Acquired Stenosis of All Four Heart Valves in a Boxer Mix Dog

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INTRODUCTION

Although valvular pulmonic stenosis and subvalvular aortic stenosis are commonly reported among congenital heart diseases in veterinary medicine, acquired valvular stenosis, particularly multivalvular stenosis, is rare.[1,2] We present findings of stenosis of all four heart valves in a 7-year-old Boxer mix dog, using echocardiography to assess the underlying structural cardiac changes. To our knowledge, this is the first case report of a veterinary patient with quadrivalvular stenosis.

CASE PRESENTATION

A 7-year-old female spayed Boxer mix dog was presented to an emergency referral clinic for further evaluation of progressive lethargy of 1 month's duration and recent onset of tachypnea. The patient was reported to have been otherwise healthy, with no significant reported medical history. There were no concurrent systemic illnesses described, nor any recent travel history reported. Baseline physical examination revealed a grade IV/VI left basilar systolic murmur with a regular rate and rhythm; six-lead diagnostic electrocardiography was performed, revealing normal sinus rhythm with increased P-wave amplitude. Abdominal distention was present, with a palpable fluid wave, and point-of-care ultrasound confirmed the presence of a small amount of anechoic abdominal effusion. A Doppler blood pressure reading was attempted but could not be obtained; this was suspected to reflect systemic hypotension. Thoracic radiography was performed and revealed generalized cardiomegaly with normal pulmonary vasculature and an overall unremarkable pulmonary parenchyma with no obvious evidence of pulmonary edema. On the basis of physical examination findings and history, echocardiography was recommended.

Transthoracic two-dimensional, color Doppler, and M-mode echocardiography revealed severely thickened and irregular mitral valve leaflets (Figure 1, Videos 1 and 2). Subjectively, the diastolic excursion of both mitral valve leaflets, but primarily the posterior leaflet, was reduced. There was reduced central coaptation of the leaflets, and color Doppler at the level of the mitral valve revealed aliasing of the color jet into the left ventricle during diastole, as well as systolic mitral regurgitation (Video 3).[2,3] There was moderate enlargement of the left atrium compared with aortic diameter, with a left atrial/aortic diameter measurement of 1.8 obtained via the Hansson or "Swedish" method, with upper limits normally <1.5 to 1.6 (Figure 2, Video 4).[4] The aortic valve leaflets were severely thickened and irregular, with reduced excursion of the leaflets observed during systole (Figures 2 and 3, and Video 4).[2] Color Doppler at the level of the aortic valve revealed aortic insufficiency (Figure 4). Mild concentric hypertrophy of the interventricular septum (measuring 1.1 cm in diastole; upper limit 1.0 cm on the basis of body weight) and left ventricular free wall (measuring

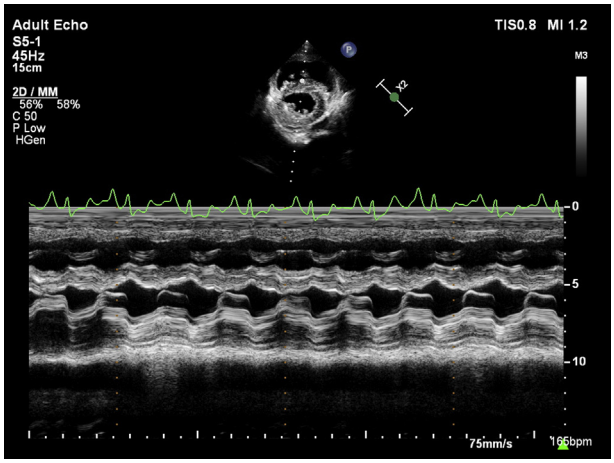

Figure 1 Right parasternal short-axis M-mode imaging at the level of the mitral valve, demonstrating severe thickening of both mitral valve leaflets with reduced diastolic excursion.

