J. Vet. Med. A **51,** 138–142 (2004)

ISSN 0931–184X

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Tissue Doppler Imaging and Echo-Doppler Findings Associated with a Mitral Valve Stenosis with an Immobile Posterior Valve Leaflet in a Bull Terrier

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With 5 figures

Received for publication: October 30, 2003

Summary

A mitral valve stenosis was diagnosed in a 2-year-old female Bull Terrier by use of two-dimensional (2-D) and M-mode echocardiography, colour-flow imaging and spectral Doppler examinations. Tissue Doppler Imaging was also performed to assess the segmental radial myocardial motion. The mitral valve stenosis was characterized by a decreased mitral orifice area/left ventricle area ratio (0.14), an increased early diastolic flow velocity (E wave = 1.9 m/s), a prolonged pressure half-time (106 ms) and a decreased E-F slope (4.5 cm/s) on pulsed-wave Doppler examination. This mitral stenosis was associated with an immobile posterior leaflet, as seen on 2-D and M-mode echocardiography. Immobility of the posterior mitral leaflet is considered to be a rare finding in humans and, to our knowledge, has not been precisely documented in dogs with mitral valve stenosis.

Introduction

A 2-year-old, 25-kg, female Bull Terrier was admitted for evaluation of a systolic heart murmur detected during routine examination 1 month earlier. The dog had no history of symptoms related to the respiratory or the cardiovascular system, and did not have any disease (such as infection), which may have contributed to a valve abnormality. Physical examination revealed a left apical, grade II/VI systolic murmur with a soft diastolic component, grade I/VI. The resting heart rate was 113/min and respiratory rate 32/min. A standard 6-lead electrocardiogram (ECG) revealed a normal sinus rhythm with amplitudes and durations of the complexes within the reference ranges (Tilley, 1985). Thoracic radiographs were normal. Systemic arterial pressure measured by Doppler technique was 113/60.

Conventional echocardiography and Doppler examination

Two-dimensional (2-D) echocardiography, colour-flow imaging and spectral Doppler examinations, and Tissue Doppler Imaging (TDI) were performed using a Vingmed vivid 5 (General Electric Medical System, Waukesha, WI, USA) with a 3.5 MHz phased-array transducer. During the whole ultrasound examination, the heart rate was stable (mean: 120 bpm). Measurements of the left atrium (LA, 26.6 mm) and the aorta (AO, 23.1 mm) were performed with a 2-D method (Hansson et al., 2002), using a short-axis right-sided parasternal view obtained in diastole at the level of the aortic valve. The normal LA/AO ratio (1.15) excluded left atrial dilation. The left ventricular end-diastolic diameter (LVEDD, 47.3 mm) appeared slightly enlarged and the fractional shortening (FS, 27%) within the lower ranges, but specific reference ranges for this breed are lacking. There was no sign of hypertrophy of the left ventricular wall (LVWD, 9.3 mm) or of the interventricular septum (IVSD, 11.4 mm). The anterior and the posterior mitral valve leaflets were thicker than normal, 5.5 mm and 6.3 mm, respectively (reference value for our clinic: ≤2 mm). The posterior leaflet appeared immobile on 2-D and M-mode echocardiography (Fig. 1), maintaining a closed position throughout the cardiac cycle. There was no doming, defined as restricted leaflet tip motion with preserved mobility of the leaflet body, of the anterior mitral leaflet. Colour-flow imaging identified a narrow aliased mitral inflow during diastole (Nyquist limit, 0.85 m/s; Fig. 2), as well as regurgitant mitral flow during systole. A right parasternal long-axis view showed that the end-diastolic mitral valve orifice diameter at the tip of the leaflets (0.4 cm) was reduced to approximately 20% of the size of the orifice diameter at the base of the leaflets (1.8 cm). Mitral valve area (MVA) was measured by tracing the inner area of the mitral valve orifice in the frame that illustrated the maximal opening of the leaflets on a right parasternal short-axis view (Fig. 3), as described in dogs by O'Grady et al. (1986). The area of the left ventricle (LVA) was measured in the same frame, and the two areas were compared. The mitral orifice measured 2.58 cm^2 (reference value: 3.69 ± 1.42 cm^2, Boon, 1998b), and the MVA/LVA ratio was 0.14 (reference range: 0.38–0.52, O'Grady et al., 1986).

