Bacterial cholecystitis is apparently uncommon in small animals, with published information on the condition in dogs limited to small case series and clinical reports.1–3 There are limited reports4 of hepatobiliary infections in dogs with the following subclassifications described: cholangitis, cholangiohepatitis, or both; cholecystitis; choledochitis; focal suppurative lesions; and multiple hepatic microabscesses. Culture of bile from dogs with bacterial cholecystitis has yielded Klebsiella spp, Clostridia spp, Corynebacterium spp, Bacteroides spp, Streplococcus faecalis, Peptostreptococcus anaerobius, and Escherichia coli.3,5,6 An apparent difference in susceptibility to bacterial cholecystitis between cats and dogs, with cats having an increased susceptibility, has been suggested to result from differences in pancreatic and bile duct anatomy; however, the pathogenesis of bacterial cholecystitis is poorly understood.7

Characterization, treatment, and outcome of bacterial cholecystitis and bactibilia in dogs

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Objective—To characterize historical, clinicopathologic, ultrasonographic, microbiological, surgical, and histopathologic features of bacterial cholecystitis and bactibilia in dogs and evaluate response to treatment and outcomes in these patients.

Design—Retrospective case-control study.

Animals—40 client-owned dogs (10 with bacterial cholecystitis on histologic analysis or bactibilia on cytologic examination [case dogs] and 30 without bactibilia [controls]) evaluated at a veterinary teaching hospital between 2010 and 2014.

Procedures—Signalment, history, clinicopathologic findings, ultrasonographic features, microbiological results, surgical findings, histopathologic changes, treatments, and outcomes of case dogs were derived from medical records and summarized. Demographic and clinicopathologic data and ultrasonographic findings were compared between case and control dogs. Relationships among prior antimicrobial treatment, sediment formation in the gallbladder, presence of immobile biliary sludge, and presence of bactibilia or bacterial cholecystitis were assessed.

Results—No finding was pathognomonic for bactibilia or bacterial cholecystitis in dogs. Case dogs were significantly more likely to have immobile biliary sludge and had a greater degree of biliary sediment formation than did control dogs. All case dogs for which gallbladders were examined histologically (8/10) had bacterial cholecystitis. Five of 10 case dogs were Dachshunds. Medical or surgical treatment resulted in good outcomes.

Conclusions and Clinical Relevance—Bactibilia and bacterial cholecystitis were important differential diagnoses in dogs with signs referable to biliary tract disease. Dachshunds were overrepresented, which may suggest a breed predisposition. Cytologic evaluation of bile should be considered in the routine assessment of dogs with hepatobiliary disease if immobile biliary sludge is present. (J Am Vet Med Assoc 2015;246:982–989)

Materials and Methods

Case selection—A retrospective search of medical records was performed for dogs evaluated at the Oregon State University College of Veterinary Medicine from January 1, 2010, to February 15, 2014. Computerized medical records were searched to identify dogs

ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>ALP</td>
<td>Alkaline phosphatase</td>
</tr>
<tr>
<td>ALT</td>
<td>Alanine transaminase</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>GGT</td>
<td>γ-Glutamyltranspeptidase</td>
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<tr>
<td>IBD</td>
<td>Inflammatory bowel disease</td>
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The purpose of the study reported here was to retrospectively examine cases of bacterial cholecystitis and bactibilia in dogs to gather information regarding signalment, clinical signs, clinicopathologic findings, ultrasonographic changes of the biliary system, bacterial culture results, surgical findings, histopathologic changes, treatment response, and outcome. Through characterization of these features, we sought to identify clinicopathologic or ultrasonographic imaging findings that can increase clinical suspicion for bacterial cholecystitis or bactibilia in dogs.
with bacterial cholecystitis or bactibilia (case dogs) and dogs that underwent cholecystostentesis during assessment of apparent hepatobiliary disease in which bacterial cholecystitis or bactibilia was not detected (control dogs). The criteria for case definition allowed inclusion of dogs in which cytologic evidence of bactibilia was noted but selection of medical rather than surgical management precluded the collection of gallbladder tissue to allow a histologic diagnosis.

