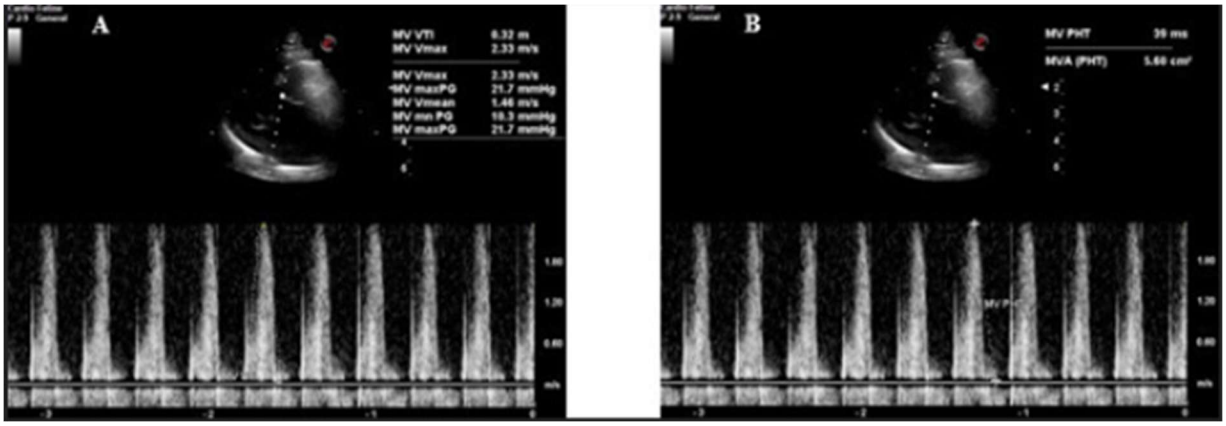

Figure 4. *A. Mean pressure gradient (MPG) (10.3 mmHg) and B. Pressure half-time (PHT) (5.60 cm2) of the mitral valve were calculated using CW Doppler in the left apical window.*

Treatment was aimed at stabilizing the clinical condition associated with congestive heart failure. The cat was placed in an intensive care unit to ensure adequate oxygenation (100 ml/kg/min). To resolve the pulmonary edema, high doses of furosemide (4 mg/kg) were administered intramuscularly (IM), as intravenous catheter placement was a very stressful procedure. After the improvement of tachypnea and dyspnea improved, the patient was discharged for home care. Furosemide (2 mg/kg PO BID) and spironolactone (1 mg/kg PO BID) were prescribed for the treatment of pulmonary edema. Clopidogrel (18.75 mg/cat PO SID) was administered to reduce the risk of arterial thromboembolism. In addition to medical treatment, the patient received regular cardiologic follow-up, and the sleeping respiratory rate was recorded daily by the owner to for recurrence of pulmonary edema. The cat was still alive at the time this manuscript was written.

Hypertrophic cardiomyopathy (HCM) is defined as concentric left ventricular hypertrophy in the absence of other cardiac or systemic disease (Cirino and Ho, 1993; Ommen et al., 2020). This condition can lead to both structural and functional changes in the heart. Although mitral valve stenosis has been rarely reported in cats (Stamoulis and Fox, 1993; Takemura et al., 2003; Campbell and Thomas, 2012; Lu et al., 2016), the prevalence of HCM in the feline population has been estimated at approximately 15% (Payne et al., 2015).

In cats with HCM, systolic function is typically preserved. However, diastolic function is impaired because the myocardium is unable to relax normally. As a consequence, LVstiffness increases, leading to elevated LV diastolic pressure (Schober and Chetboul, 2015). The rise in LV filling pressure is transmitted backward to the left atrium (LA), resulting in elevated LA pressure and subsequent LA dilation-(Kittleson and Côté, 2021). At the same time, mitral valve stenosis can cause partial obstruction of blood flow, further impeding flow and contributing to elevated left atrial (LA) pressure . This results in increased resistance to blood flow and consequently an increase in LA pressure (Lu et al., 2016). Increased left atrial pressure can lead to elevated pulmonary capillary pressures, resulting in pulmonary edema and congestive heart failure (Neema, 2015; Carpenito et al., 2021). In this case, the tachypnea and dyspnea were most likely secondary to LA dilation and pulmonary edema. An E/Em ratio greater than normal also indicates elevated LA pressure and an increased risk of pulmonary edema.

Mitral valve stenosis is a rare valvular disease in cats. Although previous studies have reported different forms of mitral stenosis in this species (Stamoulis and Fox, 1993; Campbell et al., 2012; Lu et al., 2016), only one study has described MS associated with HCM. In the present case, restricted leaflet motion and mitral annular inflow turbulence in diastole were diagnostic findings of MS. In humans, mitral orifice area and the mean LA-to-LV gradient are commonly used to assess the severity of MS (Carabello, 2005). However, no standardized method or guideline has been established for determining MS severity in cats. In people, severe MS is generally characterized by a mitral orifice area of <1.0 cm² and a mean gradient >10 mmHg (Carabello, 2005). Although the mitral valve area (MVA) in our case was greater than 5 cm², the mean transmitral gradient was close to 10 mmHg, which may suggest a clinically significant degree of MS in this cat.

The etiology of MS in cats is still not elucidated. In humans, rheumatic fever can cause fibrosis and thickening of the leaflet edges, commissural fusion, and shortening and thickening of the chordae tendineae, ultimately leading to fibrosis of the mitral apparatus and the development of MS (Waller, 1986). This pathophysiologic condition is still unproven in cats. However, some studies suggest that HCM may contribute to the development of mitral stenosis through several mechanisms (Takemura et al., 2003). Hypertrophic cardiomyopathy is characterized by thickening of the left ventricular wall and dilation of the left atrium (Schober and Chetboul, 2015). The enlarged left atrium can put pressure on the mitral valve and affect its function (Roberts and Perloff, 1972). Over time, this chronic pressure on the mitral valve can cause structural changes and fibrosis of the mitral valve (Boyden et al., 1982). This may lead to loss of elasticity and stenosis of the mitral valve (Calafiore et al., 2021).

In addition, when the papillary muscles of the left ventricle hypertrophy as part of the pathological process of HCM, they are positioned in close proximity to each other. Consequently, interchordal spaces become narrowed and eventually obliterated by fibrous proliferation associated with mechanical friction during systole, causing chordal and commissural fusion (Takemura et al., 2003).

CONCLUSION

In conclusion, this case highlights a rare coexistence of hypertrophic cardiomyopathy and mitral valve stenosis in a cat. Recognition of this association is clinically important, as it influences hemodynamic interpretation, therapeutic decisions, and long-term management of affected cats. Echocardiography is a useful tool in the diagnosis of hypertrophic cardiomyopathy and mitral valve stenosis. In particular, color Doppler and spectral Doppler techniques are useful in both the diagnosis of stenosis and the assessment of cardiac function in cats.

CONFLICT OF INTEREST

The authors declared no conflict of interest.

AUTHOR CONTRIBUTION

Conceptualization: HBŞ, AN; Data collection: HBŞ; Analysis and interpretation of results: AN; Writing - original draft: HBŞ, HA, GS; Writing - review & editing: HBŞ, AN. All authors reviewed and approved the final version of the manuscript.

ETHICAL APPROVAL

This research study complies with research and publishing ethics. The scientific and legal responsibility for manuscripts published in MJAVL belongs to the authors.

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DATA AVAILABILITY

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

CONSENT FOR PUBLICATION

Written informed consent was obtained from the owner of the patient for publication of this case report and accompanying images.

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