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Case Report

Tachycardia-induced cardiomyopathy in a geriatric Bucovina Shepherd dog

Radu A. Baisan, Vasile Vulpe
Department of Clinics, Faculty of Veterinary Medicine, University of Agricultural Sciences and Veterinary Medicine "Ion Ionescu de la Brad", Romania.

Abstract. A 9 years old Bucovina Shepherd male dog was referred to our teaching hospital with signs of congestive heart failure. Electrocardiography revealed atrial fibrillation and multiple QRS complexes with left bundle branch block aspect consistent with right ventricular complexes. Echocardiography showed left atrial and ventricular dilatation with depressed systolic and diastolic function. The diagnostic was consistent with congestive heart failure due to tachycardia induced cardiomyopathy (TIC) secondary to persistent high penetration atrial fibrillation associated with ventricular complexes.

Key Words: arrhythmia, congestive heart failure, dog.

Corresponding Author: R. A. Baisan, email: baisan.andrei_mv@yahoo.com

Introduction

Atrial fibrillation (AF) is the most common chronic arrhythmia in large breed dogs being frequently associated with organic heart disease (Nattel 2002; Brundel et al 2005).

It is characterized by a fast atrial rhythm (400-600 bpm) and variable ventricular rate, between 130-260 bpm, dependent on the ventricular penetrance (Santilli and Perego 2014). Chronic rapid ventricular rate may induce dilatation and systolic and diastolic dysfunction of the ventricular myocardium, mimicking primary dilated cardiomyopathy (DCM). The aim of the study is to report and describe the physical, electrocardiographic and echocardiographic findings of a dog with tachycardia induced cardiomyopathy (TIC) secondary to persistent high penetrance atrial fibrillation and to perform a differential diagnosis between TIC and DCM.

Case description

A nine years old Bucovina Shepherd male dog, weighing 40 kg was referred to our teaching hospital with a history of distended abdomen for seven days, weight loss and breathlessness. Physical examination revealed pale mucosa, fast respiratory rate, weak and fast arterial pulse and increased respiratory effort. Auscultation revealed fast arrhythmic rhythm. No heart murmur was present.

A five minutes six lead electrocardiography was performed in right lateral recumbence, as previously described (Tilley and Smith Jr 2016). The ECG recording revealed high penetrance atrial fibrillation, with a heart rate of 192 bpm and absence of P-wave (figure 1). The QRS complexes were enlarged (0,072 sec) with positive polarity and amplitude of 0,95 mV. The mean electrical axis was +80°. There were 17 positive QRS complexes present on five minutes recording with normal morphology and normal mean electrical axis in the frontal plane, but with evident higher amplitude (1.30 mV).

Also, multiple QRS complexes showed abnormal morphology, with an aspect of left bundle branch block, with low amplitude (0.84 mV) and increased length (0.136 sec). The polarity was positive in DI, II, III, and aVF and negative in aVL and aVR leads. These complexes were followed by a tall T-wave (0,37 mV), with the same polarity. Some of these bizarre complexes were followed by compensatory period (figure 2). They were present with a frequency of 1:1, 2:1, 3:1 and more than 3:1 normal to abnormal QRS complex. Within the basic rhythm, prolonged RR intervals were present, the longest being of 0.44 sec. The QT interval was 0.21 sec. (QTc = 0.232) and T-wave amplitude 0.2 mV. The ST segment was within normal limits. The mean RR interval was 0.312 sec.

Cardiac ultrasonography was performed on a special table, in left and right lateral recumbence, as previously described (Thomas et al 1993). Left atria and ventricle appeared enlarged, with thinned myocardial walls. No evidence of valvular structural changes was observed, although a moderate regurgitating jet was present through the mitral valve. The left atrial diameter was 68 mm and the left atrial diameter indexed to body weight (LAbw) was 1,9 (normal range 0.59-0.97). The left ventricle internal diameter in diastole (LVIDd) was 50,5 mm and the left ventricle internal diameter in systole (LVIDs) was 43,1 mm. The left ventricle diastolic (LVIDdbw) and systolic (LVIDsbw) diameters scaled to body weight were 1.7 in diastole (normal range 1.27-1.85) and 1,34 in systole (normal range 0.71-1.26) respectively. The left atrium to aorta ratio (La/Ao) was 2.61 (normal value <1.4) (Borgarelli et al 2012). The left ventricular end diastolic volume indexed to body surface area (EDVbsa) was 102 ml/m^2 (normal value < 95 ml/m^2) and the left ventricle end systolic volume indexed to body surface area

