What Is Your Diagnosis?

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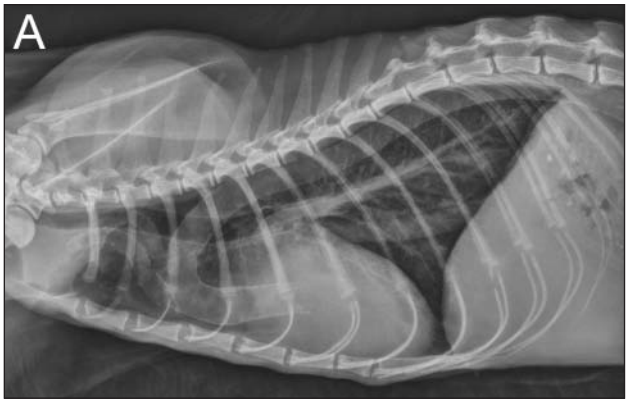

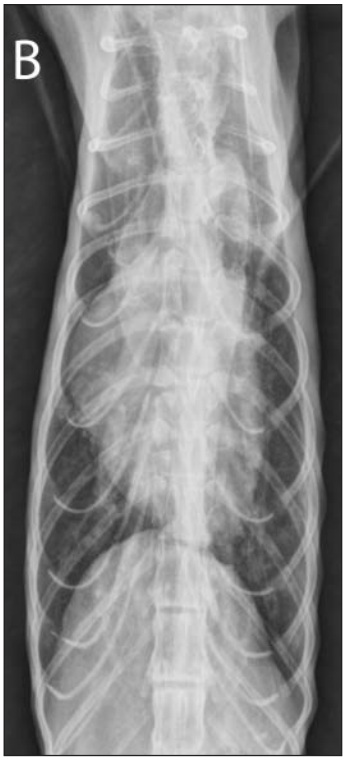

Figure 1—Right lateral (A) and dorsoventral (B) radiographic views of the thorax of a 4-year-old spayed female Maine Coon cat evaluated for acute collapse and a 2-month history of progressively decreasing appetite and weight loss.

History

A 4-year-old spayed female Maine Coon cat was evaluated for acute collapse and a 2-month history of progressively decreasing appetite and weight loss. Dry kibble commercial cat food was being fed, which had expired according to the date on the food bag. Results of tests performed within the last 6 months for detection of FeLV and FIV infection were negative.

During physical examination, the cat remained in lateral recumbency. It had an extremely poor body condition (body condition score, 1/9) and shallow respirations. Oral mucous membranes were pale and tacky, with a capillary refill time of > 2 seconds. A grade 4/6 right parasternal systolic murmur was evident during thoracic auscultation; the cat had bounding femoral pulses and jugular pulsations. Two small nodules could be palpated over the midventral portion of the trachea in the region of the thyroid glands. Slightly small, firm kidneys were found during abdominal palpation. Serum biochemical analysis and CBC revealed high BUN (130 mg/dL; reference range, 16 to 36 mg/dL) and serum creatinine (5.3 mg/dL; reference range, 0.8 to 2.4 mg/dL) concentrations. Compared with reference range values, there were substantial increases in serum sodium (187.9 mmol/L; reference range, 147 to 153 mmol/L), potassium (5.58 mmol/L; reference range, 3.91 to 4.40 mmol/L), and chloride (149.5 mmol/L; reference range, 110.6 to 115.5 mmol/L) concentrations. The cat had severe anemia (PCV, 7%; reference range, 30% to 45%), a total serum calcium concentration within the reference range (9.1 mg/dL; reference range, 7.8 to 11.3 mg/dL), and severe hyperphosphatemia (16.1 mg/dL; reference range, 3.1 to 7.5 mg/dL). The calcium-phosphorus product was 147 mg^2/dL^2 (reference limit, < 70 mg^2/dL^2). Radiographs of the thorax were obtained (**Figure 1**).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

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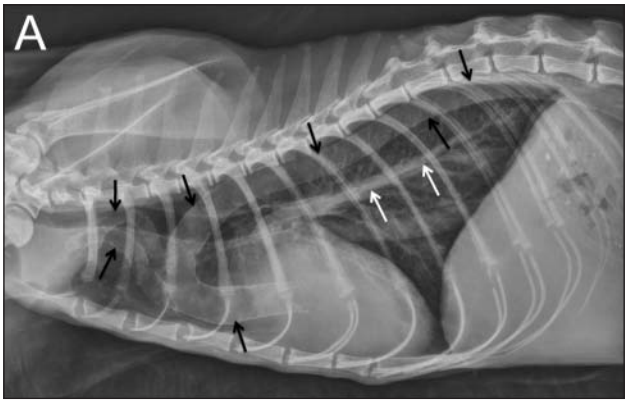

