ECG of the Month

A 10-year-old 4.5-kg (9.9-lb) castrated male Siamese cat was referred to the Oradell Animal Hospital because of suspected pericardial and pleural effusions. The cat had been examined immediately prior to the hospital visit by the referring veterinarian because of a decline in appetite of 1 week's duration and a decline in activity level during the preceding 24 hours. The effusions were detected via thoracic radiography. The cat was kept indoors and fed a diet of ground beef and a dry formulation of commercial cat food.

At the hospital, the cat was recumbent and had signs of depression; its body condition score was 4 (scale of 1 [overly thin] to 9 [obese]). The mucous membranes were pale and tacky, and capillary refill time was 2 seconds. The degree of dehydration was estimated at 9%. The cat's heart rate was 160 beats/min with an audible diastolic gallop; the respiratory rate was 28 breaths/min, and a slight increase in respiratory effort was evident. On auscultation, heart and lung sounds were muffled ventrally. Pulse quality was poor. Rectal temperature was 36.2°C (97.1°F). Systolic arterial blood pressure (determined by use of a Doppler ultrasonographic technique) was markedly low (54 mm Hg; lower reference limit, 107 mm Hg³).

Thoracocentesis was performed, and 130 mL of serosanguineous fluid was removed. Serum biochemical analyses revealed a mild hyperglycemia (261 mg/dL; reference range, 64 to 170 mg/dL), moderate azotemia (BUN concentration, 64 mg/dL [reference range, 14 to 36 mg/dL]; creatinine concentration, 3.6 mg/dL [reference range, 0.6 to 2.4 mg/dL]), hyperbuninemia (2.2 g/dL; reference range, 2.5 to 3.9 g/dL), hyperbilirubinemia (1.6 mg/dL; reference range, 0.1 to 0.4 mg/dL), hypocalcemia (7.4 mg/dL; reference range, 8.2 to 10.8 mg/dL), and hyponatremia (142 mEq/L; reference range, 145 to 158 mEq/L). Serum potassium concentration was with reference limits (4.3 mEq/L; reference range, 3.6 to 5.6 mEq/L). A CBC revealed evidence of a stress leukogram with neutrophilia, lymphopenia, and monocytosis. Serum thyroxine concentration was low (0.6 µg/dL; reference range, 0.8 to 4.0 µg/dL). Echocardiography revealed a severely dilated left ventricle with poor systolic function. Mild right ventricular and right atrial dilation were present. Mild mitral valve and tricuspid valve regurgitation were detected. The left atrium was markedly large. Pleural effusion was detected via echocardiography. An ECG recording was obtained during the echocardiographic examination (Figure 1).

ECG Interpretation

Review of the initial ECG tracing revealed a heart rate of 180 beats/min (Figure 1). The duration of QRS complexes was prolonged (0.07 seconds). Q waves were not evident in leads I, II, III, or aVF. The ST segment was slurred, and the t wave was subjectively large. Differential diagnoses were an accelerated idioventricular rhythm (ventricular origin rhythm) or a normal sinus rhythm with left bundle branch block. It could not be determined whether the wide QRS complexes were supraventricular or ventricular in origin because occurrence of a p wave immediately following a t wave could not be distinguished from a biphasic t wave. An extended ECG recording was obtained and revealed an isolated premature complex that was most consistent with a supraventricular origin; the following pause allowed detection of a distinct p wave and the determination that the predominating rhythm was sinus in origin (Figure 2). The PR interval was prolonged (0.11 seconds), indicative of first-degree atrioventricular (AV) block. The mean electrical axis was considered normal (+80°), and QRS complexes were positive in leads I, II, III, and aVF on a 6-lead recording. The 2 causes of prolonged duration of supraventricular-origin QRS complexes are ventricular enlargement or a conduction disturbance. The cat’s abnormally large heart could have contributed to the wide QRS complexes; however, complexes that are prolonged to that extent and that occur in association with the detected changes in ST segments and t waves are thought to be consistent with left bundle branch block. Hyperkalemia could also cause similar ECG