Spectral Doppler of the mitral inflow confirmed an increased early diastolic flow velocity (E wave), 1.9 m/s (reference range: < 1.1 m/s, Yuill and O'Grady, 1991), and a prolonged pressure half-time, 106 ms (reference range: < 50 ms, Lehmkuhl et al., 1994). The early diastolic closure slope of the septal leaflet, i.e. E-F slope, was markedly decreased, 4.5 cm/s (reference: 10.6 ± 3.29 cm/s, Boon et al., 1983). Aortic flow velocity was within the reference range (1.16 m/s, Boon, 1998a), and there was no sign of aortic insufficiency. Pulmonary artery flow velocity was within the reference range (0.7 m/s, Yuill and O'Grady, 1991), and the flow profile was normal (Johnson et al., 1999). Pulmonary insufficiency of a velocity of 1.82 m/s was detected. Using colour Doppler and according to Rishniw and Erb (2000), the pulmonary regurgitation was classified as medium (colour jet extending above the most rightward level of the aortic valve throughout diastole, length: 0.72 mm/kg). Tricuspid regurgitation was

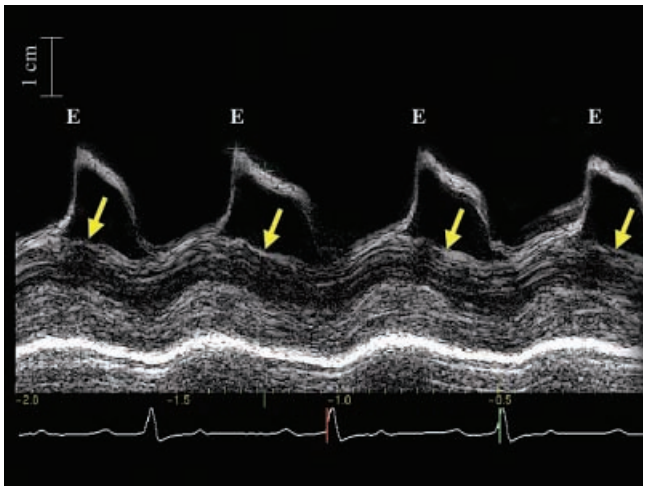

Fig. 1. M-mode recording at the level of the mitral valve leaflets (100 mm/s). The posterior leaflet (arrows) is closed throughout the whole diastole, and only follows 'passively' the posterior ventricular wall motion. The diastolic opening of the anterior leaflet is abnormal, without any significant late-diastolic opening.

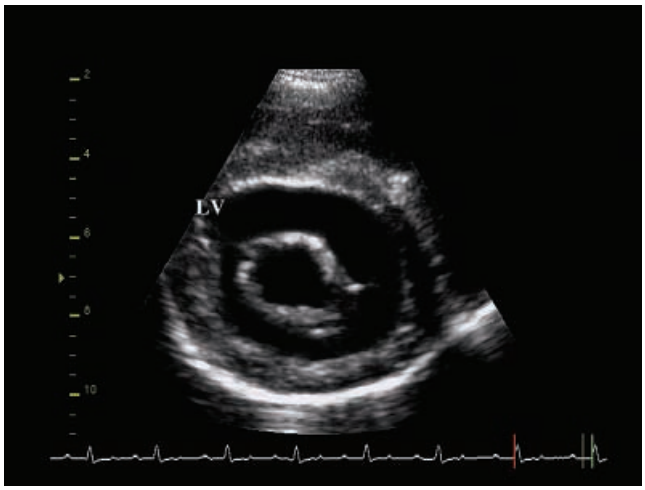

Fig. 3. Two-dimensional echocardiogram (right parasternal short-axis section of the ventricles). The maximal mitral valve opening was seen on this diastolic frame, which permitted to calculate the mitral valve area by tracing the inner area of the mitral valve orifice. The area of the left ventricle (LV) was measured in the same frame.