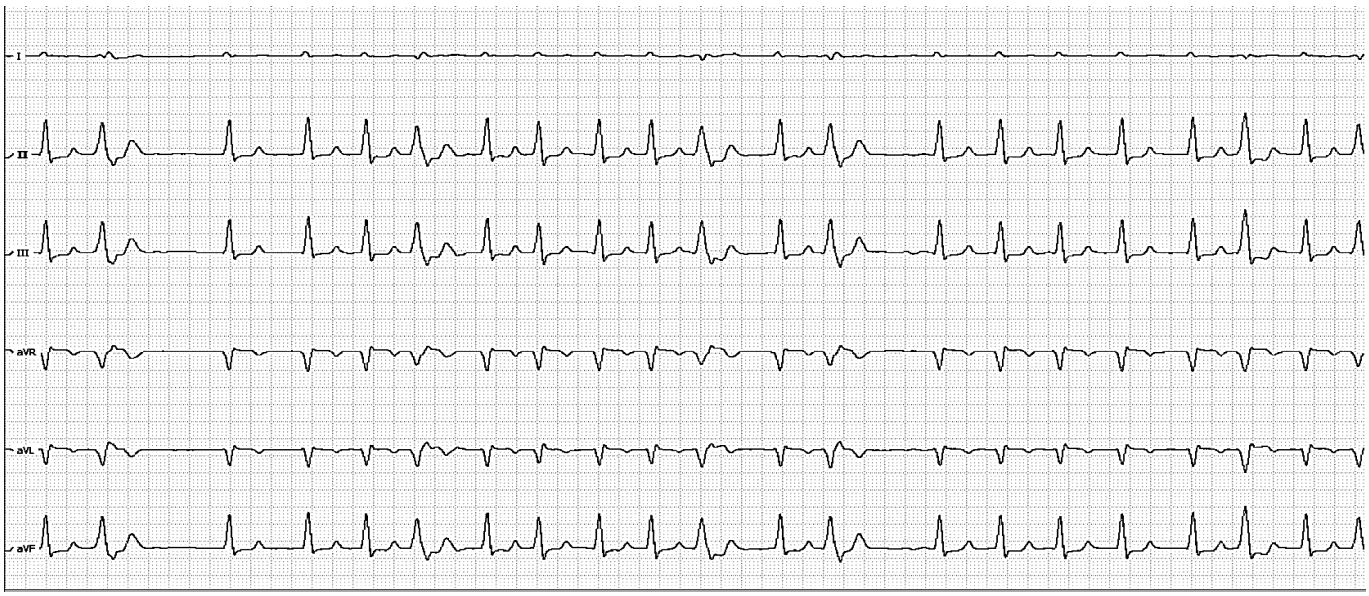

Figure 1. Six lead ECG, 50 mm/s, 10 mm/mV showing atrial fibrillation rhythm, and absence of P-wave; note the 2nd, the 5th, the 11th and the 13th QRS complexes with a bizarre and enlarged aspect, followed by a compensatory period

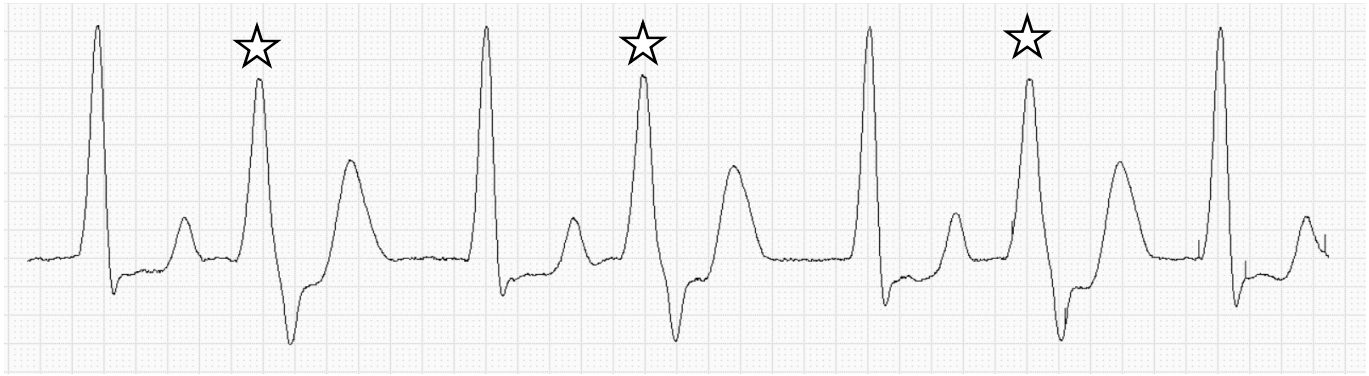

Figure 2. ECG recording in DII, 100 mm/sec, 40 mm/mV –large bizarre QRS complexes (star), with 1:1 ratio - ventricular bigeminy

(ESVbsa) was 70.45 ml/m^2 (normal value < 55 ml/m^2) (Wess et al 2010). The shortening (SF%) and the ejection fraction (EF%) was 14% and 31% respectively, showing a low systolic function of the left ventricle. The sphericity index (SI) was 1,22 (normal value >1,65) and the E point to septal separation (EPSS) was 13,3 mm (Holler and Wess 2014). The right atrium and ventricle appeared within normal limits. The tricuspid valve was also normal.

The dog was released from the hospital with assigned therapy that consisted of atenolol 40 mg p.o. BID and digoxin 0.2 mg p.o. BID.