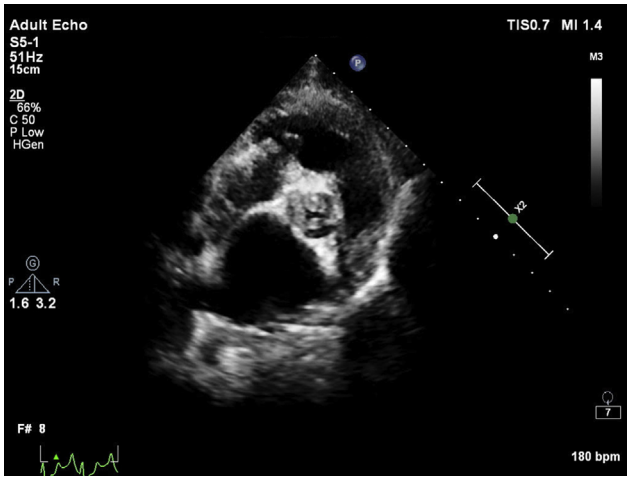

Figure 2 Right parasternal short-axis view at the level of the heart base optimizing the aortic valve, demonstrating severe thickening of the aortic valve leaflets and left atrial dilation. Severely thickened pulmonic valve leaflets are partially visible.

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VIDEO HIGHLIGHTS

Video 1: Right parasternal long-axis four-chamber view demonstrating moderate left atrial and severe right atrial dilation, mild right ventricular dilation, left and right ventricular concentric hypertrophy, and severe thickening of the atrioventricular valve leaflets.

Video 2: Left apical four-chamber view demonstrating severe thickening of the atrioventricular valve leaflets, biatrial enlargement, and diffuse concentric hypertrophy.

Video 3: Left apical four-chamber view with color-flow Doppler over the mitral valve, demonstrating a narrow aliased color jet flowing into the left ventricle during diastole secondary to mitral stenosis in addition to systolic mitral regurgitation.

Video 4: Right parasternal short-axis view at the level of the heart base demonstrating left atrial enlargement and severe thickening of the aortic valve leaflets. Additionally, the tricuspid valve and pulmonic valve are partially visible and are severely thickened.

Video 5: Right parasternal short-axis view at the level of the papillary muscles demonstrating mild right ventricular dilation as well as left and right ventricular concentric hypertrophy.

Video 6: Right parasternal short-axis view at the level of the heart base optimizing the tricuspid valve with color-flow Doppler, demonstrating tricuspid valve regurgitation. The severely thickened pulmonic valve leaflets are also visible.

View the video content online at www.cvcasejournal.com.

0.9 cm in diastole; upper limit 0.8 cm on the basis of body weight) was present (Videos 1, 2, and 5).[2] The tricuspid valve leaflets were severely thickened and irregular (Videos 1 and 2). Color Doppler at the level of the tricuspid valve revealed tricuspid regurgitation (Video 6). Subjectively, severe right atrial enlargement was present

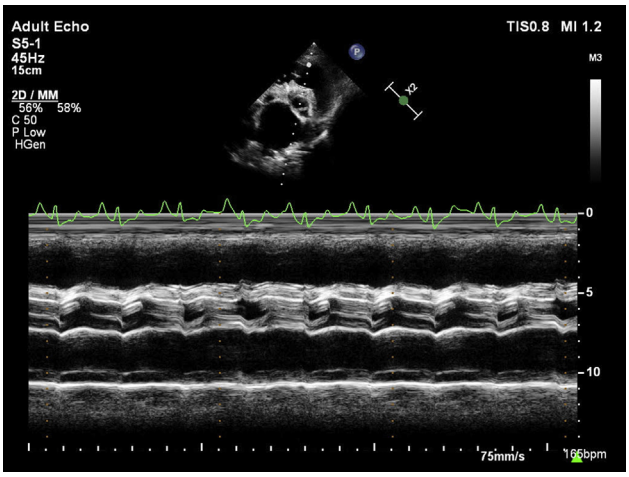

Figure 3 Right parasternal short-axis M-mode imaging at the level of the aortic valve, demonstrating severe valvular thickening and reduced systolic excursion of the aortic valve leaflets.