Bacterial cholecystitis was diagnosed by histologic examination of the gallbladder in canine patients with bile cytologic evaluation demonstrating bactibilia and clinicopathologic evidence of cholestatis: elevated ALP activity, GGT activity, or total bilirubin concentration. Bactibilia was diagnosed on cytologic evaluation in cases where medical management precluded histologic examination of the gallbladder. Each control dog had cholecystostentesis performed during evaluation for hepatobiliary disease. The bile in all control dogs had been assessed as cytologically normal, with no growth on aerobic or anaerobic bacterial culture.

Medical records review—Information collected from the medical records included signalment, clinical signs, history (including antimicrobial administration or corticosteroid administration within the 3 months prior to diagnosis), CBC and serum biochemical analysis results, findings on ultrasonography of the gallbladder, gross surgical findings, results of histologic analysis of the gallbladder (if applicable), cytologic findings, presence of preoperative or intraoperative gallbladder rupture, evidence of extrahepatic biliary tract obstruction, treatments, and outcome (determined by status 14 days after surgery or hospital discharge). Follow-up for dogs that survived beyond this time was completed by telephone contact with the owner.

Ultrasoundography and ultrasound-guided sample collection—Board-certified radiologists performed all ultrasonographic examinations. Dogs were sedated with dexmedetomidine (5 µg/kg [2.3 µg/lb], IV) and butorphanol (0.1 mg/kg [0.045 mg/lb], IV) or anesthetized with propofol (4 to 6 mg/kg [1.8 to 2.7 mg/lb], IV) for cholecystostentesis at the discretion of the attending clinician. Percutaneous ultrasound-guided cholecystostentesis was performed with a 22-gauge, 3.81-cm needle directly connected to a 6- or 12-mL syringe. The needle was positioned and a bile sample was aspirated by the radiologist following routine preparation and disinfection of the skin. A transhepatic approach through the right medial liver lobe was used.

All ultrasonographic images were retrospectively rerevaluated by 1 board-certified radiologist (SN) who was blinded to the results of the other diagnostic tests. Ultrasonographic characteristics were recorded, including gallbladder wall thickness, gallbladder content, bile duct dilation, and other abnormal or normal findings. The presence of adjacent hyperechoic fat in conjunction with focal effusion adjacent to the gallbladder was considered evidence of gallbladder wall rupture. Gallbladder wall thickness was measured at 2 sites on opposing sides of the organ, with measurements ≥3 mm considered abnormal. Gallbladder contents were evaluated for the presence of echogenic material and graded as follows: no sediment (0) or mild (1+), moderate (2+), or severe (3+) amount of sediment, as previously described. Gallbladder contents were further characterized as mobile or immobile as previously described.

Aerobic bacterial culture and antimicrobial susceptibility testing and anaerobic bacterial culture—Bile samples for all case and control dogs were collected for aerobic bacterial culture and antimicrobial susceptibility testing and anaerobic bacterial culture. Samples were submitted to the Oregon State University Veterinary Diagnostic Laboratory ≤1 hour after collection in a sterile tube containing a reduced transport medium. Specimens underwent aerobic bacterial culture on trypticase soy agar supplemented with 5% sheep blood and MacConkey agar with selenite broth enrichment in 5% CO₂ at 35°C. For anaerobic bacterial culture, specimens were inoculated on preduced anaerobically sterilized media. Anaerobic bacterial cultures were processed and incubated in an anaerobic chamber. All culture plates were examined for growth daily for 3 days. Growth was subjectively scored as growth in broth only as follows: 1+ = 1% to 25%, 2+ = 26% to 50%, 3+ = 51% to 75%, and 4+ = 76% to 100%. Bacterial isolates were identified by means of standard identification procedures. Anti-microbial susceptibility testing for aerobic isolates was performed via the Kirby-Bauer method. Interpretations of susceptible, intermediate, or resistant isolates were made according to breakpoints assigned by the Clinical Laboratory Standards Institute.

Surgical management—An exploratory ventral midline celiotomy was performed in surgically managed case dogs to resect and acquire additional samples from the gallbladder and bile, respectively. Hepatic and full-thickness intestinal biopsy specimens were obtained at the discretion of the attending surgeon. Perioperative IV antimicrobial administration was withheld until completion of intraoperative tissue collection. An exploratory ventral midline celiotomy was performed when warranted in surgically managed control dogs, and full-thickness intestinal biopsy specimens were obtained. All surgical procedures were performed or directly supervised by 1 board-certified small animal surgeon (MM).

