ECG of the Month

A 16-month-old 3.73-kg (8.21-lb) neutered male domestic shorthair cat that had outdoor access was evaluated at a local veterinary service because of lethargy, expectoration of clear mucus, anorexia, and adipsia of 1 day's duration. Physical examination revealed tachypnea with a restrictive respiratory pattern, increased bronchovesicular pulmonary sounds, muffled heart sounds on the left side, and estimated 7% dehydration. After receiving 200 mL of fluids SC, the cat was referred to the Kansas State University Veterinary Health Center.

At the referral examination, the cat was lethargic and hypothermic (36.7°C [98.1°F]) with a heart rate of 150 beats/min. The respiratory rate was 36 breaths/min; respiratory effort was increased, and oxygen

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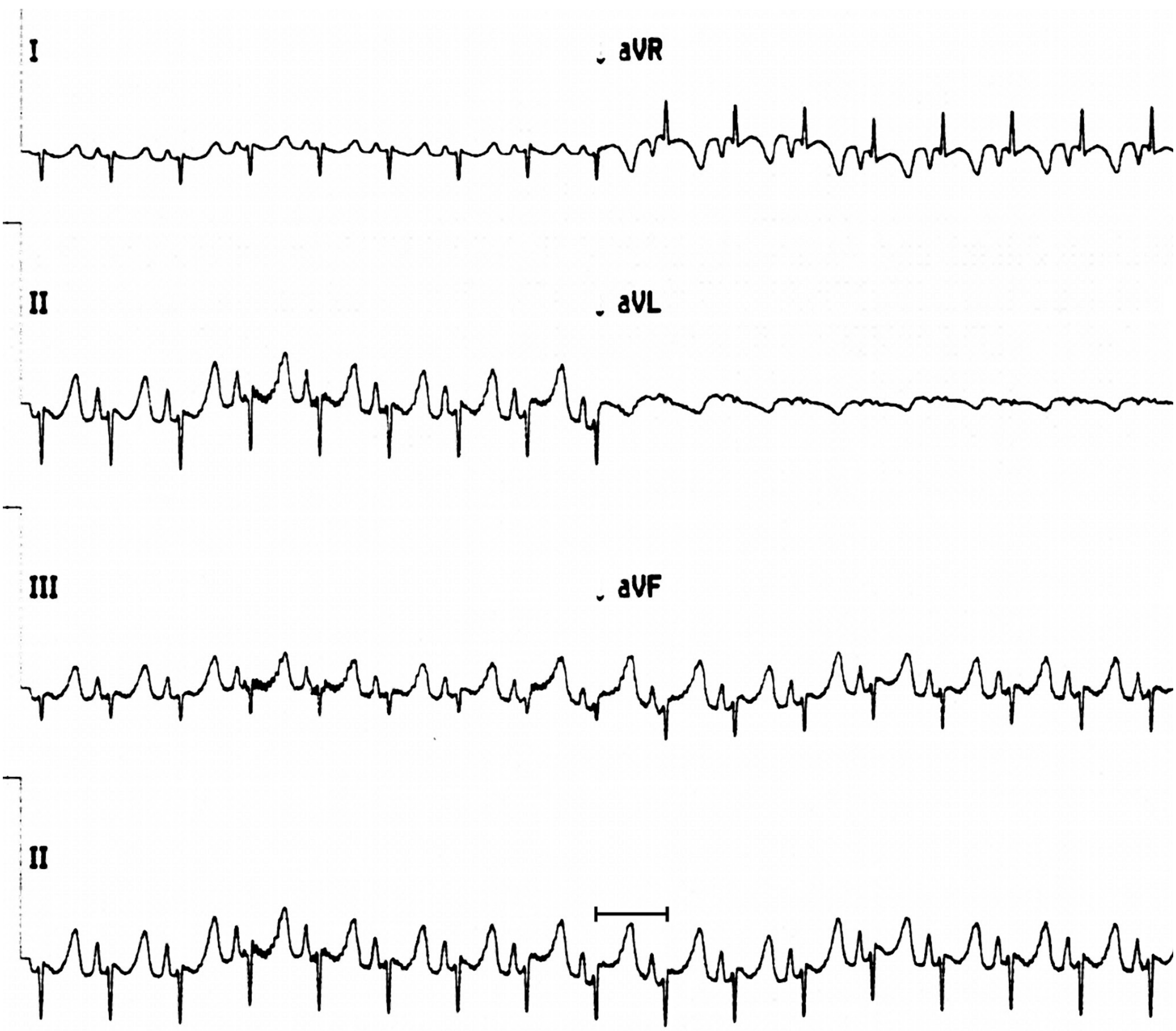

