ECG of the Month

A 9-year-old 35-kg (77-lb) castrated male Doberman Pinscher was referred to the Cornell University Hospital for Animals Emergency Service because of syncopal episodes preceded by a 1-day history of nonproductive coughing and a single episode of vomiting. On initial physical examination, the dog was in good body condition (body condition score, 5/9) with apparently normal mentation. Rectal temperature was 36.4°C (97.5°F), mucous membranes were pale pink, and capillary refill time was 2 seconds. Heart rate during auscultation was 186 beats/min, and an irregularly irregular rhythm was present. A grade 3/6 systolic murmur was auscultated, with point of maximal intensity at the left sixth intercostal space. Lung sounds were diffusely harsh, and respiratory crackles were detected in the left cranioventral lung field. Femoral pulse pressure was weak, and pulse deficits were detected. Arterial blood pressure measured with a Doppler method was initially 80 to 120 mm Hg.

Thoracic radiography revealed moderate cardiomegaly with loss of the caudal waist, dilated pulmonary veins in the cranial lung lobes, and a moderate diffuse interstitial pattern that was accentuated in the right cranioventral lung field. These findings were consistent with cardiogenic pulmonary edema. Results of a CBC and serum biochemical profile were unremarkable, with the exception of mildly high serum aspartate aminotransferase activity (81 U/L; reference interval, 14 to 51 U/L).

Echocardiography revealed reduced left ventricular (LV) ejection fraction (26%) and shortening fraction (11.5%), LV dilation (LV end-diastolic dimension, 53.7 mm; LV end-systolic dimension, 47.5 mm), moderate mitral valve regurgitation, and severe left atrial enlargement (left atrium-to-aorta ratio, 2.3:1). On the basis of breed, clinical signs, and echocardiographic findings, a presumptive diagnosis of idiopathic dilated cardiomyopathy was made. Electrocardiography was performed.

ECG Interpretation

An initial ECG examination revealed atrial fibrillation (AF) with a rapid ventricular response rate (186 beats/min). A higher heart rate (260 beats/min) was recorded shortly after hospital admission. Doppler-assessed blood pressure was decreased to 70 mm Hg during higher heart rates. On further ECG monitoring, beats of supraventricular origin were present with an intraventricular conduction delay, as noted by the prolonged QRS complex duration (90 milliseconds; reference limit, < 70 milliseconds) and decreased slope of the RS segment **(Figure 1)**. Also referred to as slurring of the QRS complex, a decreased slope of the RS segment is suggestive of depolariza-

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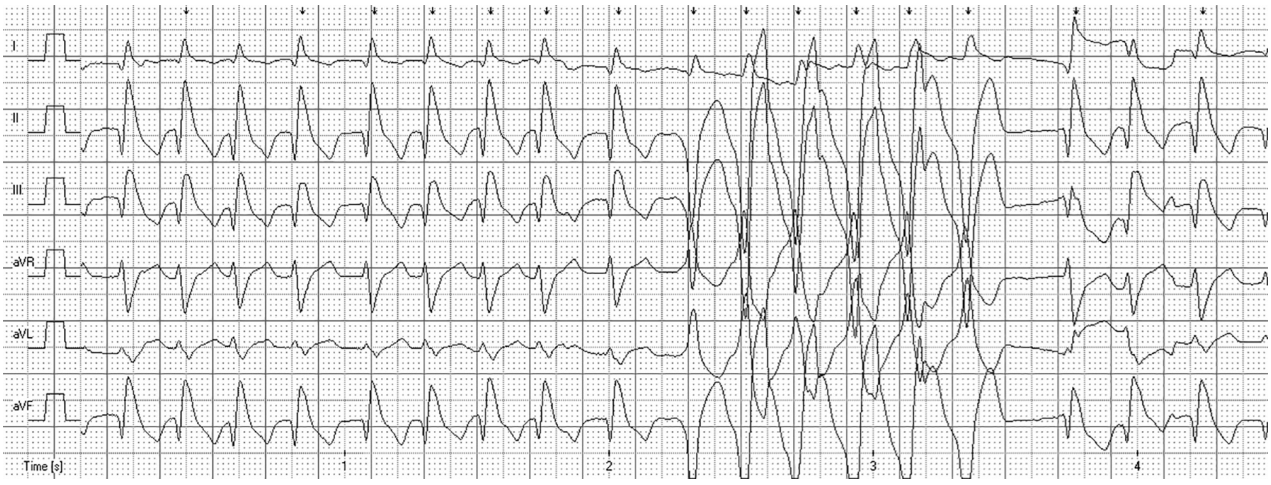