Statistical analysis—Data were analyzed with a commercial biomedical statistics package. Data from continuous variables were assessed for normality with the D’Agostino-Pearson omnibus test. Data consistent with a normal distribution (ALP activity) were compared between cases and controls with a 2-tailed, unpaired Student t test. Data not consistent with a normal distribution were compared with the Mann-Whitney U test (age, weight, ALT activity, GGT activity, total bilirubin concentration, and gallbladder filling grade). Contingency table analyses were used to assess relationships among binary variables of interest (presence of immobile biliary sludge, presence of abdominal pain, presence of neutrophils, and presence of immature neutrophils). Sensitivity, specificity, and likelihood ratios (relative risk) for the detection of immobile biliary sludge as a potential indicator of bactibilia were calculated along with 95% CIs. For all statistical analyses, values of P ≤0.05 were considered significant.
**Results**

**Dogs**—Of 40 dogs that underwent cholecystocentesis within the study period, 10 were identified as case dogs (6 with bacterial cholecystitis and 4 with bactibilia) and 30 were identified as controls. Examples of cytologic preparations from case and control dogs are provided (Figure 1). One dog, in which recent antimicrobial treatment may have inhibited bacterial culture but that had marked bactibilia noted cytologically, was included in the analysis as a case dog.

Median ages of the case and control dogs were 11 years (range, 6 to 15 years) and 9 years (range, 4 to 16 years), respectively. Median body weight of case dogs was 16.8 kg (37 lb; range, 5.6 to 36.9 kg [12.3 to 81.2 lb]), and that of control dogs was 14.6 kg (32.1 lb; range, 4.5 to 32.5 kg [9.9 to 71.5 lb]). Age and weight did not differ significantly between groups. There were 6 spayed females, 1 sexually intact female, 2 neutered males, and 1 sexually intact male in the case group and 15 spayed females, 14 neutered males, and 1 sexually intact female in the control group. Breeds of case dogs included Dachshund (n = 5), Flat-Coated Retriever (1), Weimaraner (1), Beagle (1), Gordon Setter (1), and mixed (1). Breeds of control dogs included Labrador Retriever (n = 6), Australian Cattle Dog (3), Miniature Poodle (3), Chihuahua (2), Jack Russell Terrier (2), Lhasa Apso (2), Pomeranian (2), and American Staffordshire Terrier, Australian Shepherd, Belgian Sheepdog, Border Collie, Greyhound, Havanese, Pug,

![Figure 1](link)
High ALT activity, ALP activity, GGT activity, and total bilirubin concentration (each found in ≥ 5 dogs). No significant differences were found between the case and control groups for any measured clinicopathologic variables, including ALP activity, ALT activity, GGT activity, total bilirubin concentration, presence of neutrophilia, or presence of immature neutrophils. The number of dogs with signs of abdominal pain also did not differ between the case (1) and control (3) groups. There was a significant (Fishers exact test; P < 0.001) relationship between a history of prior antimicrobial administration and the presence of bactibilia (OR, 130.7; 95% CI, 6.07 to 2,815, Fisher exact test; P = 0.027 [relative risk, 2.8; 95% CI, 1.29 to 6.06]).

Ultrasoundographic findings—The amount of echogenic material within the gallbladder was graded as severe in 3 of 10 case dogs and moderate in 7 of 10 case dogs (Figure 2). One dog had ultrasonographic evidence of a choledolith. There was no ultrasonographic evidence of gallbladder mucocele development in any dog. The median luminal filling grade for echogenic material observed in case dogs was 2+ (moderate; range, 2+ to 3+), and that of control dogs was 1+ (mild; range, 0 to 3+); this difference was significant (Mann–Whitney U test; P = 0.021). Immobile biliary sludge was identified in 7 case dogs and no control dogs; this difference was significant (Fisher exact test; P < 0.001). Detection of immobile biliary sludge had 70% sensitivity (95% CI, 34% to 93%) with 100% specificity (95% CI, 88% to 100%) for diagnosis of bactibilia. The relative risk for diagnosis of bactibilia with immobile sludge was 11 (95% CI, 3.73 to 32.37). No complications during cholecystocentesis were reported for the case or control groups.