Figure 1—Six-lead surface ECG recording obtained from a 16-month-old indoor-outdoor male domestic shorthair cat that was evaluated because of lethargy, expectoration of clear mucus, anorexia, and adipsia of 1 day's duration. A physical examination performed prior to referral revealed tachypnea with a restrictive respiratory pattern, increased bronchovesicular pulmonary sounds, muffled heart sounds on the left side, and estimated 7% dehydration. At the referral examination, the cat was lethargic and hypothermic. The respiratory rate was 36 breaths/min, and respiratory effort was increased. Oxygen saturation (as measured by pulse oximetry) was 85%. Beating of the heart against the right thoracic wall was noticeable. The ECG tracings indicated a heart rate of 200 beats/min and a sinus rhythm conducted with right-axis deviation (mean electrical axis, –120°). Paper speed, 25 mm/s; 2 cm = 1 mV.

saturation (as measured by pulse oximetry) was 85%. Beating of the heart against the right thoracic wall was noticeable. A CBC and serum biochemical profile revealed a stress leukogram characterized by neutrophilia (10.6×10^3 neutrophils/μL; reference interval, 1.9×10^3 neutrophils/μL to 8.1×10^3 neutrophils/μL), lymphopenia (0.5×10^3 lymphocytes/μL; reference interval, 1.1×10^3 lymphocytes/μL to 9.9×10^3 lymphocytes/μL), and monocytosis (1.2×10^3 monocytes/μL; reference interval, 0×10^3 monocytes/μL to 0.5×10^3 monocytes/μL); hyperglycemia (255 mg/dL; reference interval, 80 to 130 mg/dL); hypocalcemia (8.9 mg/dL; reference interval, 9.2 to 12 mg/dL); hyponatremia (145 mmol/L; reference interval, 149 to 159 mmol/L); hypochloremia (104 mmol/L; reference interval, 111 to 120 mmol/L); hyperphosphatemia (5.8 mg/dL; reference interval, 2.6 to 5.3 mg/dL); and high creatine kinase activity (855 U/L; reference interval, 60 to 420 U/L). An ECG examination was also performed.

ECG Interpretation

The initial 6-lead surface ECG recording was obtained with the patient in right lateral recumbency **(Figure 1)**. Positive QRS complexes in the lead aVR and negative QRS complexes in leads I, II, III, and aVF were evident. The lead aVL tracing appeared attenuated and difficult to interpret and was likely isoelectric. The mean electric axis (MEA) was deviated toward the right (MEA, –120°; reference interval, 0° to +160°). Mild tachycardia was present, with a calculated heart rate of 200 beats/min. The rhythm was regular, with a P wave present for every QRS complex and a QRS complex present for every P wave. The P waves appeared uniform, indicating a single sinus origin. The narrow QRS complexes in the lead II tracing indicated a supraventricular source and normal conduction. ST segment elevation, saddle-backed ST-T configuration, and tall T waves were also noted.

On the basis of the abnormal ECG findings and respiratory signs, thoracic radiography was performed, and results were consistent with a diaphragmatic hernia (DH). Abdominal exploration revealed a radial tear in the left pars costalis that was completely filled by the spleen, along with the absence of the entire stomach, segments of the jejunum, and left liver lobes in the abdominal cavity. Diaphragmatic defect extension and gastric decompression were required prior to hernia reduction. Apart from congestion of the left lateral and middle liver lobes and the spleen, no other abnormality was found during intraoperative examination of the abdomen and the left hemithorax. After lavage and suction, diaphragmatic herniorrhaphy, with the placement of a transdiaphragmatic catheter, and routine abdominal closure were performed. Continuous IV infusion of fentanyl citrate, short-term transmucosal administration of buprenorphine hydrochloride, and oral administration of robenacoxib were provided after surgery. The cat's oxygen saturation (as measured by pulse oximetry) was 99% without oxygen supplementation within a few hours after surgery. Six-lead surface ECG was also performed immediately following surgery **(Figure 2)**.

