



Management of syncope associated with ventricular premature contractions in a dog

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Abstract

A four-year-old male Golden Retriever dog was presented with a history of episodes of syncope. Electrocardiography (ECG) revealed atrio-ventricular dissociation and monomorphic ventricular premature contractions (VPCs). Echocardiography revealed moderate left ventricular hypertrophy. The animal responded initially with the treatment of anti-arrhythmic drug, Sotalol at the dose rate of 0.5 mg/kg body weight BID along with Enalapril, diuretics and antibiotics. However, after few months, the animal showed peaked T waves in ECG, indicating myocardial hypoxia and the Sotalol dose was increased to 1 mg/kg body weight BID. The animal again showed an attack of syncope with VPCs, six months after the initiation of treatment and responded well with the combination therapy of Sotalol at the rate of 1 mg/kg BID and Mexiletine at the rate of 4 mg/kg BID.

Keywords: Ventricular premature contractions, left ventricular hypertrophy, Sotalol and Mexiletine

Syncope is a sudden loss of consciousness associated with loss of postural tone, from which recovery is spontaneous. This is caused by transient cerebral hypoxia due to anaemia, respiratory tract diseases like tracheal collapse and cardiac diseases. Distinguishing syncope clinically from seizure will be difficult. Collapse episodes precipitated by excitement, exercise, stress, cough, vomiting, micturition, defaecation, and pain are most likely due to syncope. Altered mentation before and after the episode occurs most commonly in seizures. The most common cause of syncope is cardiac arrhythmias. Sick sinus syndrome, advanced atrioventricular block, atrial standstill, and ventricular tachycardia are common arrhythmias that lead to syncope in dogs (Fuentes et al., 2010).

A four-year-old male Golden Retriever dog was presented with the history of episodes of syncope at one-month intervals. The animal was under treatment with oral Phenobarbital sodium at a dose rate of 2 mg/kg Bid orally. The dog was apparently healthy and the vital parameters were (Body temperature 101 °F, pulse rate 110/minute and respiratory rate 40/minute) within the normal range. Thoracic auscultation revealed exaggerated breath sounds. Blood smear and wet film examinations were negative. Complete blood count showed values (Total leucocyte count $7.11 \times 10^3/\mu\text{L}$, erythrocyte count $7.61 \times 10^6/\mu\text{L}$ and platelet count $203 \times 10^3/\mu\text{L}$) within the normal range. Serum biochemistry revealed normal glucose (100 mg/dL), creatinine (1.17 mg/dL), alanine aminotransferase (35 U/L) and alkaline phosphatase (28 U/L) values.

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Serum electrolytes showed slightly elevated sodium - 155 mEq/L, chloride- 134 mEq/L and potassium - 5.8 mEq/L when compared to the normal range. The serum creatine phosphokinase (CPK) value was (286U/L) was elevated. Radiography showed no gross cardiac enlargement, bronchial pattern, and mild oedema of lungs (Fig.1). Electrocardiography (ECG) revealed atrio-ventricular dissociation and frequent monomorphic ventricular premature contractions (VPCs) as shown in Figure 2. Echocardiography revealed moderate hypertrophy of the left ventricular free wall and interventricular septum (Fig.3). Mild regurgitation was noticed at the mitral and tricuspid valve levels on colour Doppler and pulse wave Doppler echocardiographic examinations. Interventricular septum thickness during diastole (IVSd) was increased, and the systolic functions were within the normal range (Table 1).

Table 1. M-mode echocardiographic values (Right parasternal short axis view)

Parameters	Values on the first day of presentation	Values 6 months after initiation of treatment	Values of healthy adult Golden retriever dogs (Bagardi <i>et al.</i> , 2025)
LVIDd (cm)	3.94	3.0	3.46-5.80
LVIDs (cm)	2.70	2.15	2.02-3.63
IVSd (cm)	1.31	1.17	0.94 ±0.150
IVSs (cm)	1.31	1.43	1.32± 0.209
LVPWd (cm)	0.83	0.98	0.952± 0.203
LVPWs (cm)	1.18	1.43	1.324±0.275
FS (%)	31.58	28.26	20.0-49.0
EF (%)	60.14	56.21	38.64-78.11

LVIDd- Left ventricular internal dimension diastole, LVIDs- Left ventricular internal dimension systole, IVSd – Inter-ventricular septum diastole, IVSs - Inter-ventricular septum systole, LVPWd- Left ventricular posterior wall diastole, LVPWs- Left ventricular posterior wall systole, FS- Fractional shortening, EF – Ejection fraction.