Culture and antimicrobial susceptibility testing—Aerobic bacterial isolates from case dogs included an Enterobacter sp (n = 1 dog), Enterococcus spp (5), and Escherichia coli (6), and anaerobic bacterial isolates included Bacteroides fragilis (1) and Clostridium perfringens (1). Among aerobic bacterial isolates, all Enterococcus spp and 1 E coli isolate were multidrug resistant (ie, resistant to ≥ 3 antimicrobial drug classes14), and these isolates were all obtained from 5 of the 7 dogs that had received prior antimicrobial treatment. One sample from a dog

<table>
<thead>
<tr>
<th>Variable</th>
<th>Reference range</th>
<th>Case dogs (Median range)</th>
<th>Control dogs (Median range)</th>
<th>No. of abnormal results</th>
<th>No. of abnormal results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>150–275</td>
<td>203 (67–844)</td>
<td>160 (227–326)</td>
<td>3*</td>
<td>16*</td>
</tr>
<tr>
<td>Total bilirubin (mg/dL)</td>
<td>0.0–0.05</td>
<td>2.5 (0.05–8.3)</td>
<td>2.5 (0.05–8.3)</td>
<td>5*</td>
<td>13 (0.05–6.1)</td>
</tr>
<tr>
<td>ALP (U/L)</td>
<td>10–94</td>
<td>2,850 (61–6,529)</td>
<td>2,652 (226–8,500)</td>
<td>9*</td>
<td>2*</td>
</tr>
<tr>
<td>ALT (U/L)</td>
<td>5–66</td>
<td>1,605 (146–1,605)</td>
<td>778 (41–2,500)</td>
<td>10*</td>
<td>26*</td>
</tr>
<tr>
<td>GGT (U/L)</td>
<td>2–10</td>
<td>95 (3–130)</td>
<td>40 (0–229)</td>
<td>6*</td>
<td>22*</td>
</tr>
<tr>
<td>Total protein (g/dL)</td>
<td>2.3–4.0</td>
<td>6.1 (4.2–7.9)</td>
<td>6.0 (4.7–7.2)</td>
<td>21</td>
<td>0</td>
</tr>
<tr>
<td>Neutrophil count (neutrophils/µL)</td>
<td>3,000–11,400</td>
<td>12,000 (220–23,073)</td>
<td>9,706 (6,871–25,624)</td>
<td>21</td>
<td>11*</td>
</tr>
<tr>
<td>Lymphocyte count (lymphocytes/µL)</td>
<td>1,000–4,800</td>
<td>1,100 (248–2,083)</td>
<td>2,000 (685–4,282)</td>
<td>11</td>
<td>2*</td>
</tr>
<tr>
<td>Monocyte count (monocytes/µL)</td>
<td>545 (177–1,489)</td>
<td>545 (177–1,489)</td>
<td>643 (219–1,875)</td>
<td>3*</td>
<td>2*</td>
</tr>
<tr>
<td>Band neutrophil count (band neutrophils/µL)</td>
<td>0–300</td>
<td>19 (1–133)</td>
<td>164 (1–2,130)</td>
<td>1*</td>
<td>2*</td>
</tr>
<tr>
<td>Platelet count (X 10³ platelets/µL)</td>
<td>300–1,350</td>
<td>1,300 (200–2,939)</td>
<td>262 (99–528)</td>
<td>21</td>
<td>41*</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>37–55</td>
<td>40 (32–51)</td>
<td>38 (15–53)</td>
<td>21</td>
<td>51*</td>
</tr>
</tbody>
</table>

*Includes values above the upper limit of the reference range. †Includes values below the lower limit of the reference range.

Table 1—Selected serum biochemical and hematologic variables in dogs with bacterial cholecystitis or bactibilia (case dogs; n = 10) and dogs that underwent cholecystocentesis during assessment for hepatobiliary disease in which bactibilia was not detected (controls; 30).
receiving antimicrobial treatment had cytologic evidence of bactibilia without growth in culture. One bacterial species (E. coli) was isolated from 4 case dogs, with 2 bacterial species isolated in the remaining 6. Results of bacteriologic culture of hepatic tissue did not agree with those for bile in 4 of 6 case dogs, with conflicting isolate identification in 2 and negative tissue culture results in 2. Conflicting culture results occurred in cases where multiple organisms were cultured from bile and a single organism was cultured from hepatic tissue.

