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Incomplete right bundle branch block associated with coagulation disturbance in mitral valvular disease in a dog – case report

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Abstract

An incomplete right bundle branch block (RBBB) associated with coagulation disturbance in mitral valvular disease was diagnosed in a five-year-old female Dachshund. Morphologically moderate mitral valvular thickening and nodularity could be noticed on echocardiography. Prolonged activated partial thromboplastin time and thrombocytopaenia supported the probable underlying mechanism for myocardial infarction and RBBB. The animal showed clinical improvement after one month of therapy with enalapril and torasemide for mitral valve insufficiency.

Keywords: *Right bundle branch block, mitral valve, infarction*

Right bundle branch block (RBBB) is a condition where there is a delay or obstruction in the transmission of impulses through right bundle of His and hence the right ventricle is activated by the spread of impulse from left ventricle (Fauchier *et al.*, 2003). Bundle branch block (BBB) accompanies cardiomegaly, ventricular failure and pulmonary oedema with chronic progressive heart failure being the common recognizable inciting factors for BBB (Bauer, 1964). Bundle branch block pattern may be produced by either or all the three mechanisms including anatomic disruption of conducting bundle, ventricular hypertrophy and ischemia or functional or neurological disturbance with or without underlying cardiac pathology. Even though the RBBB was seen

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commonly in healthy normal dogs, rarely it is being reported in chronic mitral fibrosis. Higher incidence of ventricular fibrillation and sudden cardiac death was noticed in humans with RBBB and ST segment elevation in right precordial leads and the predominant substrate underlying the electrocardiographic (ECG) pattern was arrhythmogenic right ventricular cardiomyopathy (ARVC) (Gandjbakhch *et al*., 2018). Recent studies established a striking similarity between ARVC in humans and Boxer cardiomyopathy with regard to histopathological lesions and pathophysiology (Basso *et al.,* 2004). Similar cases were reported in other breeds also. The present case is about a chronic mitral valvular disease with concurrent RBBB and coagulation disturbance.

A five-year-old female Dachshund was presented with a complaint of dyspnoea during night time and exercise intolerance since two months. The animal was obese. On thoracic auscultation, systolic murmur was detected. The mucous membrane was pale roseate and capillary refill time was two seconds. The respiratory rate and pattern were normal at rest. Left lateral thoracic radiograph demonstrated tracheal elevation and interstitial pneumonic pattern in cranial lung lobe. Electrocardiographic studies showed incomplete RBBB pattern (Fig.1) with elevation of ST segment. Heart rate was 107 bpm. Two-dimensional echocardiography revealed hyperkinetic interventricular septal motion in M mode echocardiography. The end diastolic wall thickness, left ventricular internal diameter in systole and diastole, left atrium to aortic root ratio (LA : Ao), E point to septal separation, ejection fraction (EF), fractional shortening (FS) and E/A ratio were 7.6 mm, 11.9 mm, 23.5 mm,1.55, 0.2 mm, 60 per cent, 32 per cent and 1.3 respectively. Nodular thickening of the mitral valve leaflets could be observed and color Doppler study showed moderate mitral and mild tricuspid regurgitation (Fig 2). Complete blood count revealed mild anaemia and thrombocytopaenia. Haemoparasites were not detected during examination of stained peripheral blood smear. The activated partial thromboplastin time was elevated in the initial presentation (17.22 sec). The mean systolic and diastolic blood pressure values were 153- and 82-mm Hg, respectively.

Treatment was initiated for mitral valve insufficiency with oral administration of enalapril at 0.5 mg/kg BID and torasemide at 0.2 mg/kg OD. Clinical improvement was noticed after one month of therapy. After one month, RBBB with ST segment elevation was still observed in lead II electrocardiogram (Fig. 3). The aPTT was reduced to 14.52 sec by one month of therapy. The reduction of severity of mitral regurgitation and LA : Ao ratio (1.45) were observed by the end of one month of therapy.

Although reports document that RBBB is a common finding in healthy normal dogs, over the years, conflicting data have emerged out, regarding the long-term prognostic effect of RBBB for identifying subclinical cardiac diseases based on a small cohort of patients (Harkness and Hicks, 2020). The presence of incomplete RBBB in this case might be secondary to the subtle myocardial changes developed from valvular disease. The elevated aPTT observed in this case supported the probability of occurrence of myocardial infarction and the subsequent development of RBBB. This observation could be supported with the report of Bussink *et al*. (2013) who documented that RBBB was associated with higher risk of myocardial infarction and pacemaker implantation, but not