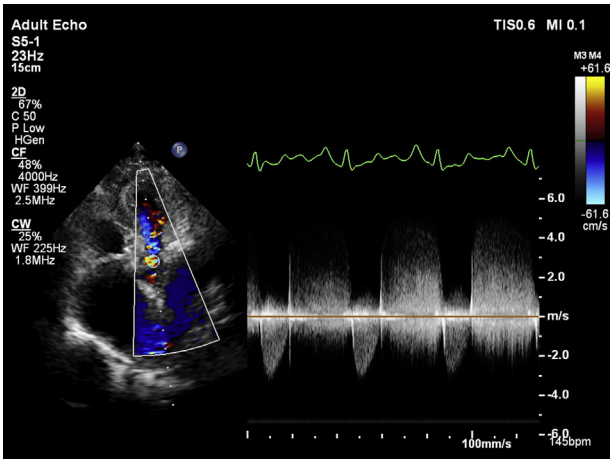

Figure 4 Left apical five-chamber continuous-wave Doppler imaging at the level of the aortic valve, demonstrating aortic valve stenosis and aortic insufficiency.

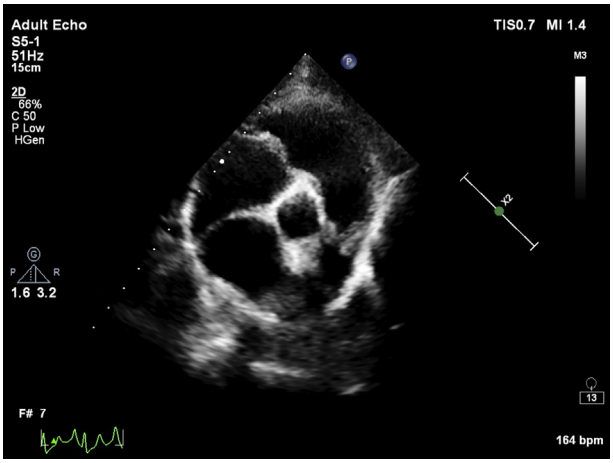

Figure 5 Right parasternal short-axis view at the level of the heart base, demonstrating severe thickening of the pulmonic valve leaflets.

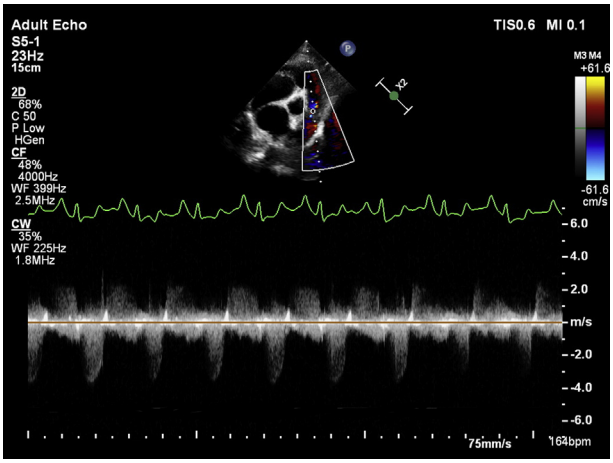

Figure 6 Right parasternal short-axis continuous-wave Doppler imaging at the level of the pulmonic valve, demonstrating pulmonic valve stenosis and pulmonic insufficiency.

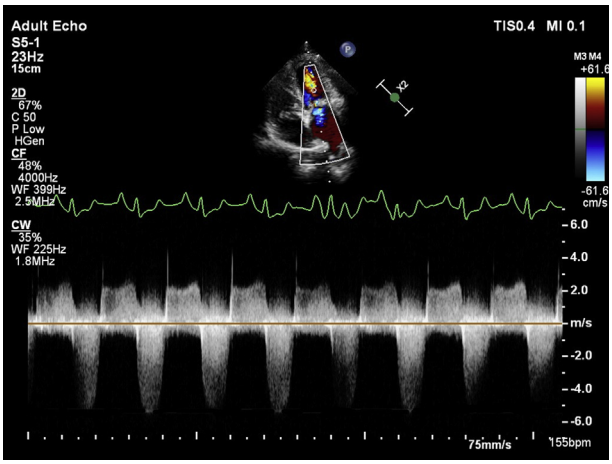

Figure 7 Left apical four-chamber continuous-wave Doppler imaging at the level of the mitral valve, demonstrating concurrent mitral valve stenosis and regurgitation.

