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What Is Your Diagnosis?

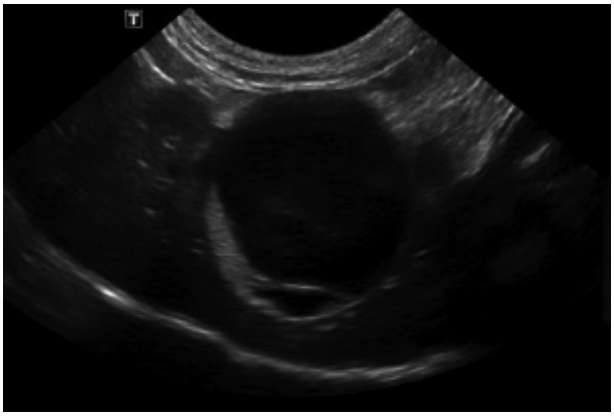

Figure 1—Sagittal, transabdominal ultrasonographic image of the liver and gallbladder of a 7-year-old 4.2-kg spayed female Terrier mixed-breed dog evaluated because of a 72-hour history of vomiting. The image was obtained with the dog in dorsal recumbency, with dorsal toward the bottom of the image and cranial toward the left.

History

A 7-year-old 4.2-kg spayed female Terrier mixed-breed dog was referred to the Louisiana State University School of Veterinary Medical Teaching Hospital for evaluation because of a 72-hour history of vomiting. On physical examination, the dog was alert and had vital signs within reference limits; however, abdominal palpation elicited signs of pain.

Results of a CBC indicated that the dog had a clinically normal Hct (50.1%; reference range, 35% to 54%) and WBC count (14 X 10^3 WBCs/µL; reference range, 8 X 10^3 to 14.5 X 10^3 WBCs/µL) but monocytosis (1.8 X 10^3 monocytes/µL; reference range, 0.1 X 10^3 to 1.4 X 10^3 monocytes/µL), lymphopenia (0.84 X 10^3 lymphocytes/µL; reference range, 1 X 10^3/µL to 4.8 X 10^3 lymphocytes/µL), and thrombocytopenia (185 X 10^3 platelets/µL; reference range, 200 X 10^3 to 500 X 10^3 platelets/µL). Findings on serum biochemical analyses included high activities of aspartate aminotransferase (194 U/L; reference range, 0 to 50 U/L), alanine aminotransferase (4,028 U/L; reference range, 0 to 60 U/L), alkaline phosphatase (2,408 U/L; reference range, 0 to 100 U/L), and γ-glutamyl transferase (45 U/L; reference range, 0 to 8 U/L); high concentrations of total bilirubin (1.7 mg/dL; reference range, 0 to 0.4 mg/dL) and cholesterol (662 mg/dL; reference range, 150 to 240 mg/dL); and low concentrations of potassium (3.3 mmol/L; reference range, 3.8 to 5.5 mmol/L) and chloride (100 mmol/L; reference range, 107 to 115 mmol/L). Results of a coagulation panel to assess prothrombin time and activated partial thromboplastin time were within reference limits. Abdominal radiographic examination was performed (not shown), and findings, reviewed by a board-certified veterinary radiologist (LAG), were unremarkable. Therefore, abdominal ultrasonography **(Figure 1)** was performed to further investigate the cause of vomiting.

Formulate differential diagnoses, then continue reading.

Diagnostic Imaging Findings and Interpretation

The gallbladder was subjectively distended and contained an echogenic, gravity-dependent layer typical of a small volume of gallbladder sludge. A thin hyperechoic rim surrounded an anechoic region at the dorsal periphery of the gallbladder wall **(Figure 2)**. The mesentery adjacent to the gallbladder was severely hyperechoic. The common bile duct, duodenal papilla, liver, and pancreas were ultrasonographically normal. Our primary differential diagnosis list included acute and chronic cholecystitis.