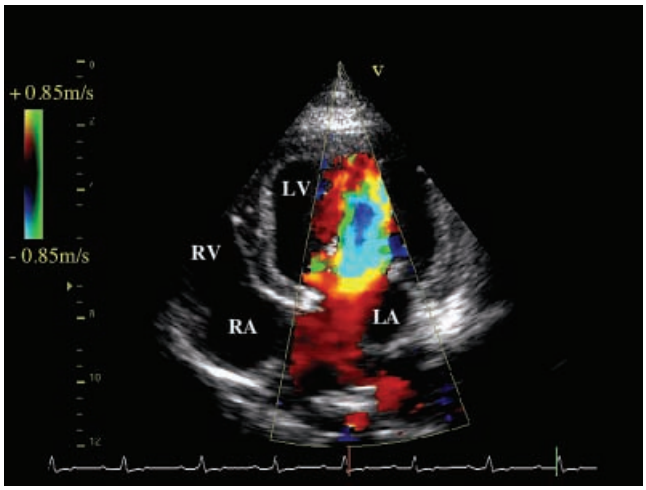

Fig. 2. Colour-flow Doppler echocardiogram (left apical four-chamber view). This diastolic frame shows turbulent (or aliased) narrow flow into the left ventricle through the stenotic valve orifice. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

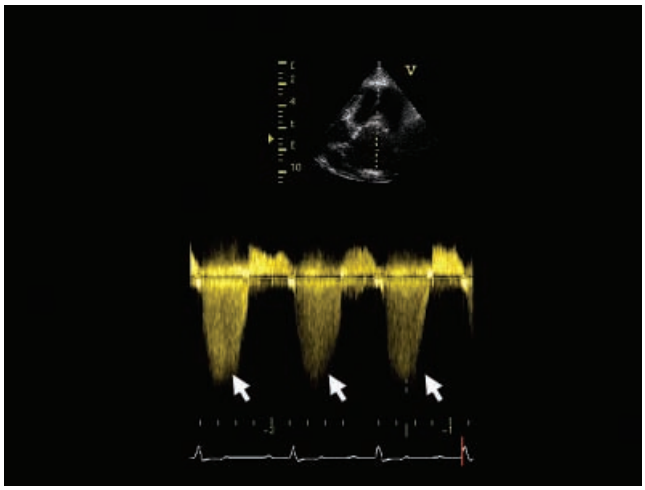

Fig. 4. Continuous-wave Doppler showing the systolic mitral regurgitant flow (arrows; velocity of ECG trace: 100 mm/s; Doppler velocity scale: m/s).

not found. Mitral regurgitant maximal flow velocity measured 5.88 m/s with a symmetric flow profile (Fig. 4). The proximal isovelocity surface area (PISA) radius was 0.32 cm, corresponding to a regurgitant flow of 2.06 ml/s (Kittleson and Brown, 2003). As cardiac output, measured using the velocity time integral of the aortic flow and the aortic end-systolic diameter on the right parasternal long-axis view (Boon, 1998c), was 4.2 l/min, the regurgitant fraction was calculated to be 2.9% confirming a slight mitral regurgitation.

Tissue Doppler Imaging examination

As the left ventricular diameters were slightly increased, quantification of radial left ventricular motion with 2-D colour TDI was performed, in order to explore more precisely the systolic function. Measurement of myocardial velocities resulting from the radial left ventricular motion was obtained with continuous ECG monitoring using the right parasternal ventricular short-axis view between the two papillary muscles. The grey-scale receive gain was set to optimize the clarity of the left endocardial and epicardial boundaries. The Doppler receive gain was adjusted to maintain optimal colouring of the myocardium, and Doppler velocity range was set as low as possible to avoid occurrence of aliasing (Nyquist limit, 21 cm/s). Digital images were obtained, stored, and reviewed later using an offline measuring system (Echo Pac for Vivid 5, GE-Vingmed Ultrasound, Waukesha, WI, USA). Measurements were made in an endocardial and epicardial segment (2×2 mm) of the left ventricular posterior wall (Fig. 5). Simultaneous endocardial and epicardial velocity profiles were obtained (Fig. 5). As previously described (Chetboul, 2002,