Medical management—Criteria that influenced the decision to pursue medical rather than surgical management included a lack of clinical signs, client refusal of recommendation to pursue surgery, and client financial constraints. Case dogs treated medically (4/10) were prescribed ursodeoxycholic acid (10 to 15 mg/kg [4.5 to 6.8 mg/lb], PO, q 24 h) and extended (6 to 8 weeks) periods of antimicrobial treatment prescribed according to the aerobic and anaerobic bacterial culture and antimicrobial susceptibility test results for bile and ability of the antimicrobial to concentrate in bile. These dogs (with bactibilia) were returned monthly for sequential serum biochemical analysis and cholecystocentesis with cytologic evaluation, aerobic bacterial culture and antimicrobial susceptibility testing, and anaerobic bacterial culture. Sequential cholecystocentesis was performed until bile was cytologically normal and negative for bacterial growth on culture. The median number of bile samples acquired for each medically managed case dog during antimicrobial treatment was 4 (range, 3 to 8). Median time to completion of treatment was 5 months (range, 4 to 9 months). Two of the 4 dogs had no clinical signs for the duration of treatment. The remaining 2 dogs had resolution of clinical signs (hyporexia and lethargy) prior to resolution of bactibilia. All medically managed case dogs had improvement or resolution of the elevated clinico-pathological variables of ALP, ALT, and total bilirubin concentration over the course of treatment.

Surgical management—Criteria influencing decisions to pursue surgical management of case dogs included failure of medical management (n = 2 dogs), evidence of extrahepatic biliary obstruction (2), and client desire for more definitive treatment (2). Five of 6 dogs with cholecystitis had clinical signs prior to surgery, and 4 of the 5 had resolution of clinical signs following surgical management. The 6 dogs were surgically managed with antegrade and retrograde flushing of the common bile duct to ensure patency and decrease bacterial burdens. Five dogs underwent cholecystectomy, and 1 underwent surgical decompression via cholecystotomy to obtain gallbladder wall specimens and contents for bacteriologic culture by flushing of the gallbladder without cholecystectomy. This 1 dog had a sterile 5F red rubber urethral catheter secured with a single 2-0 polydioxanone suture placed to act as a short-term biliary stent. None of these dogs had gross evidence of bile leakage into the peritoneal cavity at the time of surgery. All surgically treated dogs survived the procedure with no reported anesthetic complications and were discharged from the hospital 2 to 4 days after surgery.

Histologic findings—Histologic changes were found in liver samples from all 6 surgically treated dogs with bacterial cholecystitis. Findings included vascular degeneration (n = 1 sample), lymphohistiocytic hepatitis (1), neutrophilic hepatitis (2), and lymphoplasmacytic neutrophilic hepatitis (2). Cholecystitis was confirmed histologically in gallbladder samples from all 6 dogs and reported as proliferative lymphoplasmacytic cholecystitis or proliferative lymphoplasmacytic-histiocytic cholecystitis (Figure 3). Small intestinal histologic changes were noted in 3 of 4 samples from 4 dogs with bacterial cholecystitis and described as severe diffuse eosinophilic-lymphoplasmacytic enteritis, severe diffuse eosinophilic-lymphoplasmacytic-histiocytic enteritis, and moderate lymphoplasmacytic enteritis. Intestinal histologic changes described as moderate lymphoplasmacytic enteritis were identified in 1 of 3 control dogs that underwent surgical treatment and sample collection.

Outcome—One dog that had multidrug-resistant E. coli cultured from the bile at surgery and from peritoneal fluid at follow-up died of sepsis 7 days after surgery despite antimicrobial treatment. The remaining 5 surgically treated case dogs had a median survival time of 3 years (range, 21 days to 3 years after surgery), and follow-up time ranged from 21 days to 3 years. There
were no deaths among the 4 dogs treated medically, with follow-up times ranging from 4 to 12 months. Two of 30 control dogs were euthanized ≤ 14 days after diagnosis (one because of disseminated intravascular coagulation associated with idiopathic portal vein thrombosis, and the other because of liver failure–associated chronic hepatitis). The remaining control dogs had ≥ 1 condition including chronic hepatopathy (n = 10), chronic hepatitis (4), steroid hepatopathy (3), hepatocellular carcinoma (1), cholangitis (1), pancreatitis (3), primary immune-mediated hemolytic anemia (2), idiopathic portal vein thrombosis (1), enteric foreign body (1), splenic sarcoma (1), and fever of unknown origin (1). All surviving control dogs were alive at last follow-up (range, 1 to 36 months).

