Severe aortic stenosis in a Persian kitten

Estenose aórtica severa em um gato Persa

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Summary: A case of severe aortic stenosis in a kitten is reported. Although no obvious clinical signs were observed initially, congestive left heart failure developed seven months later. On presentation, severe pulmonary edema was diagnosed and an incidental thrombus within the enlarged left atrium was disclosed on echocardiographic examination. Clinical features and diagnostic methods are discussed in this paper.

Keywords: congenital heart disease, echocardiography, thromboembolism, concentric hypertrophy, pulmonary edema

Introduction

Aortic stenosis is a congenital narrowing of the left ventricular outflow tract, aortic valve, or supravalvular aorta (Bolton and Liu, 1977; Liu, 1977; Kienle, 1998). Wherever it is located, obstruction to left ventricular outflow increases left ventricular systolic pressure, resulting in concentric hypertrophy of the left ventricle (Sisson et al., 2000).

Although reported as one of the most common congenital heart disease in dogs, aortic stenosis has only been described in a few cats (Liu, 1968; Stepien and Bonagura, 1991; King, 1997). To date, Harpster (1986) has documented this defect to count for only 6% of all congenital heart disease in domestic felines.

The increased left ventricular systolic pressure propels blood at a higher velocity across the stenotic area, thereby resulting in a turbulent flow that is associated with a systolic ejection murmur (Bolton and Liu, 1977; Stepien and Bonagura, 1991), which may eventually be accompanied by a soft diastolic murmur secondary to aortic valve insufficiency (Sisson et al., 2000).

Clinical signs may be absent in many mildly-to-moderately affected patients, and most affected cats are recognized when an ejection murmur is auscultated at the time of initial vaccinations (Bonagura, 1994; Kienle, 1998). In severely affected animals, exertional fatigue, syncope, and even signs related to congestive heart failure may be observed (Stepien and Bonagura, 1991; Kienle, 1998).

Definitive diagnosis of aortic stenosis requires angiocardiography, or echocardiography (Bonagura, 1994). A fibrous obstructing lesion, left ventricular concentric hypertrophy, and post-stenotic dilatation may be observed on two-dimensional echocardiography (Belerenian, 2007; Ferraris, 2007). Doppler echocardiography should reveal an increased peak velocity of aortic flow. In dogs, Sisson et al. (2000) documented that aortic velocity over 2.2 m/s is regarded as indicative of aortic stenosis, whereas in cats, Stepien and Bonagura (1991) reported that peak aortic velocity greater than 2 m/s might suggest a narrowed aortic tract.

As documented in literature, aortic stenosis is an uncommon congenital heart defect in cats. Therefore, the aim of this paper is to report a case of severe aortic stenosis in an initially asymptomatic Persian kitten, followed by the development of left atrial thrombus and signs of congestive heart failure.
Case description

A 5-month-old male Persian kitten weighing 2.5 kg was brought to consultation for initial vaccination. Except for a grade 3/6 systolic ejection murmur heard best over the left basilar region and radiating both cranially and dorsally, no obvious abnormalities were found on physical examination.

The caretaker noted that the kitten had an adequate appetite and was as active as its sibling. Although the cat had demonstrated normal behavior and physical activity with no overt medical conditions, ancillary exams were requested to investigate the murmur. Thoracic radiographs disclosed a normal cardiac silhouette and no evidence of congestive heart failure. Cardiac examination consisted in a 6-lead computerized electrocardiogram and echocardiography with Doppler examination. Only sinus tachycardia was documented on electrocardiography (200 bpm). Echocardiography was performed from the right parasternal and left apical locations. On two-dimensionally-guided M-mode echocardiography, a mildly decreased left ventricular systolic diameter (0.55 cm), decreased left ventricular diastolic diameter (1.26 cm), and increased fractional shortening (56%) were consistent with mild left ventricular concentric hypertrophy. Left atrial diameter was within the reference range. On the apical five-chamber view, a severely increased peak aortic velocity (6.03 m/s) was identified using continuous-wave Doppler (Figure 1). Based on the echocardiographic findings, the diagnosis of aortic stenosis was established.

Four months later, the cat was brought to the Veterinary Teaching Hospital for a follow-up consultation. On presentation, he weighed 4.7 kg, was very active and his owner reported no abnormalities since we last saw him. We auscultated a grade 4/6 systolic ejection murmur, still radiating cranially and dorsally, but the remainder of the physical examination findings were unremarkable. M-mode echocardiography showed a moderate left atrial enlargement (1.74 cm, with a left atrium-to-aorta ratio of 2.3), increased interventricular septal thickness in systole (0.98 cm) and diastole (0.89 cm), and decreased left ventricular systolic (0.55 cm) and diastolic diameter (1.00 cm). Color-flow and spectral Doppler demonstrated a high-velocity turbulent flow across the aorta.