The animal was treated with twice daily oral dosing of sotalol at a low dose of 0.5 mg/kg body weight, Enalapril at the rate of 0.25 mg/kg body weight, and the tablet, Lasilactone (combination of Furosemide and Spironolactone) at the rate of 2mg/kg body weight, along with two weeks of antibiotic therapy with Amoxicillin clavulanate at the rate of 12.5 mg/kg body weight BID. The animal showed clinical improvement, and the ECG was devoid of any ventricular premature contractions after the initiation of treatment (Fig.4). The dose of phenobarbital sodium was tapered and stopped. The animal was clinically normal without any further episodes of syncope. However, the ECGs taken 4 months after initial presentation showed peaked T waves indicating myocardial hypoxia (Fig. 5). No more VPCs or atrio-ventricular dissociations were observed. The dose of Sotalol was then increased to 1 mg/kg BID orally daily along with other drugs. The animal remained asymptomatic with a normal ECG. However, after 6 months from the initial presentation, the animal again presented with episodes of syncope. The ECG then showed frequent monomorphic VPCs (Fig.6). The complete blood count values were within the normal range. Serum biochemistry revealed normal creatinine (1.08 mg/dL), sodium (142 mEq/L), chloride (108 mEq/L), potassium (4.2 mEq/L) and total thyroxine (1.93µg/dL) values. However, the creatine phosphokinase (CPK) values were elevated (286 U/L) when compared to the normal range (10- 200 U/L). Echocardiography still revealed moderate hypertrophy of the left ventricle (Fig.7). Another antiarrhythmic agent, mexiletine at the rate of 4 mg/kg BID orally, was added with Sotalol at the rate of 1 mg/kg body weight BID, along with Enalapril and Lasilactone at the previous doses. The animal remained asymptomatic and an ECG taken two weeks after starting sotalol and mexiletine was found to be normal without any VPCs or signs of myocardial hypoxia (Fig.8). The animal was not presented for further investigations. Upon enquiry, the animal continues to be asymptomatic and was advised to continue the same treatment.

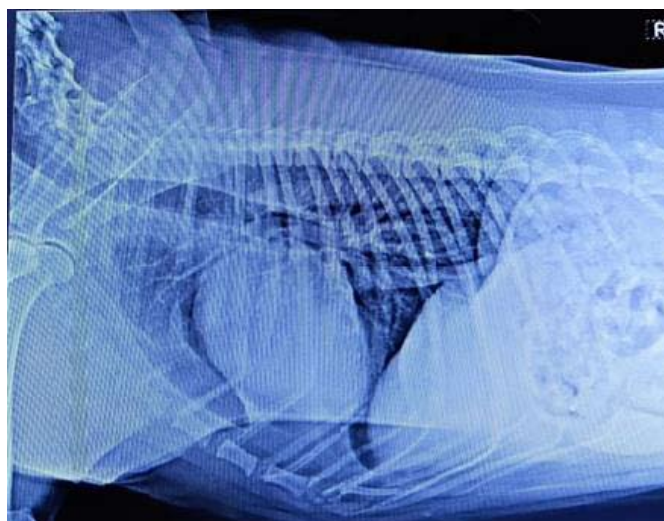


Fig.1. Right lateral radiography showing bronchial pattern and mild lung oedema

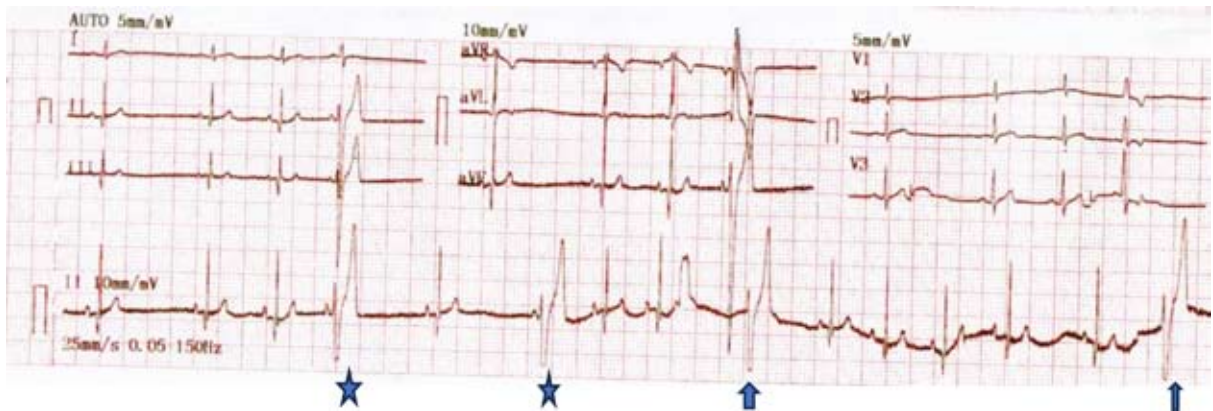


Fig.2. Electrocardiogram showing atrio-ventricular dissociation(stars) and ventricular premature contractions (arrows) – Before treatment

