KJVS
Korean Journal of Veterinary Service
https://doi.org/10.7853/kjvs.2023.46.1.75
pISSN: 1225-6552 eISSN: 2287-7630
Korean J Vet Serv 2023;46(1):75-79

CASE REPORT

The medical management of mitral stenosis in a Bull Terrier

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Received January 21, 2023
Revised February 21, 2023
Accepted March 8, 2023

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An eight-year-old, 28-kg male bull terrier who showed signs of lethargy and cough was referred for further evaluation of congestive heart failure. On presentation, physical examination revealed a systolic murmur at the left apex of the heart. Moreover, chest radiograph evaluation confirmed the mild alveolar and interstitial patterns in the caudal lung lobes and a grossly enlarged left atrium and left ventricle. Electrocardiography showed atrial fibrillation with a wide QRS complex, and transthoracic echocardiography revealed marked enlargement of the left atrium with abnormal morphology of the mitral valve. The thickened, hammer-like appearance and abnormal diastolic motion of the mitral valve leaflets were characterized by decreased leaflet separation and doming of the valve. The diagnosis was mitral stenosis with congestive heart failure and atrial fibrillation. The owner declined interventional valvuloplasty. Medical treatment included furosemide, pimobendan and diltiazem. Regular health check-ups have shown that vitality and clinical signs have improved considerably, and the dog have remained stable for 6 months after the presentation.

Key Words: Stenosis, Mitral valve, Congestive heart failure, Dog

INTRODUCTION

Mitral stenosis (MS) is a congenital or acquired narrowing of the mitral orifice. In dogs, it is probably congenital (Fox et al, 1992; Lehmkuhl et al, 1994). A narrowed mitral valve reduces or blocks blood flow to the left ventricle, resulting in enlargement of the left atrium and increased pulmonary capillary wedge pressure (Lehmkuhl et al, 1994).

Mitral stenosis can eventually lead to heart failure in dogs. Clinical signs include cough, dyspnea, exercise intolerance, and syncope (Lehmkuhl et al, 1994).

Diagnosis of MS includes physical examination, radiographs, electrocardiography, and echocardiography (Lehmkuhl et al, 1994; Arndt and Oyama, 2013). In dogs, treatment options include drug therapy and surgical procedures such as balloon valvuloplasty. Drug treatment consists of diuretics, angiotensin-converting enzyme inhibitors, and antiarrhythmic drugs administered to relieve symptoms (Lehmkuhl et al, 1994). The likelihood of successful long-term medical treatment of dogs with moderate to severe MS is considered low (Borenstein et al, 2004). In this study, we describe a dog with severe MS, which has been successfully treated with medication to date.

CASE

An eight-year-old, 28 kg male Bull Terrier presented to the Small Animal Clinic of the Veterinary Faculty, University of Ljubljana, to clarify the cause of heart failure. Prior to presentation, pimobendan (0.25 mg/kg, PO, twice daily), furosemide (2.0 mg/kg, PO, twice daily), benazepril (0.25 mg/kg, PO, twice daily) and spironolactone (1 mg/kg, PO, twice daily) had been prescribed by a local veterinary clinician.

At presentation, the dog was lethargic and coughing. Physical examination revealed a systolic murmur (3/6)

at the left apex and a weak pulse with deficit (80 beats/min). The dog showed mild tachypnea with a respiratory rate of 30 breaths/minute. Laboratory results showed normal red blood cell count, hemoglobin, hematocrit value, and elevated white blood cell count (15.6×10^3/μL, reference range: $5.2\sim13.9\times10^3$/μL with neutrophilia 11.82×10^3/μL, reference range: $2.21\sim10.75\times10^3$/μL), Moreover, dog displayed elevated blood urea nitrogen (BUN; 18.39 mmol/L, reference range: 2.5~9.6 mmol/L), and creatinine (195.12 μmol/L, reference range: 44.2~132.6 μmol/L) with mild hyperkalemia (5.7 mmol/L, reference range: 4.37~5.35 mmol/L).

