ECG of the Month

A 7-year-old 5.78-kg (12.7-lb) spayed female domestic shorthair cat was evaluated by the Emergency Service at a veterinary teaching hospital because of sudden onset vomiting of 1 day's duration. Two weeks previously, the cat had developed a subcutaneous swelling between the shoulder blades at the site of vaccine administration. Meloxicam (0.1 mg/kg [0.045 mg/lb], PO, q 24 h) was prescribed at that time, and the owner had been administering the drug for 1.5 weeks. The owner reported that the cat had no previous health problems.

On initial physical examination, the cat was bright, alert, and responsive. Rectal temperature was 36.2°C (97.1°F), heart rate was 132 beats/min, and respiration rate was 44 breaths/min. Heart rhythm was regular, and a grade 3/6 left-sided sternal systolic ejection murmur was ausculted. There was a 2-cm-diameter subcutaneous mass between the scapulae.

A CBC and serum biochemical analyses were performed. Abnormalities detected included leukopenia (2.8 × 10^3 WBCs/µL; reference range, 4.0 × 10^3 WBCs/µL to 14.0 × 10^3 WBCs/µL) characterized by neutropenia (1.039 × 10^3 cells/µL; reference range, 2.0 × 10^3 cells/µL to 12.0 × 10^3 cells/µL) and lymphopenia (1.232 × 10^3 cells/µL; reference range, 1.6 × 10^3 cells/µL to 6.0 × 10^3 cells/µL); high alanine aminotransferase activity (165 U/L; reference range, 34 to 106 U/L); high concentrations of BUN (133 mg/dL; reference range, 14 to 35 mg/dL), creatinine (11.0 mg/dL; reference range, 0.9 to 2.0 mg/dL), phosphorus (6.4 mg/dL; reference range, 3.3 to 6.1 mg/dL), and potassium (9.1 mEq/L; reference range, 3.6 to 5.6 mEq/L); low concentrations of total calcium (8.1 mg/dL; reference range, 9 to 12 mg/dL) and chloride (113 mEq/L; reference range, 114 to 125 mEq/L); and high anion gap (27.2 mEq/L; reference range, 12 to 26 mEq/L). All other variables were within reference ranges, including sodium concentration (147 mEq/L; reference range, 143 to 155 mEq/L). Electrocardiography was performed at this time (**Figure 1**).

ECG Interpretation

The ECG examination performed at the initial evaluation revealed tall tented T waves with absent P waves (Figure 1). The QRS complex duration was 0.04 seconds, and the mean electrical axis was +60°. A single ventricular premature complex was detected. The heart rate was approximately 140 beats/min; the heart rhythm appeared to be sinoventricular, consistent with hyperkalemia.

The cat was hospitalized, and lactated Ringer's solution was administered at a rate of 19 mL/h overnight (10 hours) to treat renal failure. Furosemide (10 mg) was administered once IV to promote urine production and potassium wasting. The following morning (day 1), the heart rhythm was irregular and heart rate varied from 180 to 300 beats/min. A grade 3/6 left-sided sternal systolic ejection murmur and intermittent gallop were detected. Echocardiography was performed in an attempt to determine the source of the murmur and evaluate cardiac chamber sizes after diuresis. Echocardiography revealed mild dilation of all 4 cardiac chambers consistent with volume overload and mild pleural and pericardial effusion; a decrease in fluid administration rate was advised. A second ECG examination was performed (**Figure 2**); the serum potassium concentration at the time of this procedure was 7.9 mEq/L.

With the cat in right lateral recumbency, a 6-lead ECG examination revealed a cyclic, recurring arrhythmia. Each run began with at least 2 narrow, upright QRS complexes in lead II (duration, 0.03 seconds each) without preceding p waves and followed by tall T waves similar to those observed on the previous day's ECG. These narrow QRS complexes occurred at an instantaneous rate of approximately 176 beats/min at the beginning of each run. The third beat occurred earlier than expected and was fol-