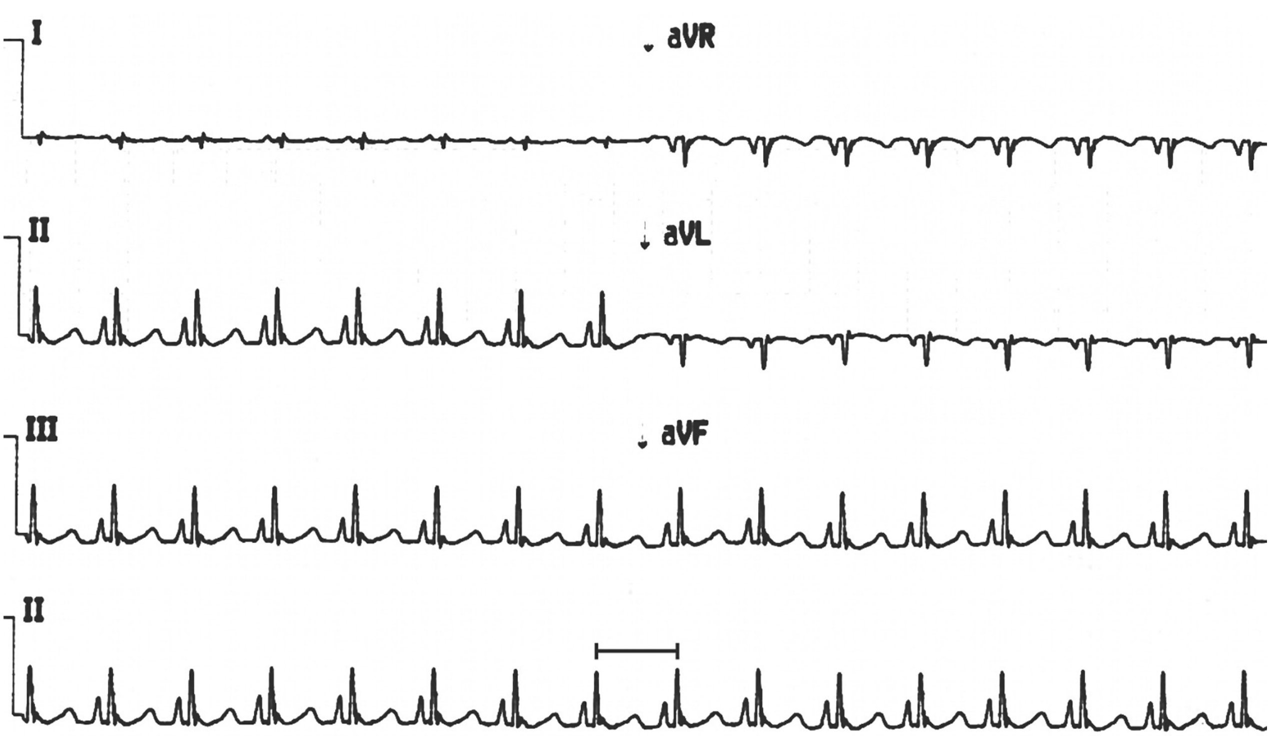

Figure 2—Six-lead surface ECG recording obtained from the cat in Figure 1 immediately after surgical repair of a diaphragmatic hernia. The previously identified right-axis deviation has resolved (mean electrical axis, +90°). The calculated heart rate is 190 beats/min. Paper speed = 25 mm/s; 1 cm = 1 mV.

The postoperative 6-lead surface ECG recording indicated a mildly high heart rate of 190 beats/min, normal sinus rhythm, and correction of the right-axis deviation (MEA, +90°). The QRS complexes were positive in leads II, III, and aVF; negative in leads aVR and aVL; and isoelectric in lead I. The ST segment and T wave changes had also resolved.

Discussion

An MEA shift to the right can be caused by rightward heart movement within the thoracic cavity, right ventricular (RV) enlargement (hypertrophy or dilation), or right bundle branch block (RBBB) or can be a normal variation.[1] Both RV enlargement and RBBB can be associated with ECG features such as right-axis deviation, deep S waves, and wide QRS complexes; the MEA shift is a result of a right-sided muscle mass increase in cases of RV enlargement and prolonged RV depolarization through myocardiocytes in cases of RBBB.[1] The lack of preoperative QRS complex prolongation argued against conduction disturbances as the cause of the MEA deviation[2] in the cat of the present report. The normal appearance of the postoperative ECG tracing obtained from the cat made RV enlargement (which can be associated with hemodynamically important conditions such as arrhythmogenic RV cardiomyopathy in cats[2,3]) unlikely. It should be noted that a diagnosis of heart enlargement should not be made on the basis of ECG findings but on the basis of the results of radiography or, preferably, echocardiography.[1]