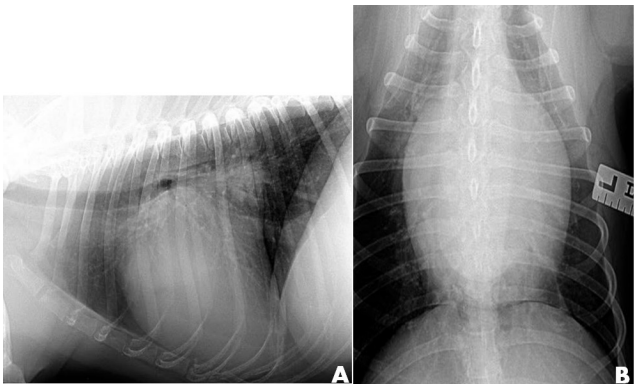

Fig. 1. (A) Left-sided cardiomegaly with prominent left atrial enlargement, elevated trachea, and mild alveolar and interstitial patterns in the caudal lung lobes. (B) Dorsoventral radiograph showing left-sided cardiomegaly and severely enlarged left atrium.

Urinalysis examination showed severe proteinuria (urine protein creatinine ratio=2.89) with normal level of urine specific gravity (1.025). Renal ultrasonography demonstrated the normal size and structure of both kidneys.

More importantly chest radiographs revealed mild alveolar and interstitial patterns in the caudal lung lobes and a marked enlargement of the left atrium with increased vertebral left atrial size (VLAS=2.5), which confirmed the diagnosis of heart failure (Fig. 1A, 1B). Electrocardiography showed atrial fibrillation (225 beats/min) with a wide QRS pattern (Fig. 2). In the echocardiographic examination, left apical four-chamber view revealed abnormal morphology of the mitral valve with thickened, hammer-like appearance of the mitral valve leaflets, and abnormal diastolic motion characterized by decreased separation of the valve leaflets and doming of the valve (Fig. 3A). Right parasternal four-chamber long-axis view showed severe left atrial enlargement (LA) (LA/Ao=2.66: reference value<1.6), and moderate left ventricular end-systolic enlargement (Fig. 3B). Also, increased mitral valve inflow velocities (E wave=2.45 m/s) with a fused A wave (1.28 m/s), prolonged deceleration time (322 m/s) with a turbulent pattern were noted from the left apical view (Fig. 3C). Doppler color flow imaging showed a severe mitral regurgitation jet (>50% of

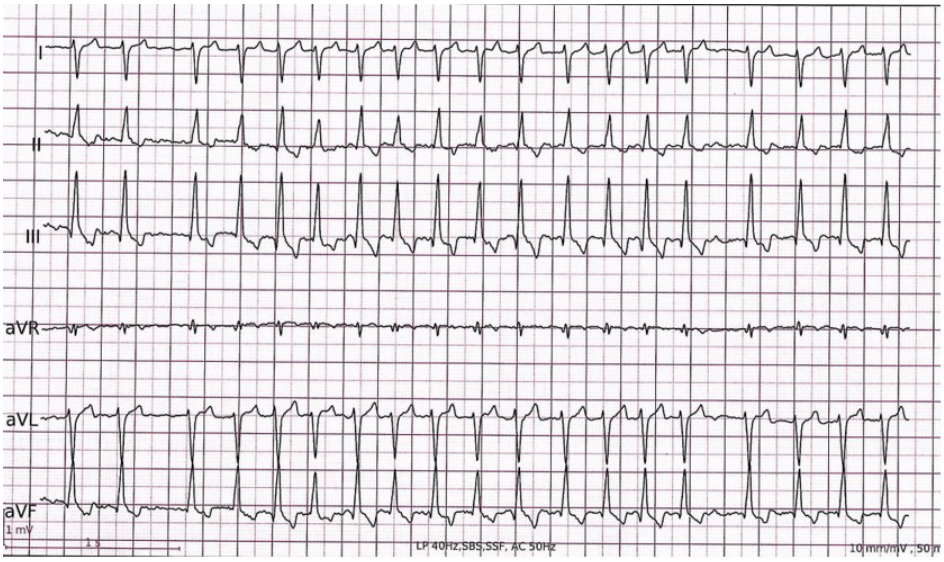

Fig. 2. Electrocardiography showing atrial fibrillation (225 beats/min) with a wide QRS pattern.