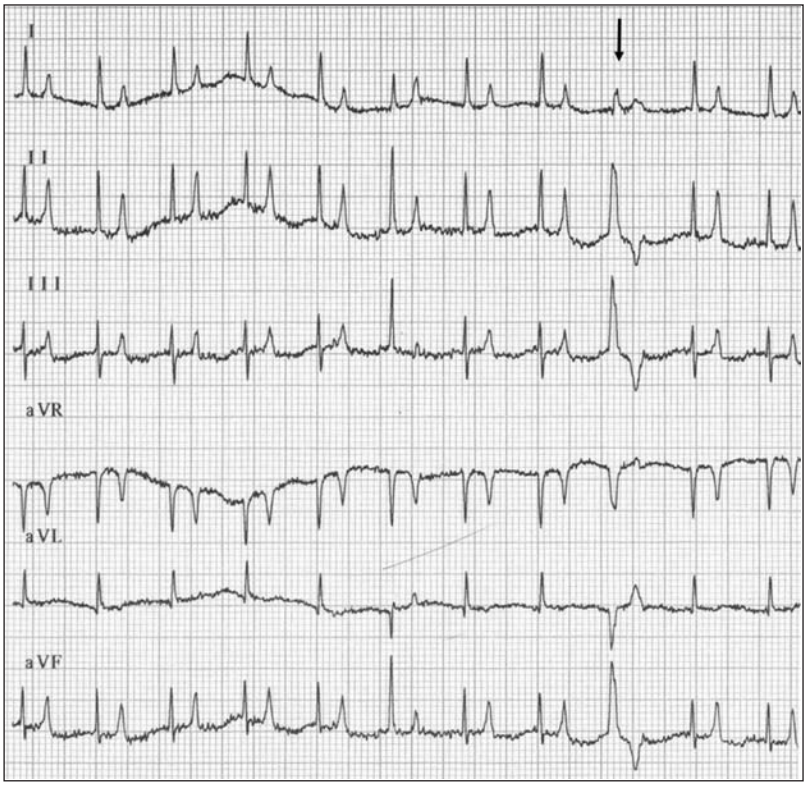

Figure 1 —Initial 6-lead ECG recording obtained from a cat that was evaluated because of vomiting of 1 day's duration. Two weeks previously, the cat had developed a subcutaneous swelling between the shoulder blades at the site of vaccine administration and had been treated with meloxicam. Serum biochemical analyses revealed evidence of acute renal failure and hyperkalemia; serum potassium concentration at the time of ECG examination was 9.1 mEq/L (reference range, 3.6 to 5.6 mEq/L). The ECG diagnosis was sinoventricular rhythm; a ventricular premature beat was detected (arrow). Paper speed = 25 mm/s; 2 cm = 1 mV.

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lowed by at least 2 others with the same cycle length. The instantaneous heart rate was approximately 315 beats/min at the end of each run. At the higher heart rate, the latter 3 beats of the cyclic rhythm revealed an S-wave morphology that appeared to gradually widen the QRS complexes (to a duration of 0.05 seconds each). Following the short run of tachycardia, there was an abrupt pause before the rhythm began again with a narrow upright QRS complex at a rate of 176 beats/min. This repeating pattern of complexes cycled continuously. The ECG diagnosis was sinoventricular rhythm with paroxysms of supraventricular tachycardia that had a right bundle branch block wave morphology.

Diuresis was continued via IV administration of saline (0.9% NaCl) solution at a rate of 7 mL/h for another 18 hours. The next day (day 2), the cat's serum potassium concentration was 4.8 mEq/L and a follow-up ECG examination was performed (**Figure 3**). With the cat in right lateral recumbency, a 6-lead ECG examination revealed that the QRS complexes were narrow and upright in lead II; there was a P wave for every QRS complex and a QRS complex for every P wave. The rhythm was a normal sinus rhythm with a rate of 220 beats/min. The cat continued to respond well to medical management for renal failure and was discharged from the hospital several days later.

