pISSN 1598-298X / eISSN 2384-0749
J Vet Clin 34(5) : 353-355 (2017)
http://dx.doi.org/10.17555/jvc.2017.10.34.5.353

Journal of Veterinary Clinics
www.ksvc.or.kr

Long Term Management of Congenital Ventricular Tachyarrhythmia in a Dog

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(Received: July 26, 2017 / Accepted: August 21, 2017)

Abstract : A four month old, intact female Jindo dog presented to the Veterinary Teaching Hospital of Kangwon National University with collapse. Physical examination revealed a heart rate of more than 200 beats per minute. Blood tests showed mild anemia and mild neutrophilia, while thoracic radiography and ultrasonography revealed no remarkable findings. Electrocardiography showed ventricular premature complexes (VPCs). The dog was diagnosed with congenital ventricular tachyarrhythmia. The condition was improved by lidocaine infusion. After 10 days, the dog was discharged from the hospital with a prescription of atenolol, pimobendan, diltiazem, furosemide, spironolactone, and L-carnitine. This dog is still alive after 31 months. However, progressive cardiac remodeling was confirmed on radiography and ultrasonography. Congenital ventricular tachyarrhythmia is rare in dogs, and the prognosis of reported cases is poor. This report describes the long-term successful management of a dog with congenital ventricular tachyarrhythmia.

Key words **:** Dog, ventricular premature complexes, ventricular tachyarrhythmia.

Introduction

Cardiac arrhythmias are conditions in which the heartbeat is irregular. Some arrhythmias have no clinical consequence, while others lead to serious hemodynamic compromise and sudden death (7).

Among arrhythmias, the inherited form of ventricular arrhythmias (VA) has been described in German shepherd dogs. Affected dogs die as a result of degeneration of ventricular tachycardia (VT) into ventricular fibrillation, which usually occurs during sleep in the early morning hours. The age of dogs afflicted by the presence of VA and sudden death is between approximately 3 and 18 months. Before death, clinical signs are absent, and laboratory data and echocardiographic parameters do not reveal any structural or functional anomalies. Postmortem examination also does not indicate the cause of death (1,6).

The suspected mechanisms of VA in German shepherd dogs are triggered by perturbations in the autonomic nervous system, lacking and heterogeneous innervation, developmental arrest in tissues related to conduction, and abnormal ventricular depolarization because of structural derangement (6). Genetic analyses have been conducted by several groups to confirm the cause of VA in German shepherd dogs. One group demonstrated a strong genetic component for this disease (1) and the other group confirmed that German shepherd dogs with inherited ventricular arrhythmias have electro physiological abnormalities in calcium cycling associated with reduced ATP2A2/SERCA2a expression (4). In veterinary medicine, there have been a few reports of inherited arrhythmias in German shepherds, Boxers, Miniature Schnauzers, English Springer Spaniels, and Golden retrievers; however, genetic analyses to confirm the causes is limited (6). In human medicine, genetic studies have led to important advances in inherited arrhythmic disorders such as long QT syndromes and Brugada syndrome (5).

Here, we report the long-term management of a dog with congenital ventricular tachyarrhythmia.

Case

A four months old, intact female, 11 kg Jindo dog presented to the Veterinary Teaching Hospital of Kangwon National University with collapse.

Physical examination on the day of presentation revealed a heart rate of more than 200 beats per minute, while blood tests showed mild anemia and mild neutrophilia. Thoracic radiography and ultrasonography revealed no remarkable findings; however, ventricular premature complexes (VPCs) were revealed upon electrocardiography (Fig 1). The dog was diagnosed with congenital ventricular tachyarrhythmia. Clinical signs of collapse were improved by lidocaine 2 mg/kg intravenously, but repeated collapses with VPCs appeared; therefore, lidocaine (30 μg/kg/min CRI) was infused to alleviate the symptoms. Three days after hospitalization with wax and wane collapse, dyspnea appeared with VPCs. On thoracic radiography, left atrial dilation was confirmed. Ultrasonography revealed mitral regurgitation and a La:Ao ratio of 2.03:1. To alleviate these symptoms, 0.5 mg/kg atenolol, 1 mg/kg diltiazem, 0.5 mg/kg pimobendan, 2 mg/kg furosemide, and 2 mg/kg spironolactone twice a day were prescribed. Ten days after hospitalization, the clinical signs were improved with improved electrocardiography (Fig 2). The dog was dis-

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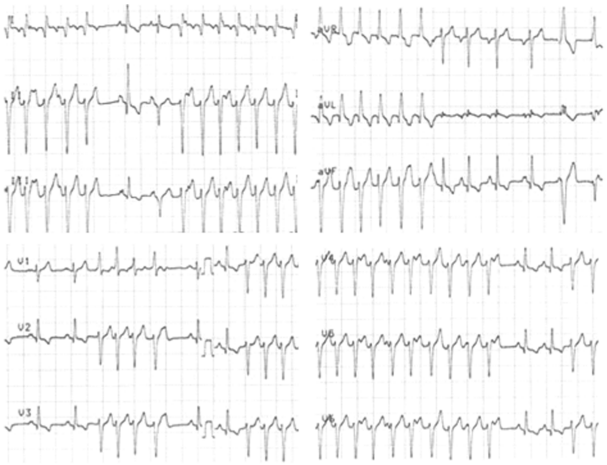

Fig 1. The 12-lead ECG recording taken on the day of presentation. The ECG trace showed ventricular tachyarrhythmia with ventricular premature complexes (VPCs).