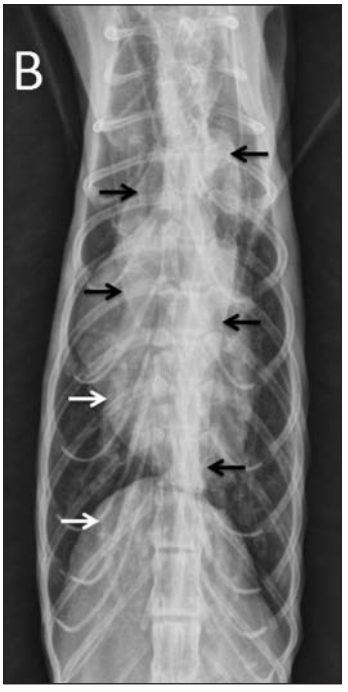

Figure 2—Same radiographic images as in Figure 1. On both the lateral and the dorsoventral views, the ascending and descending aorta, brachiocephalic trunk, and left subclavian artery (black arrows) have increased radiopacity due to mineralization. The pulmonary arteries are mineralized (white arrows). A mild, diffuse bronchointerstitial pattern is also observed and possibly caused by mineralization.

Radiographic Findings and Interpretation

The walls of the aorta and associated aortic branches have a mineral opacity (vascular mineralization; **Figure 2**). This is observed in the ascending and descending aorta, brachiocephalic trunk, left subclavian artery, and pulmonary arteries. This may be caused by disorders of calcium metabolism such as hyperparathyroidism, neoplasia, and vitamin D toxicosis, or systemic hypertension. There is also a mild, diffuse bronchointerstitial pattern, which may be attributable to tissue mineralization or inflammatory lower airway disease. The cardiac silhouette is uniformly enlarged with a vertebral heart score of 9.5 (reference limit, < 8.1 for cats). This may be caused by primary cardiac disease or pericardial effusion.

Comments

The severe vascular mineralization seen in these radiographs along with severe azotemia made renal secondary hyperparathyroidism the top differential diagnosis. Other differential diagnoses for the mineralized aorta included hypercalcemia of malignancy, idiopathic hypercalcemia, vitamin D toxicosis, granulomatous disease, systemic hypertension, and heart disease. Euthanasia was elected. Diffuse mineralization of the aorta, coronary arteries, and mitral valve was observed during necropsy. There was a small volume of fluid present in the pericardial sac. The parathyroid glands were markedly hyperplastic bilaterally. The lungs had severe, multifocal, alveolar, and bronchiolar mineralization. The kidneys had evidence of marked tubular degeneration, fibrosis, interstitial nephritis, and mineralization bilaterally. Numerous birefringent light yellow crystals were found within the renal tubules, suggestive of melamine cyanurate.

The clinical signs, diagnostic imaging changes, and necropsy findings support a diagnosis of melamine cyanurate-associated nephrotoxicosis, with renal secondary hyperparathyroidism and severe metastatic calcification of the aorta and other soft tissues. This type of mineralization can be caused by dystrophic, metastatic, or idiopathic calcification.

Dystrophic mineralization is caused by deposition of calcium salts in damaged tissue, with serum calcium and phosphorus concentrations within reference ranges.[1] Vascular mineralization secondary to hypertension-induced arteriosclerosis has been reported as a cause of dystrophic mineralization.[2] Although the cat in this report had a blood pressure reading within the reference range at the time of evaluation, this type of arteriosclerosis cannot be entirely excluded as a cause of the mineralization.

Metastatic mineralization is associated with abnormal calcium-phosphorus metabolism, usually attributable to a disorder resulting in hypercalcemia, hyperphosphatemia, or both. Renal secondary hyperparathyroidism is associated with calcium-phosphorus imbalance. A high calcium-phosphorus product is predictive of the tendency for metastatic calcification of soft tissues in affected animals.[3]

Pet food–associated nephrotoxicosis caused by melamine and cyanuric acid was reported in March 2007.[4] A massive recall helped limit the number of affected pets, but the bag of food being offered to this cat had been purchased prior to the recall. Melamine cyanurate–associated nephrotoxicosis has been reported to cause both acute and chronic renal damage.[4,5] The tissue mineralization in the cat of this report was suspected to be caused by chronic kidney disease with associated renal secondary hyperparathyroidism. Cats with vascular mineralization should be evaluated for cardiovascular diseases and calcium-phosphorus imbalance.

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2. Lefbom BK, Adams WH, Weddle DL. Mineralized arteriosclerosis in a cat. *Vet Radiol Ultrasound* 1996;37:420–423.
3. Ettinger SJ, Feldman EC. *Textbook of veterinary internal medicine*. 6th ed. Elsevier, 2005;1767–1785.
4. Brown CA, Jeong KS, Poppenga RH, et al. Outbreaks of renal failure associated with melamine and cyanuric acid in dogs and cats in 2004 and 2007. *J Vet Diagn Invest* 2007;19:525–531.
5. Puschner B, Poppenga RH, Lowenstine LJ, et al. Assessment of melamine and cyanuric acid toxicity in cats. *J Vet Diagn Invest* 2007;19:616–624.