The dog was hospitalized for medical treatment with an isotonic solution of balanced electrolytes (Normosol-R; 3.8 mL/kg/h, IV), fentanyl (3 µg/kg/h, IV), maropitant (1 mg/kg, IV, q 24 h), and S-adenosyl methionine (Denamarin Advanced; 7 mg/kg, PO, q 24 h). Although the dog was alert and active, it showed signs of persistent abdominal pain; therefore, abdominal ultrasonography was repeated on day 3 of hospitalization. Compared with the initial ultrasonographic findings, repeated ultrasonography revealed that the gallbladder was similarly distended but contained more of the hyperechoic material and that the thin hyperechoic rim surrounding an anechoic region associated with the dorsal aspect of gallbladder wall appeared unchanged **(Figure 3)**. The diameter of the common bile duct was 2.9 mm (reference range, < 3 mm), the mesentery surrounding the gallbladder was progressively hyperechoic, the pancreas appeared morphologically normal, and no peritoneal effusion was evident. On the basis of these findings and clinical signs, we prioritized potential gallbladder rupture.

Treatment and Outcome

We initiated treatment with enrofloxacin (5 mg/kg, IV, q 24 h) and ampicillin-sulbactam (30 mg/kg, IV, q 8 h), and the dog underwent exploratory laparoto-

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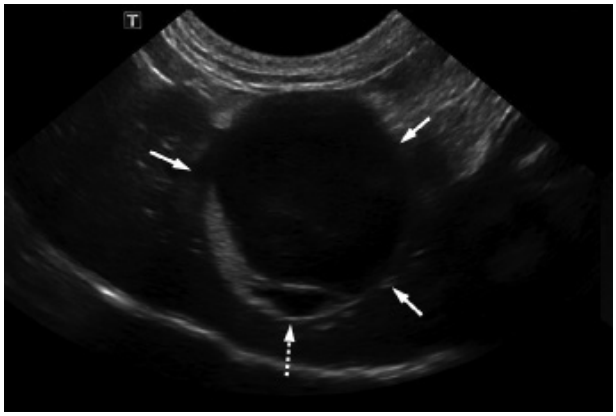

Figure 2—Same image as in Figure 1. The gallbladder (white arrows) is subjectively distended. Evident at the dorsal periphery of the gallbladder wall is a gravity-dependent structure with a thin hyperechoic rim around an anechoic region (dotted arrow).

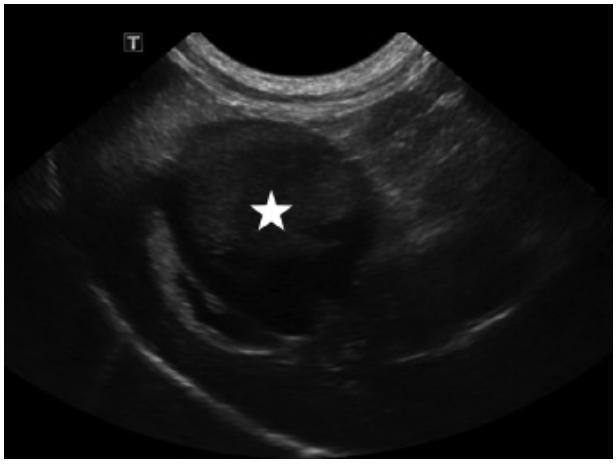

Figure 3—Sagittal, transabdominal ultrasonographic image of the liver and gallbladder of the same dog in dorsal recumbency 3 days later, showing that the gallbladder is similarly distended and that the gravity-dependent structure is basically unchanged but that the lumen of the gallbladder now also contains hyperechoic, nonmotile, non–gravity-dependent material (star).

my. At surgery, the gallbladder was severely distended but appeared to have been intact. Cholecystectomy was performed. The common bile duct was not obstructed but the cystic duct contained gelatinous material, which was flushed. Liver biopsy samples were obtained for histologic examination, and biopsy samples of liver and gallbladder were obtained for aerobic and anaerobic bacterial culture. The dog recovered from surgery without complication.

