CASE REPORT
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MITRAL VALVE DISEASE IN A GERMAN SHEPHERD DOG – DIAGNOSIS AND MANAGEMENT

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ABSTRACT

Mitral valve disease is an acquired degenerative valvular heart disease and most commonly seen in geriatric small dog breeds and less common in other breeds. The present paper reports the diagnosis and management of mitral valve disease in a nine year old German Shepherd dog. Auscultation of heart revealed (5/6) grade holosystolic murmur over the mitral valve region. Weak femoral pulse and pulse deficit was observed. Electrocardiography showed ventricular premature complex (VPC) and echocardiography confirmed the diagnosis of mitral valve disease. The dog was treated with pimobendan, enalapril, furosemide and spironolactone.

Keywords: Mitral, Enalapril, Pimobendan

INTRODUCTION

Mitral valve disease is a most common acquired heart disease reported in small breeds of geriatric dogs. This disease occurs due to the changes in the collagen content and the alignment of collagen myofibrils in the mitral valve leaflets. Due to the degenerative changes, valvular leaflets are thickened which leads to improper coaptation, ends with backward regurgitation of blood and enlargement of left atrium and ventricle in the later stages of disease. Histologically, myxomatous degeneration is characterized by absence of inflammatory infiltrate, proliferation of valvular interstitial and endothelial cells, abnormal deposition of extracellular matrix and disorganization of the collagen network (Atkins *et al.,* 2009).

CASE HISTORY AND OBSERVATION

A nine year old male German Shepherd dog, weighing 33 kg was presented with the history of difficulty in respiration and cough. Vaccination and deworming history were up to date.

Clinical examination revealed normal rectal temperature of 102.1°F, pale roseate mucous membrane and nasal discharge.

Auscultation of heart revealed high intensity (grade, 5/6) holosystolic murmur over the mitral valve region. Capillary refilling time was increased to 4 seconds. Respiration was dyspnoeic and the rate was increased (65 breaths per minute). Lung auscultation revealed exaggerated breath sounds and crackles. Weak femoral pulse with pulse deficit was noted. The heart rate was about 130 beats per minute. Rhythm was irregular. There was exercise intolerance and symptoms were present even at rest. Complete blood count values were within the normal range. Biochemical studies revealed a serum creatinine level of 1.7 mg/dL. Right lateral view of thoracic radiography revealed left atrial enlargement, perihilar distribution of pulmonary oedema and apical pulmonary venous congestion (Fig. 1). Electrocardiography showed ventricular premature complex (Fig. 2) as per Smith *et al.* (2016).

Echocardiography was used to find out anatomical changes in the heart and advanced Doppler technique was used to find out regurgitation and blood flow velocities inside the cardiac chambers through mitral valve. Mild tricuspid valve regurgitation was also recorded. Two-dimensional echocardiography revealed gross enlargement of both left atrium and ventricle, thickened mitral valve leaflets (Fig. 3) and increased left atrium to aorta ratio which suggested severe left atrial enlargement (LA: 5.83cm Ao:2.35cm LA/Ao ratio: 2.48; Normal <1.6). (Fig. 4).

M-mode echocardiographic measurements of left ventricle were used to find out systolic function of heart. Ventricular dilation was characterised by increased diameter of left ventricular internal diameter at diastole (LVIDd-58mm) and systole (LVIDs-37.9mm) and fractional shortening (35%). End diastolic volume, end systolic volume, stroke volume and ejection fraction were 166.5ml, 61.6ml, 104.9ml and 65% respectively. Hyperdynamic movements of interventricular septum and left ventricular posterior wall and increased ejection fraction and fractional shortening (Fig. 5) were observed in right parasternal long axis view of the heart (Atkins *et al.*, 2009; Summerfield, 2018).

Doppler echocardiography showed severe mitral valve and mild tricuspid valve regurgitation (Fig. 6). Transmitral flow velocity was measured by pulsed wave Doppler echocardiography (E wave: 0.9 m/s and A wave: 0.38 m/s). Regurgitation velocity of mitral valve was measured by using continuous wave Doppler echocardiography technique (5.3 m/s) (Fig. 7). Based on radiography,