Figure 1—Initial 6-lead ECG tracings recorded from a 9-year-old Doberman Pinscher that was evaluated because of syncopal episodes preceded by a 1-day history of nonproductive coughing and a single episode of vomiting. Atrial fibrillation with a rapid ventricular response rate and runs of nonsustained pleiomorphic ventricular tachycardia (VT) are present. The heart rate is approximately 240 beats/min during atrial fibrillation and approximately 300 beats/min during VT. Notice the intraventricular conduction delay during supraventricular beats (QRS complex duration, 100 milliseconds), indicative of diseased myocardium. In addition, the deep Q waves in leads I, II, III, and aVF are suggestive of biventricular enlargement. Arrows along the top of the ECG denote sensed QRS complexes. Paper speed = 50 mm/s; 5 mm = 1 mV.

tion abnormalities and myocardial ischemia.[1,2] Deep Q waves in leads I, II, III, and aVF are typically associated with biventricular enlargement.[3,4] Nonsustained runs of multifocal ventricular tachycardia (VT) were also present.

The dog was administered furosemide (2 mg/kg [0.91 mg/lb], IV, q 6 h), pimobendan (0.5 mg/kg [0.23 mg/lb], PO, q 12 h), extended-release diltiazem hydrochloride (2.5 mg/kg [1.14 mg/lb], PO, q 12 h), and digoxin (0.003 mg/kg [0.0014 mg/lb], PO, q 12 h). Hair on the thorax was clipped bilaterally over the palpable apex beat, and transcutaneous pacing patches were placed to permit continuous ECG monitoring and defibrillation if necessary. A few minutes later, rapid, sustained VT developed, and the dog developed weakness and syncope **(Figure 2)**. Loss of consciousness associated with ventricular fibrillation (VF) then ensued. The dog was immediately defibrillated with one 200-J biphasic transthoracic direct current shock, which resulted in restoration of sinus rhythm with intermittent, single ventricular premature complexes, and a heart rate of 120 beats/min. The dog was administered mexiletine hydrochloride (4.3 mg/kg [1.95 mg/lb], PO, q 8 h) and, for postdefibrillation pain control, methadone hydrochloride (0.2 mg/kg [0.09 mg/lb], IV, q 4 h [2 doses given]). Administration of diltiazem and digoxin were discontinued. Continuous ECG monitoring was continued, which revealed that sinus rhythm was sustained throughout the duration of hospitalization.

The dog's respiratory rate and effort normalized within the first 6 hours after hospitalization, and 24 hours later, the dog was discharged from the hospital. The owner was instructed to administer the following medications: furosemide (2 mg/kg, PO, q 12 h), enalapril maleate (0.3 mg/kg [0.14 mg/lb], PO, q 12 h), pimobendan (0.29 mg/kg [0.13 mg/lb], PO, q 12 h), mexiletine (4.2 mg/kg [1.9 mg/lb], PO, q 8 h), and amiodarone hydrochloride (11.4 mg/kg [5.18 mg/lb], PO, q 24 h for 7 days, then 5.8 mg/kg [2.6 mg/lb], PO, q 24 h).

Two weeks later, the dog had sinus rhythm with intermittent single, monomorphic ventricular premature complexes. Appetite, activity, and respiratory rate were normal. Results of a repeated CBC and serum biochemical profile were within reference intervals. At 3 months after defibrillation, the dog was without clinical signs and an ECG examination revealed sinus rhythm with an intraventricular conduction delay **(Figure 3)**. Serum biochemical analysis revealed high activities of alanine aminotransferase (358 U/L; reference interval, 20 to 98 U/L) and aspartate aminotransferase (133 U/L). Given the serum biochemical findings, the dosage of amiodarone was decreased to 2.85 mg/kg (1.29 mg/lb), PO, every 24 hours.