Conditions of the pericardium may also lead to MEA deviation. In cats with peritoneopericardial DH, changes in the position of the heart can result from the herniation of abdominal viscera.[2] Pericardial effusion in cats has also been associated with MEA deviation and conduction abnormalities.[4] Electrocardiography performed on 126 cats with pericardial effusion (diagnosis obtained by means of echocardiography) revealed right-axis deviation and RBBB in 8.3% and 2.3% of those cats, respectively; furthermore, left-axis deviation and left bundle branch block were detected in 11.1% and 4.7% of the cats, respectively.[4]

It is worth noting that ECG measurements can be affected by a patient's body position during the acquisition process. In a study[5] involving 41 cats that underwent ECG examination while positioned sequentially in right lateral, sternal, and left lateral recumbency, the MEA when cats were in right lateral recumbency (mean MEA ± SD, 26.1° ± 77.7°) was significantly different from that recorded when cats were in left lateral recumbency (mean MEA, 45.9° ± 70.2°). Therefore, accurate MEA results are best obtained with cats in right lateral recumbency.[5] Furthermore, a study[6] in dogs revealed substantial effects of left forelimb position on the appearance of the QRS complexes in some leads. Because MEA triangulation is dependent on the amplitudes of QRS complexes in 2 chosen leads,[1] forelimb positions could impact MEA calculations.

The case described in the present report involved right-axis deviation in association with DH in a cat. The cat's indoor-outdoor lifestyle made trauma a possible cause, but the nature of the lesion could not be determined on the basis of available information. The rightward MEA shift was attributed to the presence of herniated viscera in the left hemithorax and to the resultant right-sided displacement of the heart. The finding of a positive P vector—an indication of preservation of the mean direction of the atrial depolarization wave—could be explained by the smaller allowable change in anatomic position of the atria owing to their relatively fixed position near the heart base. In another report,[7] a Maine Coon that underwent peritoneopericardial DH repair subsequently developed an arrhythmia and a leftward MEA shift (with a left anterior fascicular pattern) that resolved after the identification and removal of a newly formed pericardium-associated cyst within the ventrolateral right hemithorax. When assessing axial deviation, a shift in cardiac position as a result of a space-occupying intrathoracic mass should be considered.

In the case described in the present report, the ST and ST-T segments in the preoperative ECG recording were of interest. The elevated ST segments and the tall T waves were most likely caused by myocardial hypoxia.[8] However, the ST elevation and saddle-backed ST-T configuration can be consistent with a type II Brugada pattern. In humans, the Brugada ECG pattern (type I or II) is observed in individuals with Brugada syndrome, a congenital channelopathy of the heart, or with Brugada phenocopies, an array of clinical conditions known to result in the same ECG phenomenon that is reversible in nature.[9] Of the reported causes of Brugada phenocopies, electrolyte imbalances and mediastinal compression were of particular relevance to the present case.[9] The postoperative resolution of the ST-segment and T-wave changes was most supportive of the etiologic role of myocardial hypoxia. Furthermore, the ECG characteristics of the Brugada phenomenon in precordial lead (V1, V2, and V3) recordings obtained from humans[7] make the interpretation of the resemblance of the frontal lead ECG pattern detected in the cat of the present report difficult. However, partial contribution of one or more of the aforementioned conditions underlying Brugada phenocopies to the observed ST and ST-T segment changes cannot be ruled out.

Radiography of the thorax is the preferred imaging technique for the initial diagnosis of traumatic DH and can also be used to evaluate concurrent intrathoracic conditions, such as pleural effusion.[10] Thoracic ultrasonography is a useful complement to radiography, particularly in the presence of pleural effusion, and aids in the differentiation of DH from pulmonary parenchymal lesions or peritoneopericardial DH from a primary cardiac condition.[11]

Right-axis deviation in cats can be caused by a range of conditions, such as a change in cardiac position because of a space-occupying lesion within the thorax, RV enlargement, RBBB, or pericardial diseases. The cat of the present report had an MEA deviation that was presumed to result from cardiac displacement secondary to development of a DH. The resolution of right-axis deviation after surgical correction of the hernia made a primary heart condition unlikely. Clinicians should consider DH when assessing patients with a shift in MEA.

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