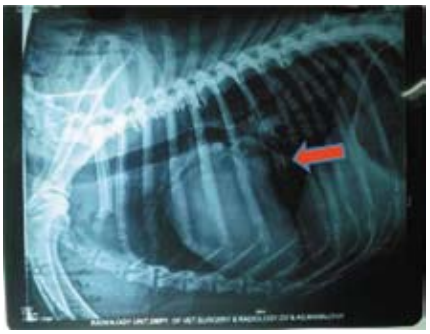

Fig. 1. Thoracic Radiography-Right lateral view showing left atrial enlargement

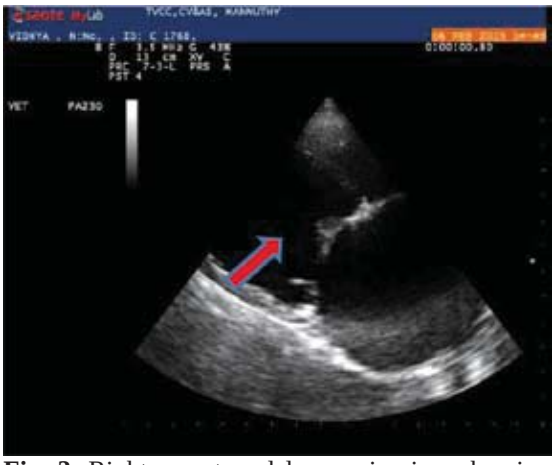

Fig. 3. Right parasternal long axis view showing thickened mitral valve leaflets

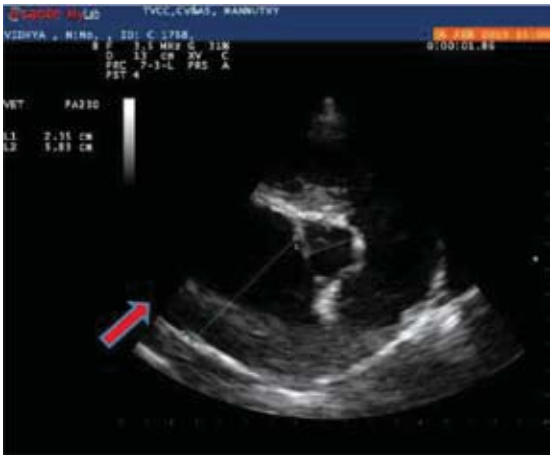

Fig. 4. Right parasternal short axis view showing left atrial enlargement

electrocardiographic and echocardiographic studies the case was diagnosed as of mitral valve disease stage D (Keene *et al.*, 2019).

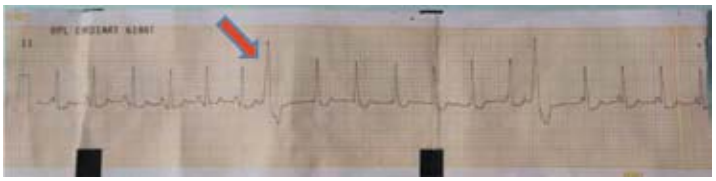

Fig. 2. Electrocardiography (Lead II, 25mm/s, 10mv) showing ventricular premature complexes

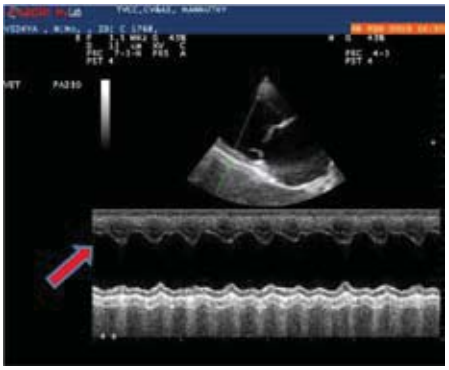

Fig. 5. M-mode echocardiography

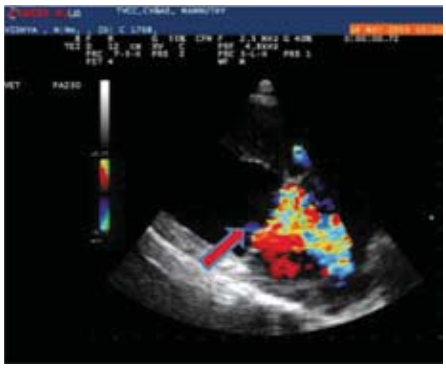

Fig. 6. Colour Doppler echocardiography showing mosaic pattern of colours at mitral valve indicating severe regurgitation

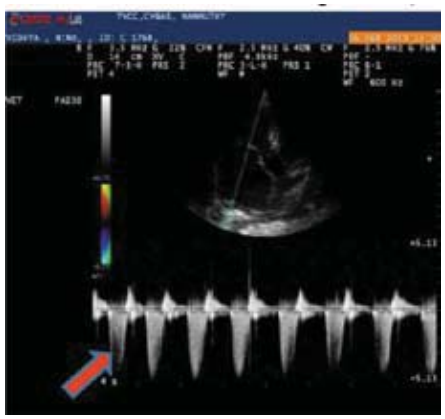

Fig. 7. Continuous wave Doppler showing mitral valve regurgitation velocity

TREATMENT

In hospital, treatment started with furosemide @ 2mg/kg bw as intravenously to reduce pulmonary oedema. Oral therapy was started with enalapril @ 0.5 mg/kg bw q12h, furosemide @ 2mg/kg bw q12 h and cardiac supplement Vencard ® Venky's two tablets. After initiation of treatment animal showed improvement in the condition. Spironolactone was combined with furosemide after seven days to control adverse effects of RAAS system along with enalapril and Vencard and the treatment was continued. Renal functions and electrolytes were monitored every fourteen days and the creatinine values were in the higher reference level. After three months animal was presented with the complaint of mild inappetence and weakness. Then the inodilator, pimobendane @ 0.25mg/kg bw q12h and bronchodilator deriphyllin @ 10mg/kg bw q12h were also added to the therapeutic regimen. These drugs will reduce electrolyte abnormalities and delay the cardiac remodelling, thereby improving the quality of life (Boswood, 2018). Moderate salt restriction was advised because it has lesser effect on RAAS activation, and potentiate angiotensin converting enzyme inhibitor (Jordan, 2003).

Improvement was noticed and the patient had a good quality of life for more than one year.

SUMMARY

A nine-year-old male German shepherd dog, weighing 33 kg was presented with the history of difficulty in respiration and cough. Mitral valve disease was diagnosed based on clinical examination and using diagnostic aids including electrocardiography, radiography and echocardiography. The patient was treated with pimobendan, enlapril, furosemide and spironolactone. These drugs slowed the progression of cardiac remodelling and improved quality of life by alleviating symptoms.

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