**What Is Your Diagnosis?**

*Figure 1—Right lateral (A) and ventrodorsal (B) radiographic views of the abdomen of a 3-year-old spayed female mixed-breed dog evaluated because of chronic weight loss and chronic vomiting of 1 year’s duration.*

**History**

A 3-year-old 8-kg (17.6-lb) spayed female mixed-breed dog was evaluated because of chronic weight loss and vomiting of 1 year’s duration, with episodic diarrhea reported by the owners. On clinical evaluation, the dog was severely cachectic (body condition score, 1/9), with no other abnormalities found on physical examination. A CBC revealed leukocytosis (20.7 × 10³ leukocytes/µL; reference range, 8 × 10³ leukocytes/µL to 14.5 × 10³ leukocytes/µL), neutrophilia (16.8 × 10³ neutrophils/µL; reference range, 3 × 10³ neutrophils/µL to 11.5 × 10³ neutrophils/µL), and monocytosis (1.7 × 10³ monocytes/µL; reference range, 0.1 × 10³ monocytes/µL to 1.4 × 10³ monocytes/µL). Serum biochemical analysis revealed hypoproteinemia (4.4 g/dL; reference range, 5.8 to 7.5 g/dL), hypoalbuminemia (1.5 g/dL; reference range, 2.6 to 4.2 g/dL), hypercholesterolemia (131 g/dL; reference range, 150 to 240 g/dL), mild hyperkalemia (5.9 mmol/L; reference range, 3.8 to 5.5 mmol/L), and hyperchloremia (120 mmol/L; reference range, 107 to 115 mmol/L). The dog had a total calcium concentration within reference range (10.3 mg/dL; reference range, 9.4 to 11.4 mg/dL), and blood phosphorus concentration was mildly decreased (2.9 mg/dL, reference range, 3.4 to 6.3 mg/dL). Because of the gastrointestinal clinical signs, abdominal radiography was performed (Figure 1).

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

On the abdominal radiographs, the patient appeared severely emaciated, with severe loss of abdominal serosal detail, attributed to the poor body condition of the dog. Radiographic assessment of the abdominal organs was therefore difficult because of the lack of abdominal contrast. An ill-defined, irregularly shaped heterogeneous mineralized structure with multiple linear mineral opacities associated with it was observed within the right cranial abdominal quadrant, in the region of the gastric body and pyloric antrum (Figure 2). On the basis of these radiographic findings, a mineralized foreign body within the gastric body and pyloric antrum was considered likely.

The severe emaciation was assumed to be secondary to partial chronic pyloric outflow obstruction. Differential diagnoses for this lesion included gastric wall mineralization or a mineralized mass in the right cranial aspect of the peritoneal cavity.

Abdominal ultrasonography was performed and revealed mild distension of the stomach with intraluminal gastric content (Figure 3). A large hyperechoic interface with strong distal shadowing was also observed at the level of the stomach. The duodenal mucosa was mildly hyperechoic with usual thickness and layering otherwise. Gastrointestinal hyperperistalsis and severe mesenteric lymphadenopathy were observed. Because of the severe ultrasonographic distal acoustic shadowing within the stomach, it was not possible to differentiate a gastric foreign body from severe gastric wall mineralization.

Treatment and Outcome

For further evaluation, upper gastrointestinal endoscopy was performed. No foreign material was found within the gastric or duodenal lumen, but severe discoloration and thickening of the duodenal mucosa and mild discoloration of the gastric mucosa were observed. Mucosal biopsy samples of the duodenum and pyloric antrum were obtained endoscopically.
Histopathologic results were consistent with focal severe granulomatous duodenitis with intraluminal trematode eggs and also diffuse mild lymphoplasmacytic and eosinophilic duodenitis samples, but there was no evidence of mucosal mineralization or granulomas. Finally, results of a fecal saline sedimentation test revealed medium numbers of *Heterobilharzia* spp, leading to the final diagnosis of gastrointestinal schistosomiasis. The dog was treated with praziquantel (40 mg/kg [18.2 mg/lb], PO, q 24 h, for 6 days) and cyanocobalamin (400 µg, SC, q 7 d, for 4 weeks, then every other week). Four months after initiation of treatment, the dog had regained some weight (body condition score, 2/9) and no more gastrointestinal signs were reported.

**Comments**

*Heterobilharzia* infection in dogs occurs mainly in the southeastern United States. Treatment consists of oral administration of high doses of praziquantel (25 to 40 mg/kg [11.4 to 18.2 mg/lb], q 12 or 8 h, for 3 to 10 days) or fenbendazole (40 mg/kg, q 24 h, for 10 days) and is usually effective, resulting in resolution of the clinical signs in most affected dogs. Infected dogs have adult *Heterobilharzia* worms present in the mesenteric veins, where oviposition occurs. Some eggs will also spread hematogenously to the liver or other organs, such as lymph nodes, stomach, pancreas, spleen, kidneys, brain, and lungs. A severe granulomatous reaction can induce tissue alteration and necrosis. Injured or dying cells are not able to maintain normal calcium homeostasis, leading to increased intracellular calcium concentration and secondary dystrophic mineralization of the affected tissues. *Heterobilharzia americana* infection and more generally granulomatous diseases have also been reported to induce hypercalcemia secondary to hypercalcitriolemia or parathyroid hormone-related protein synthesis. In these instances, when extracellular calcium concentration exceeds the homeostatic capacity of cells and tissues (when the calcium-phosphorus concentration product exceeds 60 to 70 mg²/dL²), soft tissue mineralization can also occur and is then termed metastatic mineralization. In contrast to dystrophic mineralization, metastatic calcification is associated with hypercalcemia and is typically more generalized, although some tissues are more prone to mineralization (eg, gastrointestinal tract, kidneys, cardiac and skeletal muscles, intima of the arteries, ligaments, and tendons). In the case described in the present report, the dog was normocalcemic with mild hypophosphatemia, and the calcium-phosphorus concentration product was not high enough to induce calcium deposition within the soft tissue. Therefore, the gastric mineralization observed on the abdominal radiographs was considered to be dystrophic mineralization secondary to severe granulomatous inflammation of the gastric wall. The mineralization likely affected the submucosal, muscularis, or serosal layers, considering that mucosal biopsy samples did not reveal evidence of mineralization.

The ultrasonographic and radiographic appearance of *Heterobilharzia* infection has been described in dogs. Faint linear intestinal wall mineralization can be observed on abdominal radiographs, and multiple mineralized irregular foci are often visible ultrasonographically within the pancreas and hepatic parenchyma. Additional ultrasonographic changes in the gastrointestinal tract have included a thickened intestinal submucosa, with multiple hyperechoic submucosal pinpoint foci also present within the muscularis layer. In these reports, most of the eggs were mineralized, surrounded by granulomatous inflammation, and mainly observed within the small intestinal submucosa on histologic evaluation, with smaller numbers visible in the mucosa and muscularis layers. In the dog of the present report, the lack of mineralization or granulomas on the gastric biopsy samples was therefore most likely the result of the limited thickness of the samples acquired endoscopically, only allowing histologic evaluation of the superficial gastric mucosa.