https://doi.org/10.7853/kjvs.2023.46.1.75

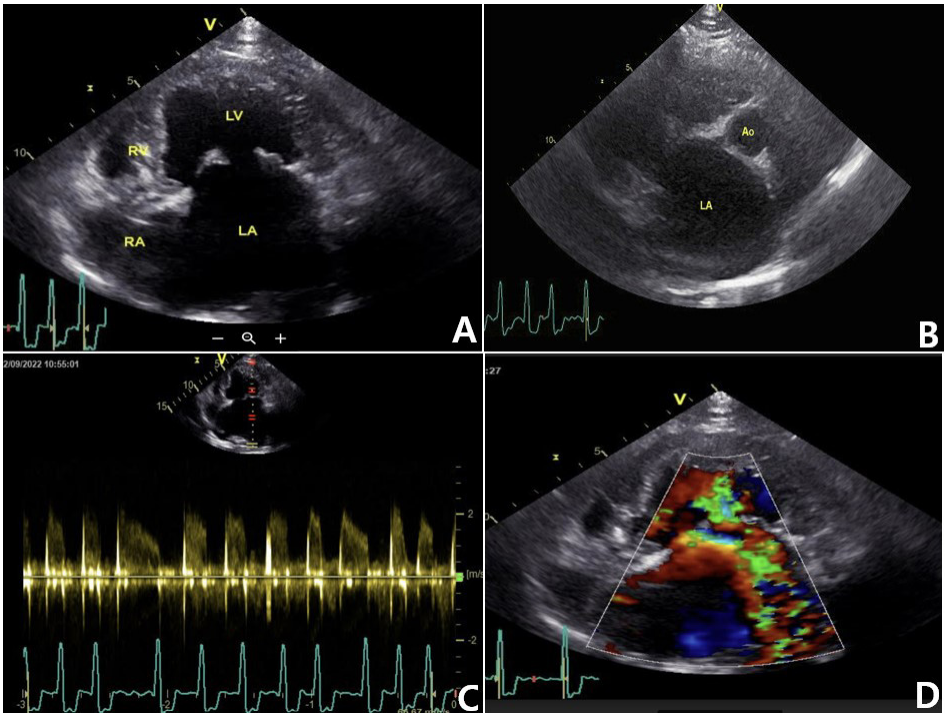

Fig. 3. (A) Left apical view showing left atrial enlargement and mitral valve leaflets thickening and diastolic doming. (B) Right parasternal four-chamber long axis view showing severe left atrial enlargement (LA/Ao=2.66). (C) Pulsed-wave Doppler pattern with high velocity fused E and A wave mitral inflow. (D) Severe mitral regurgitant area and a turbulent inflow jet within the left ventricle.

LA) and a turbulent jet in the left ventricle due to mitral stenosis (Fig. 3D).

Based on the above findings, the dog was diagnosed with mitral stenosis with congestive heart failure (ACVIM stage C2) and atrial fibrillation. Medical treatment included furosemide (2.1 mg/kg, PO, twice daily), pimobendan (0.3 mg/kg, PO, twice daily), and diltiazem (1.1 mg/kg, PO, three times daily). Due to initial inadequate reduction of heart rate after diltiazem administration (190 beats/min), the dose was increased to 2.1 mg/kg, three times daily. Digoxin was avoided because of the risk of renal failure. Interventional valvuloplasty was recommended, but the owner declined this option despite presentation of bad prognosis in case of medical treatment only. In addition, it is expected that renal function will worsen on long term diuretic therapy. Vital and clinical signs were improved over the time and the dog have remained stable 6 months after the first presentation. More importantly, the disease severity has never been worse during regular follow-ups, which included physical examination, radiographs, electrocardiography, and echocardiography.

DISCUSSION

Mitral stenosis is a congenital or acquired mitral valve disease that results in narrowing of the mitral orifice. Although MS is common in humans, it rarely occurs in dogs or cats.

In dogs, Boston terriers, Newfoundlands, and Bull Terrier have been found to be predisposed to this condition (Tidholm et al, 2004; Winter et al, 2022). Cardiac murmurs include left apical soft diastolic murmurs and systolic murmurs caused by mitral regurgitation (Lehmkuhl et al, 1994).