Four months after defibrillation, a Holter monitor was used to evaluate the dog over a 24-hour period. The monitoring revealed an underlying sinus rhythm with a mean heart rate of 86 beats/min. Ventricular ectopy (approx 3%) was present, which included singlets, couplets, triplets, and 4 nonsustained runs

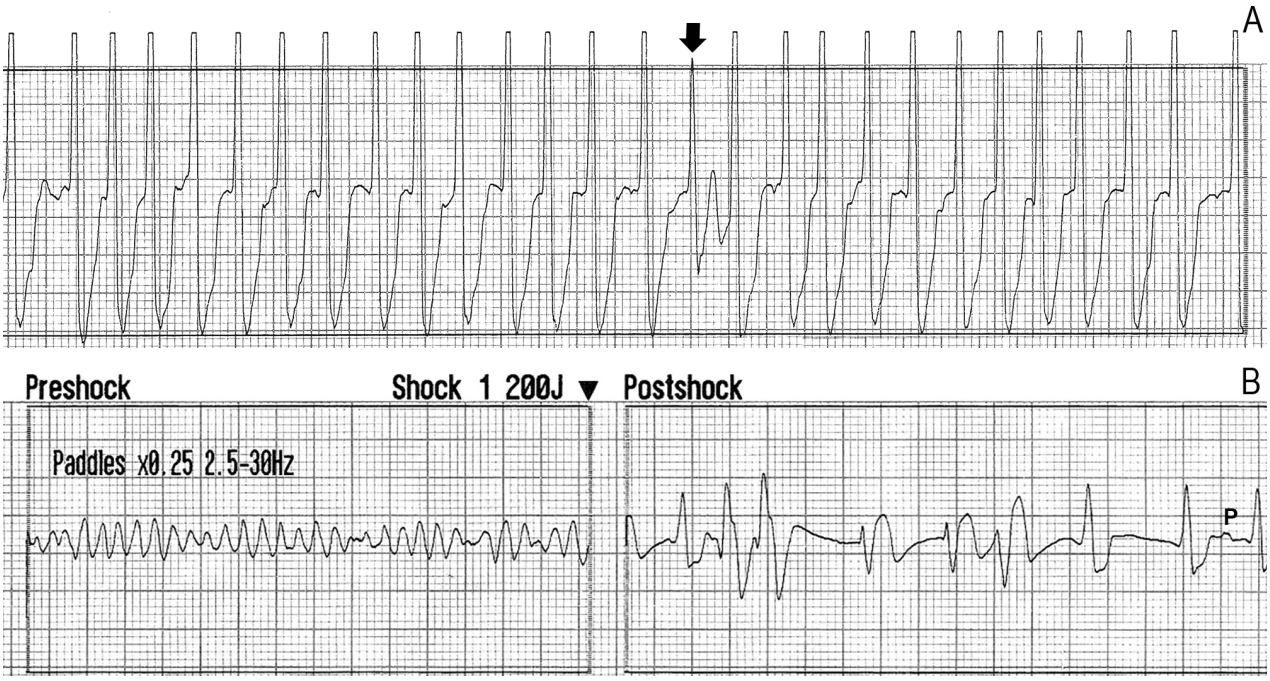

Figure 2—Electrocardiographic traces automatically recorded from a defibrillator with transcutaneous pacing patches placed on the thorax of the dog in Figure 1. A—Continuous ECG monitoring revealed rapid VT (approx 270 beats/min). The sixteenth QRS complex (black arrow) is narrower than the others and may represent pleiomorphism or a single supraventricular beat. The dog had a syncopal event during this rhythm. B—The loss of consciousness associated with the syncopal event resulted in ventricular fibrillation. The dog was immediately defibrillated with one 200-J biphasic transthoracic direct current shock (inverted arrowhead). After the shock, multifocal VT is present before recovery of sinus rhythm (P wave is labeled). In both panels, paper speed = 25 mm/s; 10 mm = 1 mV.

