Electrical cardioversion of sustained ventricular tachycardia in three Boxers

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Case Description—2 spayed female (8 and 9 years old) and 1 sexually intact male (6.5 years old) Boxers were treated because of sustained ventricular tachycardia by electrical cardioversion.

Clinical Findings—Physical examination of the 8-year-old female Boxer revealed tachycardia (heart rate, 250 beats/min), weak femoral pulses, pale mucous membranes, panting, and lethargy. The 6.5-year-old male Boxer had similar physical examination findings, with the addition of a syncopal event. Analysis of the ECG rhythm strips for the 8- and 6.5-year-old dogs indicated a right ventricular origin of the ventricular tachycardia. The 9-year-old female Boxer was being treated with an IV constant rate infusion of lidocaine hydrochloride because of ventricular arrhythmias during the initial examination; physical examination revealed weakness, pale mucous membranes, prolonged capillary refill time, weak femoral pulses, and tachycardia (heart rate, 265 beats/min). Analysis of the ECG rhythm strip for the 9-year-old Boxer indicated a left ventricular origin of the ventricular tachycardia.

Treatment and Outcome—Pharmacological cardioversion treatment was unsuccessful in all 3 Boxers; however, electrical cardioversion by use of a biphasic defibrillator synchronized to conduct 30 J of energy during the peak of the QRS complex was successful in each dog. The electrical cardioversion procedure was performed 2 times (5-day interval between procedures) in the 9-year-old female as a result of relapse of the ventricular tachycardia condition.

Clinical Relevance—Results and follow-up monitoring suggested electrical cardioversion of sustained ventricular tachycardia may be a safe and effective treatment in Boxers that are unresponsive to medical treatment. (J Am Vet Assoc 2010;236:554–557)

An 8-year-old spayed female Boxer was examined because of refractory ventricular arrhythmias that had not resolved after treatment at another referral institution. Initial physical examination revealed tachycardia (heart rate, 250 beats/min), weak femoral pulses, pale mucous membranes, panting, and lethargy. Analysis of an ECG rhythm strip (Figure 1) confirmed a heart rate of 250 beats/min attributable to a sustained ventricular tachycardia, with analysis of the QRS complex pattern that indicated a right ventricular origin of the arrhythmia. Nasal administration of humidified oxygen was initiated, and a catheter was placed in a cephalic vein. A bolus of procainamide (10 mg/kg [4.5 mg/lb], IV) was administered during a 3-minute period without an effect on the arrhythmia. A second dose of procainamide (10 mg/kg, IV) was administered during a 2-minute period without an effect on the arrhythmia. A bolus injection of lidocaine hydrochloride (4 mg/kg [1.82 mg/lb], IV) was administered; the rate of the ventricular tachycardia decreased (heart rate, 220 beats/min) with no other changes detected. A 30 mg/kg (13.6 mg/lb) dose of magnesium sulfate diluted in saline (0.9% NaCl) solution was administered IV during a 20-minute period while echocardiography was performed. Analysis of the echocardiogram revealed no cardiac masses, poor systolic function during ventricular tachycardia, mild regurgitation through the tricuspid valve, and no other valvular insufficiencies. Analysis of abdominal ultrasonographic images revealed no abnormalities. Analysis of a CBC and serum biochemical panel did not reveal abnormalities.

Owner consent to proceed with electrical cardioversion was obtained. The dog was prepared for electrical cardioversion via a biphasic defibrillator. Periprocedural analgesia was provided with an injection of meloxicam (0.2 mg/kg [0.09 mg/lb], IM). A mask secured over the nose and mouth of the dog was used to deliver isoflurane in oxygen for induction of anesthesia; thereafter, the dog was intubated with an endotracheal tube, and anesthesia was maintained with isoflurane in oxygen by use of a rebreathing circuit system and an oxygen flow rate of 1 to 2 L/min. During the first defibrillation attempt, a biphasic defibrillator equipped with ECG monitoring and synchronization capabilities was used to conduct 15 J of energy to the dog; the defibrillator was synchronized to discharge during the peak of the QRS complex. Ventricular tachycardia persisted after the first defibrillation attempt. A second defibrillation attempt was repeated with 30 J of energy. The second attempt successfully restored the sinus rhythm of the heart at a rate of 100 beats/min.