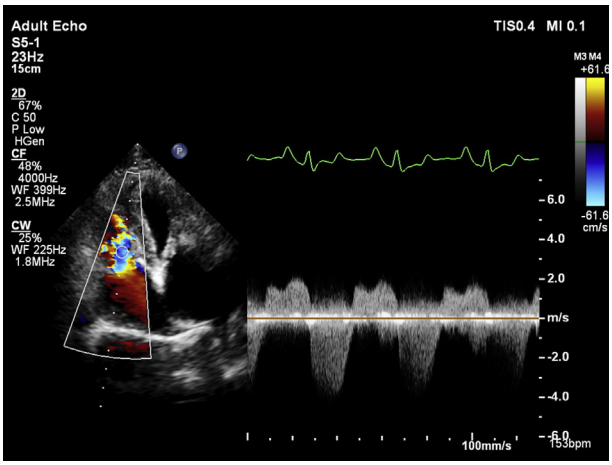

Figure 8 Left apical four-chamber continuous-wave Doppler imaging at the level of the tricuspid valve, demonstrating concurrent tricuspid valve stenosis and regurgitation.

(Videos 1 and 2). The pulmonic valve leaflets were severely thickened and irregular (Figure 5, Video 4). Color Doppler at the level of the pulmonic valve revealed pulmonic insufficiency (Figure 6). Subjectively, mild right ventricular eccentric and concentric hypertrophy was present (Videos 1, 2, and 5). Although not shown, hepatic venous distention with a moderate amount of anechoic abdominal effusion was confirmed on brief abdominal ultrasound.

Combined transthoracic color Doppler and continuous-wave Doppler echocardiography revealed an aliasing diastolic color jet into the left ventricle at the level of the mitral valve. An increased E-wave velocity (2.29 m/sec, corresponding to a 21 mm Hg diastolic pressure gradient) was observed on transmitral inflow, with a prolonged pressure half-time (144 msec; normal <50 msec) consistent with mitral valve stenosis (Figure 7).[2,3,5,6] At the level of the aortic valve, flow acceleration was identified at the level of the thickened aortic valve leaflets; peak systolic outflow velocity was 3 m/sec, correlating to a pressure gradient of 36 mm Hg, consistent with mild valvular aortic stenosis (Figure 4).[2,5] An increased E-wave velocity (1.6 m/sec; 10 mm Hg pressure gradient) was identified

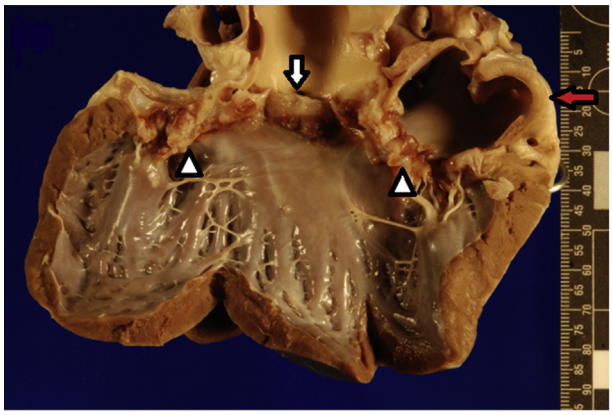

Figure 9 Gross pathology of the mitral and aortic valves. The left ventricle has been opened, revealing severe, nodular thickening of the anterior and posterior mitral valve leaflets (*arrowheads*). A portion of the aortic valve is partially visible (*arrow*) and is also grossly thickened and nodular in appearance. There is mild concentric left ventricular hypertrophy as well as hypertrophy of the posterior wall of the left atrium (*red arrow*).

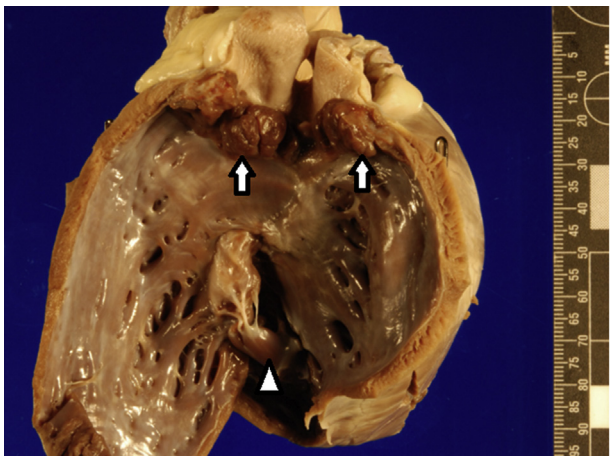

Figure 10 Gross pathology of the pulmonic valve. The right ventricle has been opened along the right ventricular outflow tract, revealing severe, nodular thickening of all pulmonic valve leaflets (*arrows*). The tricuspid valve is not completely visible, though a prominent papillary muscle can be seen (*arrowhead*). Mild concentric right ventricular hypertrophy is present.