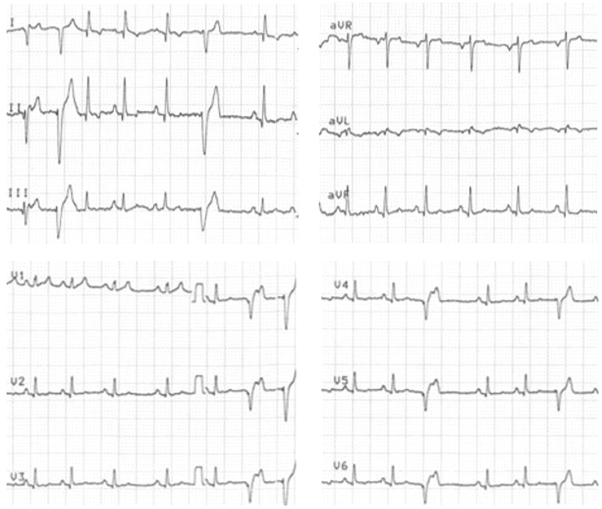

Fig 2. The 12-lead ECG recording taken 10 days after hospitalization. The ECG trace showed some VPCs, but there were no clinical consequences.

charged from the hospital with a prescription of 0.5 mg/kg atenolol, 1 mg/kg diltiazem, 0.5 mg/kg pimobendan, 1 mg/kg furosemide, 0.5 mg/kg spironolactone, and 50 mg/kg L-carnitine twice a day.

This dog was still alive after 31 months. However, progressive cardiac remodeling was confirmed on radiography and ultrasonography (Fig 3).

Discussion

VT primarily occurs in response to myocardial disease or systemic diseases. Asymptomatic animals with occasional VPCs and moderately frequent single VPCs do not require antiarrhythmic drugs, especially if underlying heart function is normal (7).

Antiarrhythmic drugs are prescribed to reduce the risk of death, decrease the frequency of a dangerous arrhythmia and abolish related clinical signs by VT. However, the side effects of antiarrhythmic drugs can lead to morbidity or mortality. Therefore, antiarrhythmic drugs should not be abused. The indications of treatment for VT are more than 17 VPCs per minute, presence of doublets or triplets, presence of multiformed VPCs, presence of runs of VPCs and presence of R on T phenomenon (2).

Antiarrhythmic drugs are composed of class I, class II, class III, and class IV. Class I drugs rapidly decrease inward Na+ current. Class II drugs induce B-adrenergic blockage, which reduces the effects of sympathetic stimulation. Class III drugs selectively prolong action potential duration and refractory period. Class IV drugs slowly decrease inward Ca++ current (7).

Among the antiarrhythmic drugs, Lidocaine, which is class I antiarrhythmic drug, is usually the first choice IV ventricular antiarrhythmic agents in dogs. Intravenous lidocaine converts the arrhythmia to a sinus rhythm. One group demonstrated that lidocaine effectively converts acute vagally associated atrial fibrillation to sinus rhythm in German Shepherd Dogs with inherited arrhythmias (9). In our study, IV lidocaine was not fully effective; therefore, CRI lidocaine was used to control VPCs.

To control VT, other antiarrhythmic drugs in addition to lidocaine have been used. One group showed that combination therapy with mexiletine (class I) and sotalol (Class III) suppresses inherited ventricular arrhythmia in German shepherd dogs better than mexiletine or sotalol monotherapy (3). Another group attempted to use an implantable cardioverter-defibrillator in a German shepherd dog with ventricular arrhythmias (8).

This dog is the only one survivor among its fellows. The

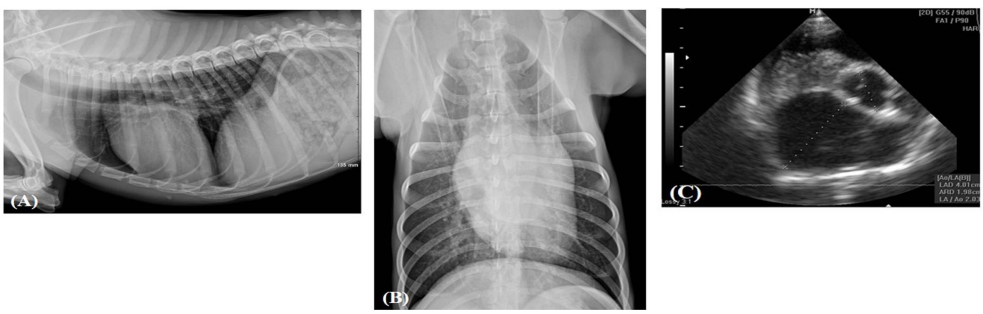

Fig 3. Thoracic radiography and ultrasonography. Thoracic radiography revealed left atrium bulging, but no pulmonary vein dilation (A, B). Ultrasonography showed the La:Ao ratio as 2.03:1 on the right short axis view (C).

other two fellows died of VT at around 3 months of age. Interestingly, this dog has survived for 31 months with medication. However, we confirmed that progressive cardiac remodeling has occurred. We assumed that this cardiac remodeling has been caused by VT for a long time.

Congenital ventricular tachyarrhythmia is rare in dogs, and the prognosis is poor in reported cases. Here, we report the successful management of a dog with congenital ventricular tachyarrhythmia for a long time.

Acknowledgement

This work was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Science, ICT & Future Planning (NRF-2014R1A1A1004339, 2017R1A1A1A05000762).

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