Histologic examination revealed that the gallbladder had extensive submucosal and luminal hemorrhage, consistent with hemocholecyst, a term which may also apply to a cyst containing blood and bile **(Figure 4)**. In addition, there were multiple dark microscopic choleliths, most abundant in the gallbladder neck. The gallbladder wall was diffusely thin, with extensive separation of the mucosal surface over 80% to 90% of the examined sections. The

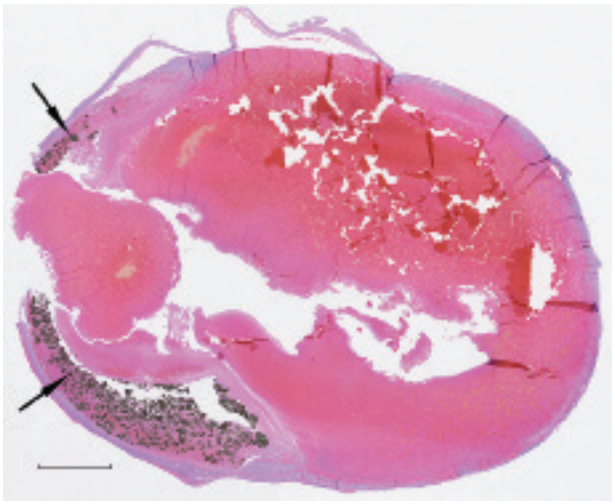

Figure 4—Photomicrograph of a transverse section of the gallbladder from the dog described in Figure 1 showing extensive submucosal and luminal hemorrhage. Also, multiple dark microscopic choleliths (black arrows) are in the neck of the gallbladder. H&E stain; bar = 3 mm.

remaining wall of the gallbladder had transmural multifocal to coalescing areas of changes that varied from necrosis and hemorrhage to fibrosis, and there were occasional sites of lymphocytic to plasmacytic inflammation within the lamina propria and on the peritoneal surface, consistent with peritonitis. The lesions within the neck of the gallbladder were suggestive of cystic mucinous hyperplasia, and findings for the liver were consistent with posthepatic cholestasis and mild reactive hepatitis.

Comments

In the dog of the present report, we diagnosed cholecystitis with hemocholecyst, microscopic choleliths, posthepatic cholestasis, and mild reactive hepatitis. Hemocholecysts are not well described in dogs but have been reported in dogs with gallbladder carcinoid tumors.[1] In humans, hemocholecysts are rare causes of acute abdominal pain and are reported secondary to gallbladder polyps, neoplasia, trauma, cholecystitis, anticoagulant treatment, or cholelithiasis.[2–8] In the dog of the present report, the abundance of microscopic choleliths, especially in the neck of the gallbladder, was deemed to have been the most likely reason for hemocholecyst formation; however, the possibility that these were crystalline heme secondary to hemorrhage was not ruled out. Alternatively, hemorrhage associated with gallbladder necrosis has been reported in humans[9,10] and could have been the underlying cause in the dog of the present report. The sudden onset of clinical signs and temporal changes in the ultrasonographic appearance of the gallbladder were consistent with active hemorrhage and development of an intraluminal thrombus of the gallbladder. The increased amount of immobile, echogenic content in the gallbladder on

repeated ultrasonography, compared with our initial ultrasonographic findings, could also have been consistent with the rapid formation of gallbladder sludge (a mixture of precipitated cholesterol, crystals, bile pigments and salts, and mucin).

With the development of echogenic material in the center of the gallbladder over 72 hours, we did not suspect gallbladder mucocele, which are nonmobile, generally have a peripheral mucin layer, border the gallbladder wall, and have various degrees of anechogenicity. The central portion of a mucocele can be echogenic and may be composed of inspissated bile (nonmobile precipitated content) or sludge (mobile with or without an anechoic layered supernatant). In the absence of echogenic central sludge or inspissated bile, a mucocele with central mobile anechoic bile could appear ultrasonographically as totally anechoic luminal gallbladder content but still cause clinical disease. The central echogenic material, if present in a mucocele, is not gravity-dependent, in part, because the anechoic mucus prevents it from settling along the gallbladder wall. Therefore, if no echogenic or precipitated sludge is present, a mucocele can be completely anechoic.

Over 3 days, the dog of the present report developed non–gravity-dependent, hyperechoic material in its gallbladder, consistent with hemocholecyst, which was confirmed after cholecystectomy and histologic examination. Findings for the dog in the present report underscored the importance of including hemocholecysts among the differential diagnoses for non–gravity-dependent, hyperechoic luminal material in gallbladders of dogs.

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