on transtricuspid inflow, with a slow rate of mid-diastolic flow deceleration. A pressure half-time of 137 msec (normal values not reported in dogs) and mean diastolic pressure gradient of 10.5 mm Hg (normal values not reported in dogs) were consistent with tricuspid valve stenosis (Figure 8).[2,5-8] Flow acceleration was identified at the level of the thickened pulmonic valve leaflets; peak systolic outflow velocity was 3.8 m/sec, correlating to a pressure gradient of 57 mm Hg, consistent with moderate valvular pulmonic stenosis (Figure 6).[2,5]

On the basis of the echocardiographic findings and the presence of abdominal effusion, a diagnosis of quadrivalvular stenosis with right-sided congestive heart failure was made. A treatment plan, including a plan for further diagnostics on the basis of these findings,

was discussed with the owner. Because of the patient's poor prognosis, as well as financial limitations, the owner elected for humane euthanasia and consented to a necropsy. Gross assessment revealed fibrous thickening and nodular deposits on all four valves (Figure 9 shows the mitral valve, and Figure 10 shows the pulmonic valve). Histopathology was performed and revealed severe, diffuse fibromyxomatous degeneration with necrotizing valvulitis, mineralization, granulation tissue formation, and hemorrhage of the pulmonic and aortic valves, and diffuse, severe, chronic fibromyxomatous degeneration with nodular chondroid and osseous metaplasia, granulation tissue formation, hemorrhage, and histiocytic inflammation of the atrioventricular valves. No infectious agents or neoplastic changes were identified. A definitive underlying etiology of the changes observed was not determined on the basis of the histopathologic results, though differential diagnoses suggested in the pathology report included rheumatic heart disease, nonbacterial thrombotic endocarditis, and autoimmune disease such as systemic lupus erythematosus.[9]

DISCUSSION

To our knowledge, this is the first case report of a veterinary patient with stenosis of all four heart valves. Despite the lack of a definitive diagnosis through histopathology, a systemic etiology would be suspected given the described underlying etiologies reported in humans with multivalvular changes.[9] As the patient did not have a history of a heart murmur, a congenital stenosis was considered unlikely.[1] Infective endocarditis is a consideration given the appearance of the mitral valve and aortic valve leaflets, which are the valves most commonly involved in cases of infectious endocarditis in small animal patients; however, it is less common to see the tricuspid valve affected in small animal patients and even rarer to see the pulmonic valve involved in cases of infectious endocarditis.[10] There has been one case report of a feline patient with endocarditis affecting the mitral, aortic, and pulmonic valves, but there are no reports of canine patients with three or more valves affected, and it is exceedingly rare to see more than one valve involved.[10,11] No infectious agents were identified on the histopathology report, making infective endocarditis with typical organisms less likely. Myxomatous degeneration is very common in dogs and is commonly diagnosed in middle to older age patients.[12] It is possible that all four of this patient's valves had myxomatous changes, but valvular stenosis does not typically accompany myxomatous valvular disease in veterinary patients and is more commonly identified as a congenital lesion.[1,12] Although not prevalent in veterinary medicine, rheumatic heart disease would be a consideration for this patient, and there are several reports of quadrivalvular stenosis in humans with rheumatic fever.[13-15] Other systemic causes, such as autoimmune disorders including systemic lupus erythematosus, would also be considered on the basis of reported causes of pathologic multivalvular changes in humans.[9] Ultimately, further diagnostics could not be pursued given the owner's decision for humane euthanasia.

CONCLUSION

This patient had significant stenosis and structural changes to all four heart valves. To our knowledge, this is the first reported veterinary case of quadrivalvular stenosis. Although a definitive diagnosis was not determined, a systemic etiology was suspected.

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