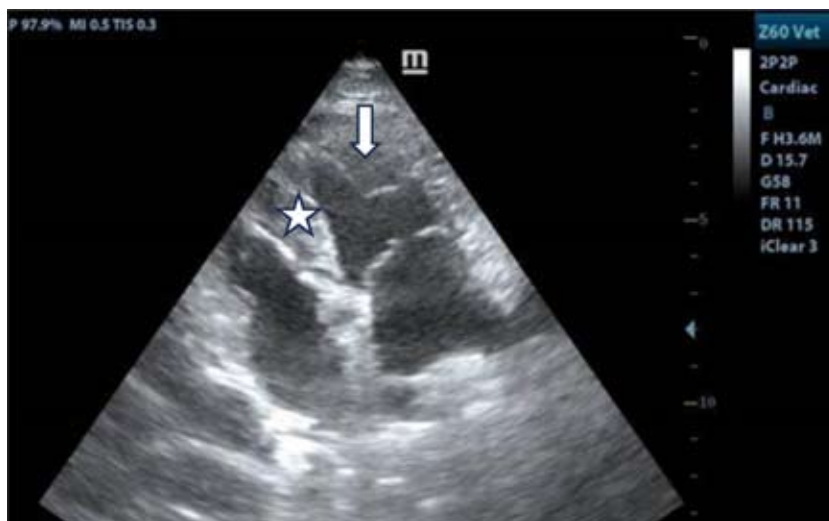


Fig.3. Echocardiography – Left apical view showing thickening of inter-ventricular septum(star) and the free wall of the left ventricle (arrow)



Fig.4. Electrocardiogram showing normal sinus rhythm without any ventricular premature complexes- one week after sotalol treatment

Atrio-ventricular dissociation is an electrocardiographic diagnosis in which atria and ventricles are depolarised independently and is a sign of impending ventricular arrhythmia. Here, the P wave and QRS complexes are not connected with normal P-R intervals. The Ventricular premature contractions are ventricular arrhythmia, originating from abnormal ectopic foci in the ventricular myocardium and are regarded as the most common pathological arrhythmias in small animals. This is characterised by an abnormal shape or 'bizarre' QRS complex not associated with a preceding P wave. Myocardial affections due to acquired or congenital heart diseases, hypoxia, electrolyte disturbances, especially

hyperkalaemia, will precipitate VPCs (Fuentes *et al.*, 2010).

In the present study, except for creatine phosphokinase (CPK) or creatine kinase (CK), all the serum biochemical values were almost within the normal limits. Creatine phosphokinase values will be increased in skeletal muscle and myocardial disorders, in kidney diseases and in certain medications. Estimation of specific isoenzyme, CK-MB, for myocardial injury would have been more appropriate in this type of study (Roberts *et al.*, 1975). Hypertrophic changes of the left ventricle and Peaked T waves in ECG suggest myocardial hypoxia as the probable

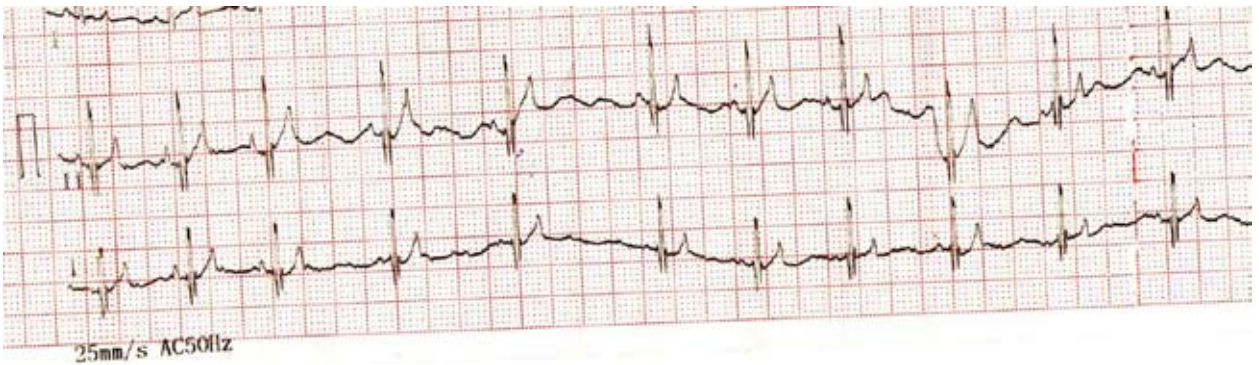


Fig. 5. Electrocardiogram showing peaked T waves (arrows)- with no VPCs - 4 months after starting sotalol treatment