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Figure 1—Lead II ECG trace obtained from a cat with severe dilated cardiomyopathy, abnormally large left and right atria, pleural effusion, and severe hypotension. Heart rate and rhythm are regular. The duration of the QRS complexes is prolonged (0.07 seconds), and there is slurring of the ST segment. On this trace, it cannot be determined whether the wide QRS complexes have a sinus origin (with a p wave occurring directly after the t wave) or have a ventricular origin (with a biphasic t wave). Paper speed = 50 mm/s; 10 mm = 1 mV.
changes, but this abnormality was ruled out on the basis of the clinicopathologic findings.

Discussion

Dilated cardiomyopathy of unknown etiology was the primary diagnosis in the cat of this report. It was thought that most of the clinicopathologic abnormalities were a consequence of forward heart failure and severe hypotension, although underlying chronic renal insufficiency could not be ruled out. The cat’s condition continued to worsen despite aggressive treatment with positive inotropes, and given the grave prognosis, euthanasia was elected. A necropsy or further diagnostic testing, such as assessment of serum taurine concentration, was not permitted.

Left bundle branch block is associated with a lesion of the conduction system that affects either the main bundle branch as it extends off the bundle of His or a larger lesion that affects both the anterior and posterior fascicles of the left bundle branch. The lack of a Q wave in this ECG suggested the former. If the bundle branch block was a result of damage to the fascicles, one would expect the intact main left bundle branch to cause normal depolarization of the septum and therefore produce a Q wave. In this case, the lack of a Q wave suggested abnormal conduction in the septum due to dysfunction of the left main bundle branch. When left bundle branch block is present, conduction occurs rapidly through the AV node and then through the bundle of His and right bundle branch. Depolarization of the left ventricle is much slower, occurring cell to cell through the myocardium rather than via the high-speed conduction system. This results in a prolonged time of depolarization and marked prolongation of the QRS complexes on ECG recordings.

Conduction disturbances are known to be associated with structural cardiac disease in cats with cardiomyopathy. In a histologic study in which the conduction systems in 63 cats with cardiomyopathy were investigated, all cats had degeneration or fibrosis of the AV node and the bundle branches. More recently, a study performed in which the cardiac conduction systems in 13 cats with hypertrophic cardiomyopathy and complete AV block were examined histologically. Fibrotic changes that affected the AV bundle and upper portions of the left and right bundle branches and a reduction of conduction fibers were detected in all affected cats in that study. These changes differed substantially, both qualitatively and quantitatively, from normal aging changes observed within a control group.

On the basis of the histopathologic changes in cats with cardiomyopathy, one would expect that any conduction disturbance of the AV node or the bundle branches could be observed clinically. Complete AV block, left anterior fascicular block, and a combination of a right bundle branch and left anterior fascicular block have been reported in association with cardiomyopathy in cats.

Complete left bundle branch block develops rarely in cats; to the authors’ knowledge, this type of conduction block has been identified in 1 cat with hypertrophic cardiomyopathy and 1 cat in which there was no evidence of structural cardiac disease. In the latter report, the cat had substantial noncardiac disease and electrolyte disturbances; in addition to the left bundle branch block, second-degree AV block was detected. Diffuse lymphohistiocytic myocarditis with focal muscle fiber necrosis in the ventricular septum was detected at necropsy and was considered the likely cause for the conduction disturbances. The cat of this report is a rare example of left bundle branch block in this species and highlights the fact that left bundle branch block can develop in a cat with dilated cardiomyopathy. It is noteworthy that the ECG tracing obtained for the cat could easily have been incorrectly assessed a ventricular-origin rhythm if the clinician had not spent the time to perform an extended ECG examination and identify a point of interruption in the rhythm. Electrocardiograms of long duration can be extremely helpful when evaluating any type of tachycardia.

References