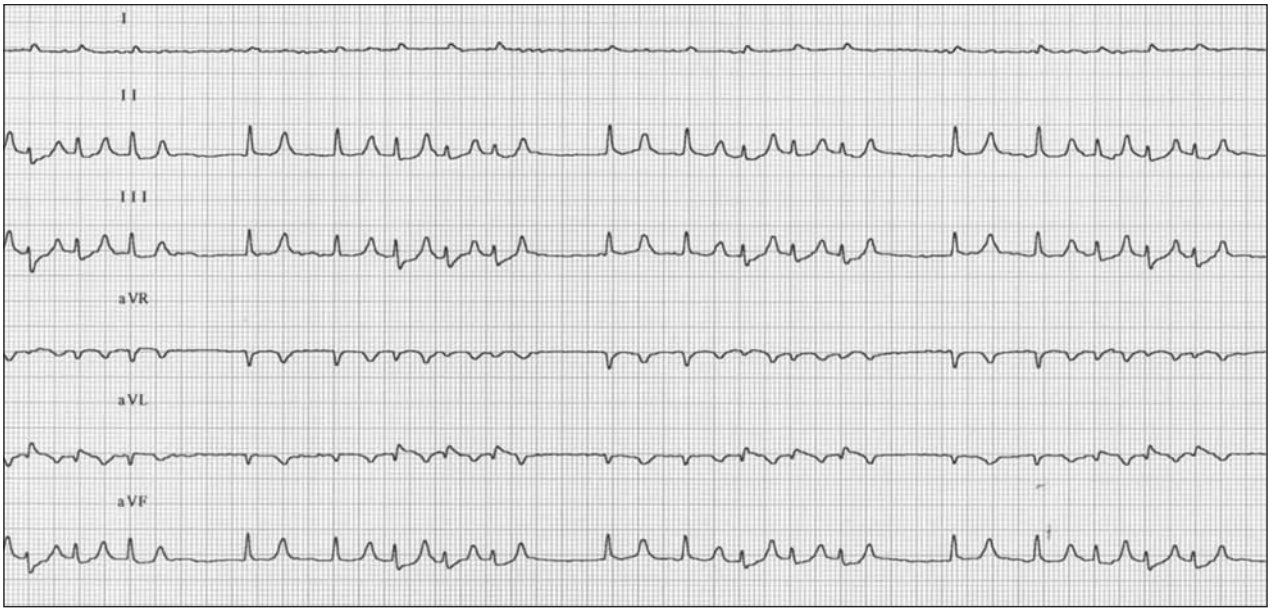

Figure 2 —Six-lead ECG recording obtained the following day (day 1) from the cat in Figure 1. The cat had received lactated Ringer's solution (19 mL/h, IV) for 10 hours; furosemide (10 mg) was administered once IV to promote urine production and potassium wasting. Serum potassium concentration at the time of this ECG examination was 7.9 mEq/L. The ECG diagnosis was sinoventricular rhythm with paroxysms of supraventricular tachycardia with aberrant conduction (QRS complexes of right bundle branch block morphology). Paper speed = 50 mm/s; 2 cm = 1 mV.

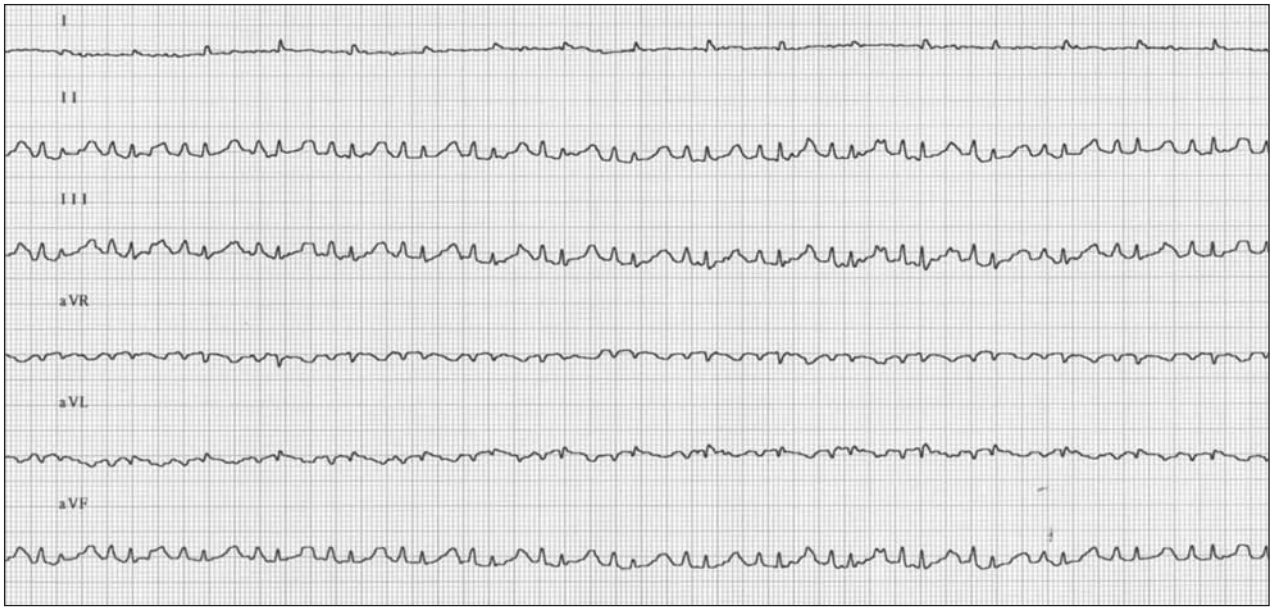

Figure 3 —Six-lead ECG recording obtained the following day (day 2) from the cat in Figure 1. The cat had received saline (0.9% NaCl) solution (7 mL/h, IV) for another 18 hours. Serum potassium concentration at the time of this ECG examination was 4.8 mEq/L. The recording revealed a normal sinus rhythm. Paper speed = 50 mm/s; 2 cm = 1 mV.