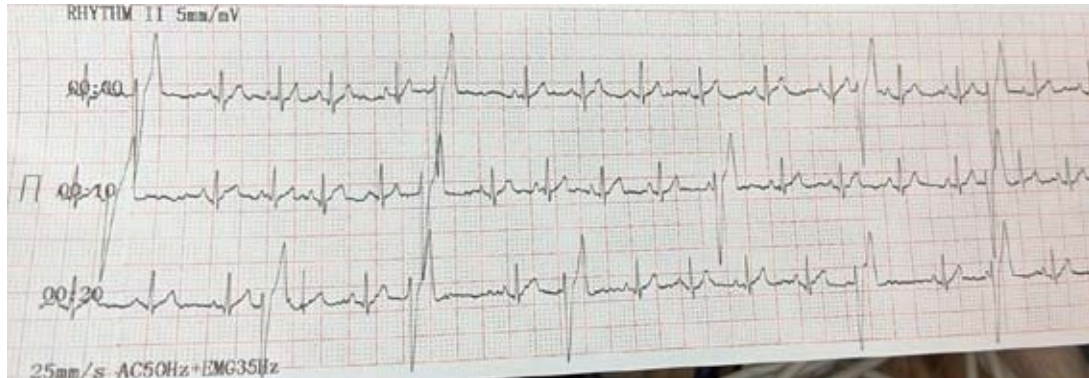


Fig. 6. Electrocardiogram showing ventricular premature contractions - 6 months after starting treatment. The animal showed recurrence of syncope.



Fig. 7. Echocardiography- Right parasternal short axis at papillary muscle level showing moderate left ventricular hypertrophy. Six months after initial presentation.

cause for ventricular premature contractions in the present study. However, the cause for cardiac hypertrophy was not elucidated with the available investigations in the present study. Even though echocardiography is not sensitive to identify subtle valve abnormalities, mitral valve thickness, systolic anterior motion of the septal mitral valve leaflet, mitral and tricuspid regurgitations in colour flow doppler were observed in echocardiography of dogs with hypertrophic cardiomyopathy (Schober *et al.*,2022). Mild atrio-ventricular valvular insufficiency associated with myocardial hypertrophy was also evident during echocardiography in the present study.

Even though the recommended oral dose of the antiarrhythmic drug, Sotalol, in dogs is 1 to 2.5 mg/kg P.O every 12 hours, administration of a lower starting dose is safe due to severe bradycardia and weakness of the animal at higher dose ranges (Mathews *et al.*,2025). Combination therapy of Sotalol (0.5 mg/kg body weight BID) and Mexiletine (4 mg/kg body weight BID) was effective in controlling the ventricular premature contractions in dogs (Daniel *et al.*,2023). In the present study, the combination of Sotalol (1 mg/kg BID) and Mexiletine (4 mg/kg BID) was also found to be effective in controlling the ventricular arrhythmia. Enalapril, diuretics and antibiotics were given

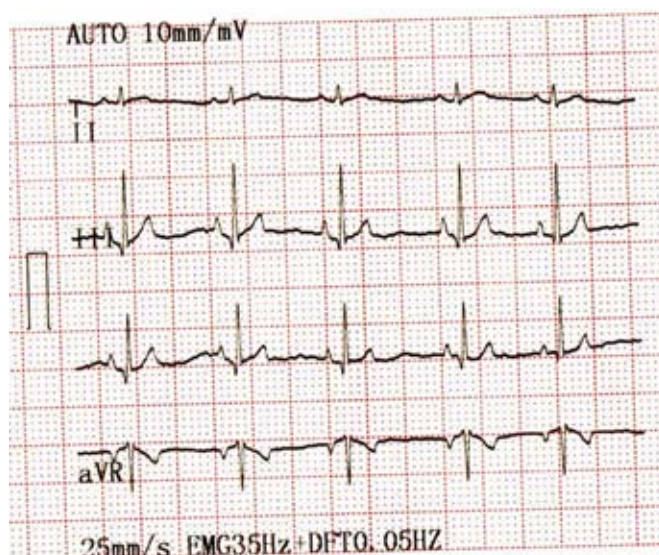


Fig.8. ECG after stabilisation with oral Sotalol 1 mg/kg BID and Mexiletine 4 mg/kg BID- Normal sinus rhythm without VPCs and signs of myocardial hypoxia.

to prevent congestive heart failure signs and secondary bacterial infections. Tall and broad-based symmetrical T waves in ECG are considered an early sign of acute myocardial ischemia in dogs (Reshma *et al.*, 2024). So, ECG signs like peaked T waves should be observed regularly during treatment to prevent the recurrence of ventricular arrhythmias.

Summary

In electrocardiography, life-threatening ventricular premature contractions were diagnosed in a dog which showed the clinical sign of syncope. On echocardiography, the animal had moderate left ventricular hypertrophy. The arrhythmia and syncope were controlled effectively by using a combination therapy of oral Sotalol and Mexiletine.

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Conflict of interest

The authors declare no conflict of interest

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