Three months later, the cat was presented to the Veterinary Teaching Hospital emergency service with acute difficulty in breathing and tachypnea. On physical examination, the patient had mildly cyanotic mucous membranes, and was tachypneic, dyspneic, and tachycardic. Peripheral pulse rate matched the heart rate at 210 pulses/minute. Cardiac sounds were inaudible over pulmonary crackles and wheezes. Lateral radiographs showed severe pulmonary edema and left atrial enlargement. Short-term emergency therapy was immediately initiated and included furosemide and a nitroglycerin transdermal patch placed over a shaved area on lateral thorax. The patient was continually supplemented with 100% oxygen via a face mask, resulting in improvement in the color of the mucous membranes. An ultrasonographic examination of the heart disclosed a large thrombus (approximately 0.94 x 0.52 cm) (Figure 2) inside the severely enlarged left atrium (2.38 cm). Due to respiratory distress, the echocardiogram was not fully performed. Therefore, no measures of left ventricular chamber and aortic peak velocity were done at this time.

Although every effort was made to treat pulmonary edema, cardiopulmonary arrest occurred about three hours after initial presentation.
Discussion

Congenital cardiac disease comprises about 10% of cardiology cases in domestic mammals (Keirstead et al., 2002). In cats, however, congenital heart diseases are less common, counting for approximately 0.02% to 0.1% of hospital admissions (Goodwin, 2001). Although rare, some reports of congenital fixed aortic stenosis in cats exist (Liu, 1968; Stepien and Bonagura, 1991).

Although no clinical signs were observed at initial presentation, a congenital heart disease was suspected due to a loud systolic ejection murmur loudest over the left basilar region. Cote et al. (2004) reported that many murmurs detectable in overtly healthy cats appear to be caused by a latent structural heart disease. Because young animals can have physiologic or innocent heart murmurs, it is important to consider that, in general, loud heart murmurs are indicative of cardiac disease (Kittleson, 1998). Therefore, ancillary exams were requested to confirm the suspected heart pathology.

Initial thoracic radiographs did not show any abnormalities. Left ventricular enlargement and widening of the mediastinum (poststenotic dilatation) may be detected by radiography in severely affected animals (Liu, 1968; Bolton and Liu, 1977; Stepien and Bonagura, 1991). In some cases, however, thoracic radiographs may be normal (Sisson et al., 2000). Only sinus tachycardia was observed on the electrocardiogram. Although the electrocardiogram is generally normal in dogs with subaortic stenosis, severely affected dogs may eventually have ventricular premature contractions (Kienle, 1998). Nevertheless, we did not observe any abnormality in nearly three minutes of ECG recording in this cat.

The suspected congenital heart anomaly was confirmed by Doppler echocardiography. The highly increased velocity across aortic valve is the definitive diagnosis of aortic stenosis (Stepien and Bonagura, 1991; Ferraris, 2007), which has been recognized to occur in subvalvular, valvular, and supravalvular forms in cats (Kienle, 1998). Also, an incomplete subaortic stenotic ring was detected at necropsy in one cat (King et al., 1988). The obstruction was assumed to be fixed rather than dynamic owing to the lack of a delayed systolic peak in the continuous-wave Doppler tracing of the left ventricular outflow tract (Thomas et al., 1984; Kienle, 1998). Chronic pressure overload associated to a normal or enhanced myocardial function resulted in secondary left ventricular concentric hypertrophy, which is usually appreciated in moderately-to-severely affected animals (Kienle, 1998). Interestingly, no poststenotic dilatation was recognized. In dogs, however, the dilatation may be slight before 6 months of age (Kienle, 1998), which is likely to be similar in affected cats.

Due to questionable or unproven value, medical therapy was not initiated in this kitten (Fox, 1991; Bonagura, 1994). Although the kitten was asymptomatic and had no electrocardiographic abnormalities, the owner was informed of the possibility of starting a beta-adrenergic receptor blocking drug and an angiotensin-converting enzyme inhibitor empirically (Kienle, 1998; Sisson et al., 2000; Belerenian, 2007), since a severely increased gradient was measured across aortic valve. By decreasing heart rate and cardiac contractility, beta-blockers may reduce myocardial oxygen demand and increase coronary perfusion, thereby reducing the occurrence of malignant arrhythmias (Kienle, 1998). However, this option was declined and only restriction to vigorous exercise was recommended.

Cats with cardiac disease are prone to develop thromboembolism (Laste and Harpster, 1995; Smith and Tobias, 2004). In general, the thrombus is formed in the enlarged left atrium due to blood stasis, endothelial damage, and altered coagulability (Pion and Kittleson, 1989; Bonagura, 1994). In the case reported herein, the abnormal diastolic filling pattern as a consequence of concentric hypertrophy and ventricular stiffness resulted in left atrial enlargement, therefore predisposing for thrombogenesis (Bonagura, 1994; Kienle, 1998). On presentation, however, the thrombus had not caused clinical abnormalities and was an incidental finding. The thrombus within left atrium was large enough to be easily observed on standard long-axis four-chamber and short-axis views. Although reports of intracardiac thrombi exist (Venco, 1997), we did not find any report of intracardiac thrombus formation in a cat with aortic stenosis.

Conclusion

Although rare, aortic stenosis may occur in cats. Loud heart murmurs auscultated in overtly healthy kittens may indicate a congenital heart disease. Therefore, clinicians are reminded to perform a detailed physical examination prior to routine vaccinations or exams. Also, cats with aortic stenosis and enlarged left atrium should be considered at risk for developing intracardiac thrombi and even thromboembolism.

Bibliography