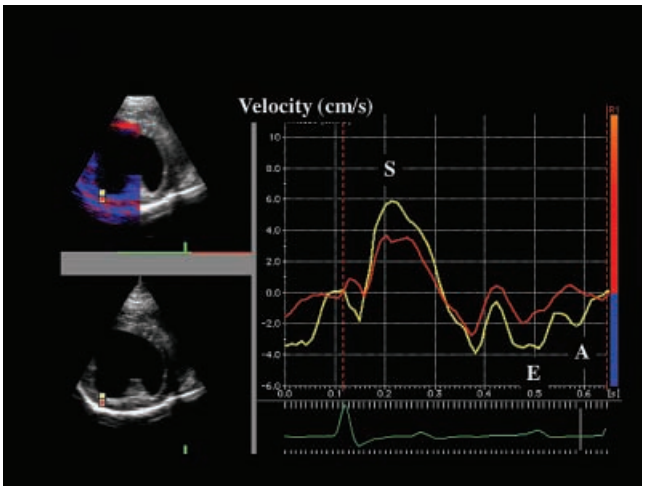

Fig. 5. Analysis of the left ventricular segmental radial motion using two-dimensional (2-D) colour Tissue Doppler Imaging (TDI) mode recorded from the right parasternal short-axis view. The simultaneous recording of the velocities in the two segments (endocardial and epicardial) shows that the endocardial layer (yellow curve) is moving with good synchrony more rapidly than the epicardial layer (red curve) during the whole cardiac cycle. The normal systolic radial myocardial velocities exclude a systolic dysfunction.

2003), the radial velocity profile included one positive systolic wave (S), and two diastolic negative waves (E and A, respectively in proto- and telediastole) with a good regional synchrony between the endocardial and epicardial layers. Both systolic and diastolic myocardial velocities were higher in the endocardial than in the epicardial layers. No alteration of the systolic radial motion was observed, with myocardial velocities of 5.7 cm/s and 3.5 cm/s in the endocardial and epicardial layers respectively (reference values: 6.0 ± 1.01 and 3.4 ± 0.77 respectively, Chetboul et al., 2003).

Discussion

Congenital malformation of the mitral valve, i.e. mitral valve dysplasia (MVD), includes multiple abnormalities of the mitral valve apparatus, such as short and thick leaflets, cleft leaflets, short and stout chordae tendinae or long and thin chordae tendinae, upward malposition of atrophied or hypertrophied papillary muscles, leaflets adhered to the septum, and insertion of a papillary muscle directly onto one or both leaflets. Abnormally formed valves usually result in regurgitation (Kittleson, 1998). In dogs, MVD has been reported most commonly in Great Danes, German Shepherds, Bull Terriers, Rottweilers, and Golden retrievers (Malik and Church, 1988; Tidholm, 1997). In contrast to mitral regurgitation, mitral valve stenosis, defined as a congenital or acquired narrowing of the mitral valve orifice, is a rare disease in dogs (Fox et al., 1992; Lehmkuhl et al., 1994; Kittleson, 1998). Mitral stenosis has been reported in Bull Terriers, New Foundlands and in other breeds, as a single lesion or in conjunction with other cardiac abnormalities, such as MVD or subaortic stenosis (Pipers et al., 1981; Fox et al., 1992; Lehmkuhl et al., 1994). Mitral valve stenosis has also been reported in cats (Stamoulis and Fox, 1993). Abnormal diastolic motion of the mitral valve in these reports include decreased leaflet separation, valve doming, concordant motion of the posterior leaflet (as opposed to the normal discordant motion in reference to the anterior leaflet), and a decreased E-F slope. Although characteristic for mitral stenosis, a decreased E-F slope may also be seen in dogs with left ventricular hypertrophy and decreased left ventricular compliance and in dogs with tachycardia (Pipers et al., 1981). To our knowledge, the presence of an apparently immobile posterior mitral leaflet, as in the dog presented here, has only been reported once before in the veterinary literature, however, with few details (Kittleson, 1998). Immobility of the posterior mitral leaflet is also considered to be a rare finding in humans with mitral valve stenosis. In humans, mitral stenosis is most often caused by rheumatic fever, which causes fusion of the valve commisures (Braunwald, 2001). It is not always possible to antemortem determine whether the abnormality of the mitral valve is acquired or congenital. In our case, a postmortem examination should have been of great interest to complete the information provided both by physical and echocardiographic findings. However, at the time of writing (6 months after the initial visit), the dog is still alive in good general condition.