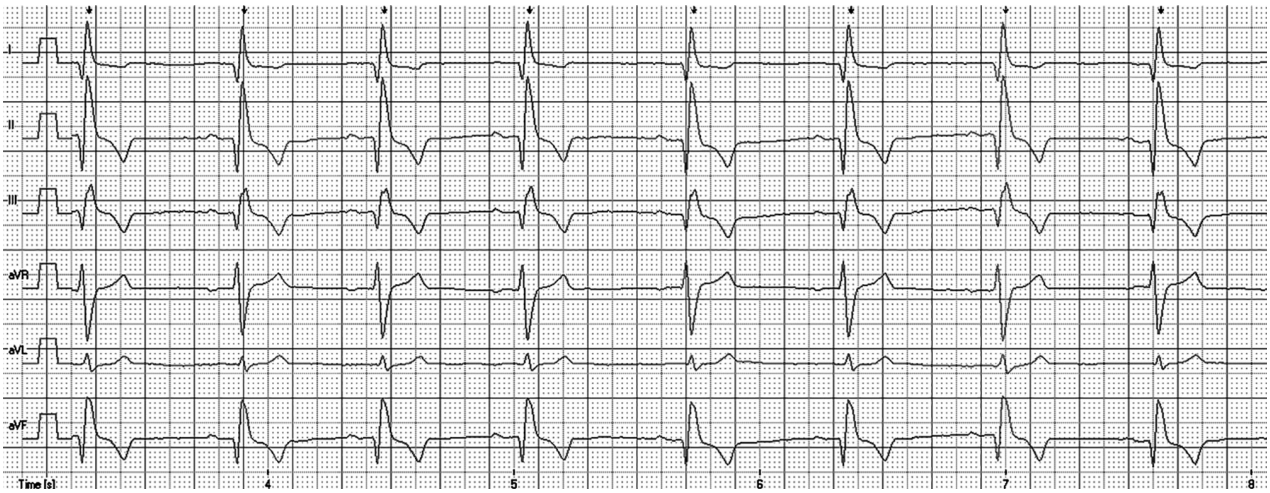

Figure 3—Six-lead ECG tracings recorded for the dog in Figure 1 during a recheck examination at 3 months after defibrillation. Notice the sustained sinus rhythm. The heart rate is approximately 120 beats/min. The PR interval (100 milliseconds) is normal (reference limits, 60 to 130 milliseconds). Intraventricular conduction delay remains present (QRS complex duration, 90 milliseconds), as do the deep Q waves. Intermittent, single ventricular premature complexes were also evident (not shown). Arrows along the top of the ECG denote sensed QRS complexes. Paper speed = 50 mm/s; 5 mm = 1 mV.

of accelerated idioventricular rhythm (< 140 beats/min). In addition, supraventricular ectopy (approx 3%) predominantly in the form of nonsustained runs of supraventricular tachycardia (maximum heart rate, 192 beats/min) was evident. Five months after defibrillation, administration of amiodarone was discontinued owing to development of inappetence, vomiting, further increases in serum activities of alanine aminotransferase (973 U/L) and aspartate aminotransferase (252 U/L), and high serum activities of alkaline phosphatase (749 U/L; reference interval, 17 to 111 U/L) and γ-glutamyltransferase (31 U/L; reference interval, 0 to 6 U/L). The dog had also developed hyperbilirubinemia (total bilirubin concentration, 0.7 mg/dL [reference interval, 0 to 0.2 mg/dL]; direct bilirubin concentration, 0.5 mg/dL [reference interval, 0 to 0.1 mg/dL]). Sotalol hydrochloride (80 mg/kg [36.4 mg/lb], PO, q 12 h) was added to the dog's treatment regimen, and all other medications were continued as previously prescribed. Clinical signs and serum biochemical abnormalities normalized within 1 week. A recheck Holter examination was scheduled to assess treatment efficacy; however, the dog died suddenly 8 months after defibrillation.