Abbreviation

| ARVC | Arrhythmogenic right ventricular cardiomyopathy |

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The dog recovered from anesthesia without complications and with no obvious signs of external skin damage or discomfort. After a 24-hour hospitalization period during which the dog received oxygen via a nasal catheter, a diagnosis of ARVC was recorded and the dog was discharged. The owners were instructed to administer amiodarone (8.8 mg/kg [4.4 mg/lb], PO, q 12 h, for 7 days, then q 24 h for 7 days), mexiletine\textsuperscript{a} (8.5 mg/kg [3.9 mg/lb], PO, q 8 h), furosemide\textsuperscript{b} (1.7 mg/kg [0.8 mg/lb], PO, q 24 h), benazepril\textsuperscript{b} (0.42 mg/kg [0.2 mg/lb], PO, q 24 h), taurine (22 mg/kg [10 mg/lb], PO, q 12 h), l-carnitine (43 mg/kg [19.5 mg/lb], PO, q 12 h), and a product containing a combination of eicosapentaenoic acid and docosahexaenoic acid\textsuperscript{d} (1,000 and 600 mg, respectively, PO, q 24 h) to the dog. The ARVC progressively worsened during a 14-month treatment period; however, sustained ventricular tachycardia did not recur. The dog was euthanatized 14 months after the electrical cardioversion procedure because of refractory heart failure and anorexia.

A 6.5-year-old sexually intact male Boxer was referred to our facility because of lethargy and cardiac arrhythmias. The dog had a syncopal event while in the hospital waiting room. Initial physical examination revealed a rapid, sustained ventricular tachycardia (heart rate, 240 beats/min) and had weak femoral pulses. Other important physical examination findings included pale mucous membranes and weight loss of 5 kg (2.27 lb) since the previous recording of the body weight 2 weeks prior to this examination. The Boxer was treated with oxygen administered via an oxygen mask, and a continuous ECG was performed. Analysis of the ECG rhythm strip revealed a rapid, sustained ventricular tachycardia (heart rate, 260 beats/min) with a pattern of the QRS complexes that indicated a right ventricular origin. A catheter was placed in a cephalic vein, and an IV bolus injection of lidocaine\textsuperscript{b} (3 mg/kg [1.4 mg/lb], IV) was administered; however, no change was detected in the sustained ventricular tachycardia. Five minutes after the first lidocaine bolus was administered, a second bolus of lidocaine\textsuperscript{b} (4 mg/kg, IV) was administered; however, the sustained ventricular tachycardia persisted. Echocardiography was performed during the arrhythmia, and analysis did not reveal obvious chamber dilatation or evidence of cardiac masses or pericardial disease; however, systolic dysfunction (presumably caused by the rapid heart rate) was observed. Analysis of abdominal ultrasonographic images did not reveal abdominal masses or abdominal fluid. The catheter in the cephalic vein was flushed with saline solution, which was followed by administration of procainamide\textsuperscript{a} (8 mg/kg [3.6 mg/lb], IV) during a 2-minute period; no change was observed in the ventricular rate. Approximately 8 minutes after the first administration of procainamide was completed, a second injection of procainamide\textsuperscript{a} (6 mg/kg [2.7 mg/lb], IV) was administered during a 2-minute period without conversion of the ventricular tachycardia to sinus rhythm.

Owner consent was obtained to proceed with electrical cardioversion. The Boxer was prepared for a cardioversion procedure with periprocedural analgesia and induction of anesthesia similar to those described for the 8-year-old female Boxer. The same biphasic defibrillator, continuous ECG monitoring system, and synchronization settings were used for the cardioversion procedure. The biphasic defibrillator was set to conduct 30 J of energy to the dog and synchronized to discharge during the peak of the QRS complex during the first defibrillation attempt. The cardioversion procedure successfully converted the ventricular tachycardia to a sinus rhythm with a heart rate of 140 beats/min. Following conversion, respiratory rate increased from 20 to 40 breaths/min. Anesthesia was terminated, and the Boxer received oxygen for 1 hour. During this 1-hour time period, the respiratory rate initially increased until the dog was panting, but respiration returned to a typical rate thereafter. The Boxer was monitored for 4 hours and then discharged. The owners were instructed to administer sotalol\textsuperscript{d} (1.3 mg/kg [0.6 mg/lb], PO, q 12 h), mexiletine\textsuperscript{a} (4.8 mg/kg [2.2 mg/lb], PO, q 8 h), and a product that contained a combination of eicosapentaenoic acid and docosahexaenoic acid\textsuperscript{d} (1,000 and 600 mg, respectively, PO, q 24 h) to the dog. During the 6-month period after discharge, the Boxer was provided the prescribed antiarrhythmics. The dog had 2 episodes of anorexia that supported a reduction of the prescribed dose of mexiletine\textsuperscript{a} (4.8 mg/kg, PO, q 12 h). During the next 2-month period, a relapse of the sustained ventricular tachycardia (heart rate, 193 beats/min) has been converted to a sinus rhythm by means of electrical cardioversion consisting of synchronization of the peak of the QRS complex (arrowheads) with conduction of 30 J of energy (arrow) delivered with a biphasic defibrillator. Paper speed = 25 mm/s; 1 cm = 1 mV.