In the present diagnosis approach, only a systolic murmur (3/6) was noted at the left apex on physical examination. In general, MS is characterized by diastolic murmur, however, diastolic murmur may be difficult to auscultate when accompanied by mitral regurgitation with systolic murmur.

Laboratory results showed an elevated white blood cell count. Increased white blood cell with neutrophilia has been noted in dogs with advanced-stage heart failure, which is due to the presence of systemic inflammation (Domanjko Petrič et al, 2018). Radiographic

examination has shown a severe enlargement of the left atrium which is the most prominent feature together with mild alveolar and interstitial patterns in the caudal lung lobes (Lehmkuhl et al, 1994). Restriction of left ventricular filling leads to an increase in left atrial pressure and dilation of the left atrium, pulmonary veins and pulmonary edema (Guglielmini et al, 2020).

Electrocardiographic examination can reveal atrial and ventricular arrhythmias (Lehmkuhl et al, 1994). Atrial fibrillation develops most frequently when there is significant atrial enlargement (Guglielmini et al, 2020) as confirmed in our case. In the study by Lehmkuhl et al. (1994), diltiazem and digoxin were successfully prescribed for atrial fibrillation, however in our case, only diltiazem was administered due to cardio-renal syndrome.

In our case, increased BUN, creatinine and potassium might have been a result of diuretic therapy or the existing cardiac and/or renal disease.

The final diagnosis of MS is usually confirmed by 2-D, M-mode, spectral, and color flow Doppler (Boon, 2011). In general, echocardiography reveals thickened mitral valves, poor leaflet separation, decreased left ventricular ejection fraction, and increased mitral E-wave pressure half-time (Fox et al, 1992; Lehmkuhl et al, 1994). 2-D echocardiograms show abnormal diastolic motion of the mitral valve characterized by decreased separation of the valve leaflets, doming of the valve, and concordant motion of the parietal mitral valve leaflet (Arndt and Oyama, 2013) with increased mitral valve inflow velocities and prolonged pressure half-times (Lehmkuhl et al, 1994). In our case, 2-D echocardiography showed abnormal morphology of the mitral valve with thickened mitral valve leaflets and abnormal diastolic motion of the mitral valve characterized by decreased separation of the valve leaflets and doming of the valve. In addition, color flow Doppler showed a severe mitral regurgitation jet, which was due to mitral regurgitation, and a turbulent jet in the left ventricle, which was due to mitral stenosis. We hypothesized that this dog might also have primary mitral valve dysplasia that led to mitral stenosis over time due to its late presentation and severe mitral regurgitation.

In humans, treatment includes medications such as diuretics, antiarrhythmics (i.e. beta-blockers), and surgical procedures such as balloon valvuloplasty, mitral commissurotomy, and mitral valve replacement (Sunamori et al, 1983; Borenstein et al, 2004; Winter et al, 2022). In dogs with MS, medical management of heart failure includes administration of furosemide, enalapril, spironolactone, atenolol, and pimobendan (Winter et al, 2022).

A combination therapy of furosemide, pimobendan, and diltiazem was prescribed for the treatment of heart failure and cardiac arrhythmias in our bull terrier. Although helpful in treating heart failure and atrial fibrillation, benazepril, spironolactone and digoxin were excluded from the prescription because of the presence of a cardio-renal syndrome in this dog, most likely due to decreased cardiac output resulting from decreased ventricular filling. Regular health check-ups have shown that vitality and clinical signs have improved considerably, and the dog have remained stable for 6 months after the presentation.

MS in humans is usually acquired secondary to endocarditis, rheumatic fever, or other causes (Chambers and Bridgewater, 2013). It is a congenital or acquired narrowing of the mitral orifice that is probably congenital in dogs (Fox et al, 1992; Lehmkuhl et al, 1994). It is not known whether this MS is congenital or acquired. Given the late onset of congestive heart failure, we would consider an acquired disorder on a primary congenital dysplastic valve.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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https://doi.org/10.7853/kjvs.2023.46.1.75

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