Methods for evaluating the severity of mitral stenosis include measurement of atrial size, estimation of the transmitral pressure gradient, calculation of the pressure half-time, and calculation of the MVA. In our case, the finding of a normally sized LA supported the statement that the mitral stenosis was not so severe. The peak early diastolic flow velocity of 1.9 m/s corresponds to a diastolic transmitral pressure gradient of 14 mmHg, indicating increased left atrial pressure of approximately 20 mmHg, assuming left ventricular pressure in early diastole of approximately 5 mmHg. Maximum transmitral gradients reportedly ranges between 7.8 and 36 mmHg in dogs with mitral stenosis (Fox et al., 1992; Lehmkuhl et al., 1994). As the transmitral flow is influenced by the pressure gradient, other concurrent cardiac lesions, such as left-to-right shunting lesions or mitral regurgitation (as in the case presented here), may influence the pressure gradient (Hatle, 1990; Boon, 1998c). The pressure half-time, i.e. the time for the pressure gradient between the LA and ventricle to decrease to one half of the initial pressure gradient, is reportedly between 52 and 330 ms, in dogs with mitral stenosis (Fox et al., 1992; Lehmkuhl et al., 1994). In comparison with the transmitral pressure gradient, the pressure half-time has been shown to be less affected by changes in heart rate, i.e. the length of the diastolic intervals, and transmitral flow. Prolongation of the pressure half-time may, however, also be attributable to aortic regurgitation or left ventricular hypertrophy (Hatle, 1990), neither of which was present in the dog presented here. The pressure half-time can be used to estimate MVA (Gorlin and Gorlin, 1951), which is considered to be a more accurate estimate of mitral stenosis severity in humans (Messika-Zeitoun et al., 2003). In dogs, measurements of the MVA from the right parasternal short-axis view have been described, and values obtained for normal dogs (O'Grady et al., 1986; Boon, 1998c).

Pulmonary hypertension may develop as a sequel to retrograde transmission of left atrial pressure, reactive pulmonary arteriolar constriction, or secondary vascular lesions in dogs with mitral stenosis. In our case, the broadening of the colour pulmonary regurgitation jet was not consistent with a physiological pulmonary insufficiency (Rishniw and Erb,

2000). The maximal velocity of the pulmonary insufficiency flow (1.82 m/s) correlating to pulmonary arterial diastolic pressure of approximately 20 mmHg, indicated a slight-to-moderate increase in the pulmonary arterial diastolic pressure (Boon et al., 1983). As no evidence of tricuspid insufficiency was found, the pulmonary arterial pressure in systole could not be estimated in the dog in this report.

Few information are available on potential therapeutic strategies (medical treatment, interventional or surgical commissurotomy) for mitral dysplasia with stenosis in dogs. Surgical treatment of mitral valve stenosis has rarely been described (White et al., 1995). The authors reported a mitral valve replacement with a bioprosthetic valve for the treatment of congenital mitral dysplasia in a Bull Terrier, in which case 17 months post-operatively, the dog was clinically normal requiring no medication (White et al., 1995). Our group reported the successful surgical management (open mitral commissurotomy) of a mitral stenosis incorporating heart-beating cardiopulmonary bypass in a 1-year-old dog (Borenstein et al., 2004). However, in veterinary medicine, the treatment of mitral valve stenosis generally consists of medical therapy, when left-sided heart failure is present. Medical therapy in asymptomatic dogs with heart disease is, however, debatable. Treatment with vasodilators may not be beneficial before the onset of congestive heart failure, as excessive preload reduction, i.e. reduction of left atrial pressure, may significantly decrease cardiac output. However, adrenergic β-receptor blocking agents (i.e. atenolol) have been shown to significantly improve exercise capacity in humans with mitral stenosis (Klein et al., 1985). On the contrary, a recent study reported impairment of left atrial appendage emptying, which may cause local thrombus formation, with the use of atenolol in symptomatic human patients with mitral stenosis (Yilmaz et al., 2003).

In case of MVD, the prognosis is largely dependent on the regurgitant volume (Kittleson, 1998). In the case reported here, the regurgitant fraction being insignificant, the major abnormality was considered to be the stenosis of the mitral valve, rather than the regurgitation. In case of mitral stenosis, the prognosis is poor, unless the stenosis is mild (Lehmkuhl et al., 1994). Long-term prognosis must be considered as guarded for the dog in this report, although still asymptomatic, as the pressure half-time was markedly prolonged (106 ms), the E-F slope markedly decreased (4.5 m/s), and the MVA markedly reduced in comparison with the LVA.

Mitral valve stenosis, although quite rare, should be included in the differential diagnosis of dogs with congenital mitral valve malformation, especially in Bull Terriers. As previous reports on MVD in Bull Terriers did not include echocardiography or Doppler examinations, it is not possible to estimate the frequency of concomitant mitral stenosis (Malik and Church, 1988). One study reported that 10 of 15 dogs with mitral stenosis had concurrent mitral insufficiency (Lehmkuhl et al., 1994). However, difficult to detect clinically, as only four of the 15 dogs with mitral stenosis had diastolic murmurs, careful echocardiographic and Doppler examinations will reveal whether dysplastic mitral valves also may be stenotic.

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