Figure 1—Monitoring lead II ECG rhythm strip recorded from an 8-year-old spayed female Boxer after admission for treatment of refractory ventricular tachycardia. Left bundle branch block pattern of the QRS complexes indicates a right ventricular origin of the arrhythmia. Mean rate of the ventricular tachycardia is 260 beats/min. Paper speed = 25 mm/s; 1 cm = 1 mV.

Figure 2—Monitoring lead II ECG rhythm strip recorded from the same 8-year-old spayed female Boxer in Figure 1. The ventricular tachycardia (heart rate, 193 beats/min) has been converted to a sinus rhythm by means of electrical cardioversion consisting of synchronization of the peak of the QRS complex (arrowheads) with conduction of 30 J of energy (arrow) delivered with a biphasic defibrillator. Paper speed = 25 mm/s; 1 cm = 1 mV.
tachycardia was not detected during 2 episodes of ECG monitoring for 24 hours and 3 cardiologic examinations. Furthermore, no adverse events were reported by the owner.

A 9-year-old spayed female Boxer was referred for treatment of ventricular arrhythmias. At the time of admission, the Boxer was being treated with lidocaine (40 µg/kg/min [18.2 µg/lb/min], IV) as a constant rate infusion, furosemide (2.8 mg/kg [1.27 mg/lb], PO, q 12 h), enalapril maleate (0.4 mg/kg [0.18 mg/lb], PO, q 12 h), and an unspecified multivitamin product. Physical examination revealed weakness, pale mucous membranes, prolonged capillary refill time (3 seconds), weak femoral pulses, mild periodontal disease, and tachycardia (heart rate, 260 beats/min). Analysis of the ECG rhythm strip revealed sustained ventricular tachycardia (heart rate, 265 beats/min) with a pattern of the QRS complexes that indicated a left ventricular origin (right bundle-branch block). A bolus injection of lidocaine (2 mg/kg [0.9 mg/lb], IV) converted the ventricular tachycardia to a sinus rhythm for 20 seconds, which was followed by a relapse of the ventricular tachycardia (heart rate, 260 beats/min). Analysis of echocardiographic images revealed mild regurgitation through the mitral valve, mild aortic valve insufficiency, poor systolic function, moderate left atrial enlargement, moderate left ventricular volume overload, and mild right atrial enlargement. No obvious pericardial disease or cardiac masses were observed during echo-cardiography. Analysis of abdominal ultrasonographic images revealed no masses or free-fluid accumulation. Analysis of a CBC and serum biochemical panel revealed no abnormalities, except for an elevated BUN (34 mg/dL; reference range, 10 to 27 mg/dL). Because of a lack of response to treatment with procainamide and lidocaine, owner consent to proceed with electrical cardioversion was obtained. Periprocedural analgesia was provided similarly to that for the 2 previously described Boxers; however, the anesthesia procedure differed in that sevoflurane in oxygen was used in place of isoflurane. The constant rate infusion of lidocaine (40 µg/kg/min, IV) was continued during the electrical cardioversion procedure. The same biphasic defibrillator and continuous ECG monitoring systems and synchronization settings as described for the 2 other Boxers were used for the cardioversion procedure. Defibrillation with 30 J of energy resulted in a sinus rhythm with a heart rate of 100 beats/min. The dog recovered from anesthesia. Relapse of the ventricular tachycardia (heart rate, 270 beats/min) was detected 20 minutes after the electrical cardioversion procedure. Despite our medical recommendations, a second electrical cardioversion procedure and hospitalization were declined by the owner, and the Boxer was discharged. The owner was instructed to administer amiodarone (200 mg, PO, q 12 h) and mexiletine (150 mg, PO, q 12 h). The owner was instructed to continue the furosemide and enalapril administrations as previously described. Recommendations were made to keep the Boxer calm and to strongly consider another attempt via pharmacological conversion or electrical cardioversion.