Discussion

The case described in the present report illustrated the rapidly progressive and near fatal consequences of the comorbidities of acutely decompensated dilated cardiomyopathy, congestive heart failure, and a hemodynamically and electrically unstable dysrhythmia in a Doberman Pinscher. The rapid deterioration of the dog's condition following emergency examination highlighted the need for close monitoring and vigilance in the clinical management of such patients. This case also demonstrated the positive short- and midterm outcomes following simultaneous electrical cardioversion of hemodynamically unstable AF and VT, which progressed to VF in combination with antiarrhythmic treatment, to address recurrence of VT and AF in the postdefribrillation period.

For the dog of the present report, treatment decisions at the time of hospital admission were influenced by concerns that IV administration of antiarrhythmic medication might exacerbate poor systolic function and severe arterial hypotension. Instead, the dog was treated orally with diltiazem and digoxin, with the goal of slowing the heart rate and improving myocardial oxygenation. The ultimate trigger for VF in this dog was unknown. Every antiarrhythmic drug has proarrhythmic properties,[5] and digoxin administration in humans with congestive heart failure and concurrent AF is controversial.[6,7] In the dog of the present report, a low dose of digoxin (0.003 mg/kg) was administered orally within 30 minutes before detection of VF. Serum digoxin concentration peaks at approximately 2 hours after administration of digoxin tablets in dogs[8]; hence, digoxin-induced VF seemed unlikely in this case. We speculated that the dilated cardiomyopathy phenotype of the myocardium provided a substrate for the ventricular arrhythmias, upon which myocardial ischemia secondary to systolic dysfunction and rapid AF provoked the development of VT and VF.

In states of hemodynamic instability, structural heart disease, and left atrial enlargement, electrical cardioversion is the treatment of choice to convert tachyarrhythmias in humans.[9–11] This procedure is performed rarely in veterinary medicine, in which pharmacological treatments are generally selected as an alternative. Rarely, IV administration of antiarrhythmics may suppress cardiac function and cause peripheral vasodilation, further worsening systemic hypotension. Lidocaine administration in humans increases the mortality rate among some patients with hemodynamically unstable VT and concurrent ischemic heart

disease.[12] It is unclear whether these data relate directly to dogs, in which myocardial infarction is rare. Data from some human clinical trials suggest that IV administration of amiodarone may provide a superior survival benefit, compared with lidocaine, in patients with ventricular tachyarrhythmias and congestive heart failure.[10,13,14] Equivalent studies in dogs are lacking, to our knowledge, but it should be noted that apparent sensitivity to amiodarone in Doberman Pinschers has been reported.[15]

Electrical defibrillation successfully treated the dog of the present report, in which cardiac status had rapidly degenerated from AF with hypotension to VT, VF, and hemodynamic collapse. Critical monitoring and immediate defibrillation were facilitated by the preemptive placement of external defibrillation patches in response to decline of the dog's clinical condition. Based on the 3-phase model of CPR,[16] defibrillatory shock should be administered within the first 4 minutes of VF (the electrical phase), before major ischemic injury has occurred.[16,17] At the Cornell University Hospital for Animals, continuous ECG monitoring via external defibrillation patches is now standard protocol in this subset of patients considered at risk for VF and comprises only approximately 5% of the total hospitalization cost for each patient.

Following recovery and hospital discharge of the dog of the present report, long-term treatment with amiodarone was justified on the basis of the persistent risk of sudden death associated with dilated cardiomyopathy in Doberman Pinschers and to prevent or delay recurrence of AF. In humans, amiodarone is considered to be an effective drug for controlling life-threatening ventricular arrhythmias,[18] to maintain sinus rhythm after cardioversion,[19,20] and to prevent atrial remodeling.[21] In dogs with structural heart disease and AF, the AF generally recurs within days to months after electrical cardioversion, especially in the presence of left atrial enlargement.[22] Whether long-term amiodarone administration may help to maintain sinus rhythm after electrical cardioversion in dogs is unknown. On the basis of its history, this dog's sudden death was suspected to be attributable to recurrence of VT and VF, although the dog's cardiac rhythm in the days preceding death was unknown. Intraventricular conduction delay, as observed in this dog, may be a negative prognostic indicator in Doberman Pinschers.[2] In addition, given that the mean and median survival times of Doberman Pinschers with congestive heart failure and AF are < 3 months,[23] it was hypothesized that conversion to sinus rhythm prolonged this dog's life.

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