Discussion

Tachycardia induced cardiomyopathy is a less common cardiac disease and these findings may be confused with DCM which is frequently associated with large breed dogs (O'Grady and O'Sullivan 2004). From authors knowledge, there is no report of DCM in Bucovina Shepherd dogs to date.

The dog was referred to our teaching hospital with severe signs of congestive heart failure. The ECG recording revealed high penetration atrial fibrillation. Studies have proven that sustained tachycardia develop cardiac remodelling, especially of the left ventricle and may benefit from partial or total reversibility once the arrhythmia control is achieved (Gopinathannair et al 2009). The left ventricle remodelling and heart failure is time, heart rate and type of arrhythmia dependent (Santilli and Perego 2014). Structural changes of the myocardial tissue during sustained rapid ventricular rhythm include cardiomyocyte lengthening, hyperplasia, extracellular matrix changes, myocardial fibrosis, myofibril misalignment, loss of sarcomere register and apoptosis (Lishmanov et al 2010).

The abnormal large, left bundle branch block aspect QRS complexes present on the ECG recording are consistent with ventricular premature complexes. Ventricular complexes in dogs, like in humans, may appear due to extracardiac reasons or systemic diseases. Heart diseases that develop right ventricular complexes in dogs may include dilated cardiomyopathy or any form of heart dilatation including the right ventricle, such as tachycardia induced-cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, cardiac tumors or right sided congenital heart diseases (Santilli and Perego 2014). Echocardiography revealed mild enlargement, of the left ventricle and signs of systolic and diastolic dysfunction. Currently there are no echocardiographic reference ranges for this breed and the authors used the allometric scaling cut off for left ventricle and left atria and the left atrial to aorta ratio measurement (Cornell et al 2004).

Clinical studies in human medicine have suggested that the left ventricle internal diameter and volume are significantly smaller in patients with TIC than in those with DCM (Fujino et al 2007; Jeong et al 2008). The dog presented in this paper had left ventricle internal diameter in diastole within the 95% CI reference ranges and only the left ventricle systolic diameter was above the reference ranges (Cornell et al 2004). The left ventricular end diastolic and systolic volumes were mildly increased (7,3% and 27,2%). The reason for a less severe dilatation of the left ventricle in TIC compared to DCM is explained by a compensatory and time dependent enlargement reflecting the gradual disease progression. Also, TIC is presumed to be a relatively acute process, limiting the enlargement of the left ventricle, compared to DCM, were the left ventricular remodelling start in the preclinical stage (Lishmanov et al 2010). A left ventricular remodelling, with a low sphericity index was also observed. This measurement was shown to have a sensitivity of 86,8% and a specificity of 87,6% to differentiate dogs with DCM from apparently healthy dogs (Holler and Wess 2014). However, this measurement has not been compared between dogs with TIC and DCM. E point to septal separation is a measurement used to assess the left ventricular filling and function. A value higher than 6,5 mm was proved to be a good indicator for the alteration of the ventricular early diastolic filling (Holler and Wess 2014). This dog had EPSS above this cut off, suggesting a depressed diastolic function of the left ventricle. The enlargement of the left atrium may be explained by the volume overload inside the left ventricle during the rapid ventricular rate resulting in a high pressure inside the left atrium and chamber dilatation. The dog was released from the hospital that day and antiarrhythmic therapy was assigned, but there was no follow-up available for this case. It remains unknown whether the patients responded well or not to therapy or the elapsed time to death.

The main differences between TIC and DCM are concerning the left ventricular internal diameter and volume. This dog had a normal left ventricular diameter in diastole and mild left ventricular volume overload with altered systolic and diastolic function. Although a Holter monitoring was not available, VPCs on 5 minutes recording were present, suggesting ventricular response to the heart rate and structural changes in the ventricular myocardium. Moreover, DCM has not been described in this breed to date. Considering these findings, this dog was affected by a tachycardia induced cardiomyopathy associated with high penetration atrial fibrillation and ventricular complexes.

One limitation of this study is that it remained unknown whether the arrhythmia control was achieved or if the left ventricle remodelling did reverse. Another limitation is that there was no histopathological characterization of the myocardial tissue to exclude the changes specific for DCM. A third limitation of this study is that there are no cardiologic reference ranges available for this breed and the authors used the scaling methods for indexation to body weight and body surface area.

Conclusions

This paper presents a rare condition of naturally occurring tachycardia induced cardiomyopathy due to persistent atrial fibrillation and ventricular complexes. This pathology must be considered when performing a differential diagnosis, the therapy being different from other cardiomyopathies.

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Authors

•Baisan Radu Andrei, University of Agricultural Sciences and Veterinary Medicine "Ion Ionescu de la Brad", 8 Aleea Mihail Sadoveanu, 700480, Iasi, Romania, EU, email: baisan.andrei_mv@yahoo.com

•Vulpe Vasile, University of Agricultural Sciences and Veterinary Medicine "Ion Ionescu de la Brad", 8 Aleea Mihail Sadoveanu, 700480, Iasi, Romania, EU, email: vasile_vulpe@yahoo.com

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