Discussion

The ECG effects of hyperkalemia in several species have been well described.[1,2] A biphasic effect of hyperkalemia on conduction and excitation may be observed, depending upon the absolute level of the resting membrane potential and the difference between the resting and the threshold potential. Increases in serum potassium concentration may initially speed up the repolarization of the ventricular myocardium because the ion gradient across the cell membrane decreases and the resting membrane potential of atrial myocytes becomes less negative. This initially causes increased excitability as the difference between the resting and threshold potentials is acutely lessened. Subsequently, conduction and excitability are reduced because the absolute resting membrane potential becomes less negative and a proportion of fast sodium channels become inactivated; thus, the cells' ability to depolarize is reduced.[3] Even though phase 3 of the action potential is more rapid because of increased membrane permeability to potassium, the slope of phase 4 is often slowed, which suppresses automaticity. Slow calcium channels remain functional in the sinus node and the atrial and nodal conducting fibers, thereby allowing transmission of impulses; however, conduction is slowed.[1] Atrial tissue is more sensitive to the effects of hyperkalemia than is ventricular or conduction tissue. It is known that as serum potassium concentration increases, sinoatrial block develops; however, ECG recordings of the sinus node and Purkinje system have revealed continued sinus activity and 1:1 conduction to the ventricles even when bizarre QRS complex morphology and atrial standstill are evident on surface ECG traces.[4] Sinus impulses are carried silently along the atrial conducting system to the atrioventricular bundle and the ventricles, which depolarize slowly. The progressive ECG changes are tented T waves, loss of P waves, bradycardia, widening of QRS complexes, and eventually asystole or ventricular fibrillation. The initial ECG examination of the cat of this report revealed the classic changes associated with hyperkalemia.

Wide-complex tachycardia in hyperkalemic cats has been reported.[5] Sympathetic drive is expected to be quite high in a typical hospitalized feline patient. In the cat of this report, fluid overload with overt congestive heart failure was evident via echocardiography. Volume overload and subsequent increased diastolic blood pressure result in a reflex tachycardia through increased sympathetic tone and decreased vagal stimulation.[6] Congestive heart failure also increases sympathetic tone.[7] This sympathetic drive likely transiently induced a tachycardic rhythm despite the severe hyperkalemia in the cat of this report. The abrupt onset and termination of the tachyarrhythmia could be consistent with a paroxysm of supraventricular tachycardia with aberrant conduction or possibly a paroxysm of ventricular tachycardia. Because of the relatively narrow QRS complexes (compared with the ventricular ectopic beat) and the progressive increase in QRS complex duration, we believed that a paroxysm of supraventricular tachycardia with decremental conduction delay was more likely. Results of studies[5,8,9] of hyperkalemic cats and humans indicated that electrical conduction is sufficiently slowed to allow transient partial or complete block in the atrioventricular node, His-Purkinje system, or ventricular myocardium. These changes in hyperkalemic myocardium are particularly apparent at higher heart rates, when the tachycardia may exacerbate conduction delay or transient conduction failure in the ventricles. Right bundle branch block is the most commonly described conduction abnormality,[9] although left bundle branch block, bifascicular block, complete atrioventricular block, and intermittent escape beats have all been reported.[5,8,10,11] In the cat of this report, it appears that when the heart rate increased to 315 beats/min, the already reduced maximum conduction capacity of the right bundle branch was exceeded, which resulted in conduction delay (QRS complexes with right bundle branch morphology) and possibly conduction block.[12] The intermittent pauses allowed the ventricular conduction tissue to fully repolarize such that the subsequent sinus impulses were more normally conducted with an upright QRS complex configuration.

When the cat's serum potassium concentration normalized, the ECG recordings had a normal appearance, which supported the hypothesis that the underlying cause of the arrhythmias was hyperkalemia. This case illustrates the fact that hyperkalemia may induce rhythms other than the classic sinoventricular rhythm that is generally described, and suggests that hyperkalemia should be a consideration in a tachycardic feline patient with aberrant conduction.

References

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