The Boxer was admitted for treatment of ventricular tachycardia (heart rate, 280 beats/min) and tachypnea at 48 breaths/min (reference range, 16 to 24 breaths/min) 5 days after discharge. Analysis of thoracic radiographs obtained at that time revealed mild pulmonary edema in the perihilar region and right caudal lung lobe consistent with acute left-sided congestive heart failure. The Boxer was treated by administration of furosemide (5 mg/kg [2.3 mg/lb], IV) and humidified oxygen. The tachypnea resolved during a 90-minute period after treatment initiation. Evaluation of abdominal ultrasonographic images revealed no abnormal findings. The Boxer was medicated with diphenhydramine hydrochloride (1 mg/kg [0.45 mg/lb], IM) prior to attempted pharmacological cardioversion with amiodarone (8 mg/kg [diluted with 5% dextrose in water], IV) administered during a 20-minute period. During amiodarone infusion, the Boxer became agitated, the skin appeared flushed, and the ventricular tachycardia rate progressively decreased to a heart rate of 215 beats/min, which led to a break in ventricular tachycardia for 3 sinus beats only. Given that sustained ventricular tachycardia remained, owner consent was obtained for another electrical cardioversion procedure by use of the same biphasic defibrillator. Periprocedural analgesia was provided similarly to that described previously. Anesthesia was induced with an injection of propofol (4 mg/kg, IV). The Boxer was intubated, and anesthesia was maintained with isoflurane in oxygen administered via a rebreathing circuit system and an oxygen flow rate of 1 to 2 L/min. The conduction of 30 J of energy from the defibrillator was synchronized with the peak of the QRS complex. A successful conversion to sinus rhythm was detected (heart rate, 120 beats/min). The Boxer recovered from anesthesia, with no obvious external damage from the defibrillator discharge; however, the auricular pinna of the dog remained hyperemic, which was attributed to IV administration of amiodarone. Oxygen was administered nasally for 6 hours; during that period, the heart remained in sinus rhythm with no ventricular arrhythmias. The Boxer was discharged, and the owner was instructed to administer medications in accordance with the previous discharge instructions for the dog. As a typical adverse effect of amiodarone use, the Boxer’s liver enzyme activities increased during the next 3 months; therefore, amiodarone was replaced with sotalol (2.2 mg/kg [1 mg/lb], PO, q 12 h). At the 3-month reexamination, pimobendan (0.27 mg/kg [0.12 mg/lb], PO, q 12 h) was added to the treatment regimen, enalapril was replaced with benazepril (0.36 mg/kg [0.16 mg/lb], PO, q 24 h), and the furosemide dose was increased (2.2 mg/kg, PO, q 12 h). Ventricular arrhythmias were detected during periodic reexaminations. The Boxer died as a result of suspected ARVC disease 26 weeks after the second electrical cardioversion procedure.

Discussion

Boxers are predisposed to ventricular arrhythmias and arrhythmia-induced sudden death, but the underlying condition responsible for these clinical features has been incompletely defined. This condition in Boxers shares many common features with ARVC in humans; thus, Boxers with this condition may prove
Useful for the study of this condition in humans. Drug therapy is the primary treatment modality for ventricular tachycardia in humans, particularly with the use of class III, potassium channel-blocking antiarrhythmics, such as amiodarone and sotalol.8 Efﬁcacious anti-arrhythmic medical treatments have been described for Boxers with sotalol9 alone or a combination of sotalol and mexiletine1 or atenolol and mexiletine.9,10 Still, the high incidence of ventricular arrhythmias, syncope, and sudden death as a result of cardiovascular disease in Boxers appears to be caused by an inherited autosomal dominant trait.11 Arrhythmias associated with ARVC in humans are often refractory to medical treatment, and electrical cardioversion is a necessary treatment option.1 The 3 Boxers reported here support the information known about this condition (ie, Boxers with ARVC can be refractory to medical management of ventricular tachyarrhythmias).12

Use of an implantable cardioverter deﬁbrillator to control clinical signs as a result of ventricular arrhythmias in a Boxer has been described,7 emulating what is commonly done in humans.8,9 However, the routine use of an implantable deﬁbrillator in veterinary medicine is currently limited by cost and technical expertise. The danger of ventricular tachycardia is that this condition may lead to ventricular ﬁbrillation and death, which makes immediate intervention a necessity. Given the lack of other viable options, electrical cardioversion was attempted in the 3 Boxers described here. The overall success for use of a biphasic deﬁbrillator and survival of all patients through the procedure was surprising, and use of a deﬁbrillator appears to provide a viable option in these grave circumstances. The obvious danger of anesthetizing a hemodynamically compromised patient with ventricular tachycardia was considered; however, performing this procedure without the aid of analgesia from anesthesia was considered dangerous and, in the author’s judgment, inhumane. An attempt was made to limit the amount of anesthetic drugs used in preparation for the deﬁbrillation procedure and to be prompt when performing the procedure. Because of its biphasic property, the deﬁbrillator worked well at relatively low energy levels (typically 30 J), and the synchronization function made the procedure safer and less stressful. I strongly encourage the use of a biphasic deﬁbrillator equipped with synchronization functions if an attempt is to be made to perform the electrical cardioversion procedure as described.

Sustained ventricular tachycardia is a life-threatening condition in Boxers and is stressful for the owners to endure, and management of this condition is difﬁcult for attending veterinarians. The 3 dogs described in this report shared similar life-threatening arrhythmias but, fortunately, responded to treatment with electrical cardioversion by use of a biphasic deﬁbrillator. The overall deﬁbrillation success and survival of all 3 Boxers treated was encouraging, and use of a deﬁbrillator appears to provide a viable option in similar grave circumstances.

References