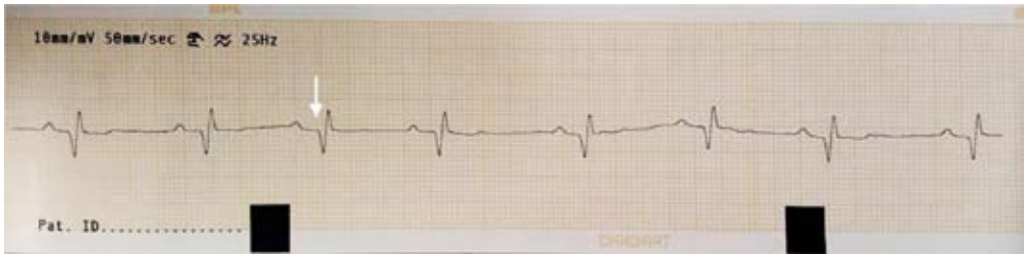

Fig. 1. Lead II ECG showing right bundle branch block (white arrow) pre-treatment (50mm/sec, 10mm=1mV)

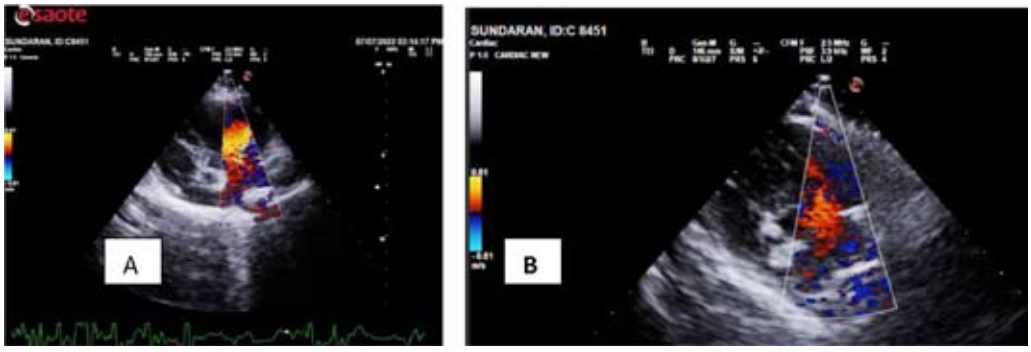

Fig. 2. Colour Doppler echocardiography at four chamber view. Comparison of mitral regurgitation before and after treatment in MVD (A: pre-treatment; B: post-treatment)

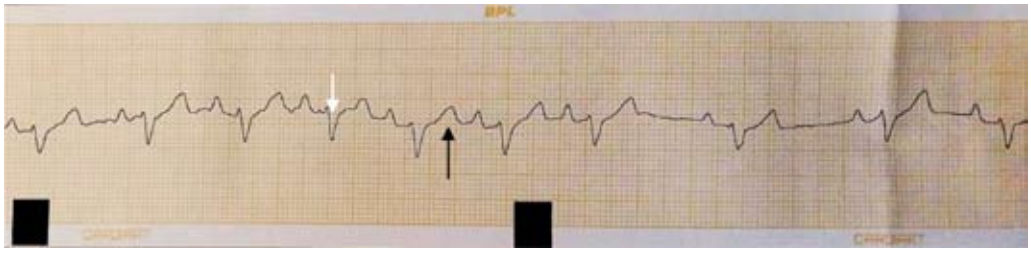

Fig. 3. Lead II ECG showing right bundle branch block (white arrow) and ST segement elevation (black arrow) post-treatment (50mm/sec, 10mm=1mV)

with chronic heart failure. Generally, incomplete RBBB was not associated with increased risk. Although, Dou *et al.* (2008) reported that, higher stress and oxygen demand developed in the interventricular septum in RBBB led to myocardial infarction, the findings of the present case suggested that the platelet aggregation during the mitral valvular disease might be the reason for the myocardial infarction and subsequently RBBB. This could be attributed to elevated aPTT and thrombocytopaenia before treatment.

The post treatment reduction of aPTT and clinical improvement noticed in the present study could be attributed to the low-risk involvement of RBBB.

The ECG and echocardiographic findings of this case correlated well with the findings by Tilley (1992). Hyperkinetic interventricular septal motion observed in M mode echocardiography might be due to the exceeding of left ventricular pressure over right ventricular pressure thus causing increased septal excursion.

Rotman and Triebwasser (1975) opined that impairment of intraventricular conduction system due to myocarditis could act as an etiological factor for BBB and the prognosis of the condition depends upon the presence or absence and the degree of severity of the associated cardiovascular disease. Birnhaum and Nikus (2020) reported that right bundle branch block could be a marker for underlying subclinical cardiac conditions, including diastolic dysfunction with preserved systolic function. However, further studies are needed to support this finding. Thus, the current case report of incomplete RBBB associated with coagulation disturbance added an important piece of information to solve the mystery about the underlying pathophysiologic mechanism and long-term significance of RBBB in patients with asymptomatic heart disease. However, further studies are needed to support this finding.

Summary

An incomplete RBBB associated with mitral valve disease in dogs is reported. Right bundle branch block developed in this case might be due to platelet aggregation which subsequently developed into myocardial infarction.

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Conflict of interest

The authors declare that they have no conflict of interest.

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