**Discussion**

Bacterial cholecystitis and bactibilia in dogs are poorly documented and poorly understood conditions. The purpose of the present study was to characterize historical, clinicopathologic, ultrasonographic, microbiological, surgical, and histopathologic features of bacterial cholecystitis or bactibilia in dogs and evaluate response to treatment and outcomes in these patients. Although no clinicopathologic variables specific to bacterial cholecystitis or bactibilia were identified, immobile biliary sludge was 70% sensitive and 100% specific for the diagnosis of bactibilia in this study. There was also histopathologic evidence of concurrent IBD in 3 of 4 case dogs from which intestinal biopsy specimens were acquired. Multidrug-resistant bacterial infections were identified in 5 case dogs.

Cholecystocentesis was performed in 40 dogs during the study period. Indications for this procedure included high liver enzyme activities, an ultrasonographically abnormal gallbladder (gallbladder wall thickening, nondependent sedimentation, or excessive sedimentation), and fever of unknown origin. The identification of bacterial cholecystitis or bactibilia in 10 of the 40 dogs evaluated suggests that this may be more common than previously thought, and analysis of bile samples should be considered more frequently in patients undergoing evaluation for suspected hepatobiliary disease.

Previous studies have identified Shetland Sheepdogs and American Cocker Spaniels as predisposed to biliary disease, specifically biliary mucoceles. Findings of the present study suggested that Dachshunds may be predisposed to bacterial cholecystitis or bactibilia. This has not previously been reported to our knowledge. Given the small numbers of affected dogs in our study, these data should be interpreted with caution. However, it is interesting to note that Dachshunds were markedly overrepresented among the case dogs, yet were not overrepresented in the patient population undergoing abdominal ultrasonographic examination at our facility and are not a particularly common breed in our practice area.

Previous antimicrobial administration was associated with significantly increased odds of bactibilia, and all 5 dogs with multidrug-resistant bacterial isolates had previously received antimicrobial treatment. Because of the retrospective nature of this study, it was impossible to determine whether animals previously administered antimicrobials were more systemically ill or whether previous antimicrobial treatment was a risk factor for development of bacterial cholecystitis or bactibilia. Additional prospective studies would be necessary to investigate a relationship among these factors. However, the results reported here suggested that antimicrobials should be administered cautiously if samples for bacterial culture have not been acquired from dogs with clinical signs attributable to hepatobiliary disease.

Previous studies have not identified factors associated with bacterial cholecystitis. There were no clinicopathologic or ultrasonographic abnormalities...
pathognomonic for bacterial cholecystitis identified in this study, which is consistent with previous reports. However, immobile biliary sludge was identified in 7 of 10 case dogs. The presence of biliary sludge was also associated with biliary stasis in a previous study. The combination of biliary ultrasonography and ultrasound-guided collection of bile samples for cytologic examination and bacteriologic culture is recommended to adequately screen for bactibilia in dogs undergoing assessment of hepatobiliary disease, particularly if immobile biliary sludge is identified.

The data reported here support prior observations from other studies that the predominant bacteria isolated from the hepatobiliary system of dogs are of enteric origin. Furthermore, the findings reported here highlight the importance of performing both aerobic and anaerobic bacterial cultures of biliary samples. The fact that 5 of the 7 case dogs in this study that received prior antimicrobial treatment had multidrug-resistant Enterococcus isolated is concerning. A high incidence of antimicrobial drug resistance has also been reported in bacteria isolated from the airways of dogs with pneumonia that received antimicrobials prior to bacteriologic culture. However, our results must be interpreted with caution, considering it is possible that dogs with multidrug-resistant infections may have received antimicrobials prior to referral or diagnosis because they had more severe clinical signs.

Repeated bacteriologic culture of bile to monitor the response to medical management identified several important findings in these patients. A good clinical response to treatment was insufficient to confirm resolution of bactibilia because 2 of 4 affected dogs managed medically did not have clinical signs. Additionally, the remaining 2 medically managed case dogs responded well clinically, with resolution of clinical signs of inappetence and lethargy and improvement in clinicopathologic variables, but these improvements were noted to occur before the resolution of bactibilia. All medically managed dogs did well clinically, but because of the low number of dogs in this group, specific conclusions cannot be drawn. Further studies evaluating the efficacy of medical management of bactibilia in larger groups of dogs are needed.

Surgical management of bacterial cholecystitis achieved a reduction in bacterial burden, typically via cholecystectomy (5/6 dogs), with (1) or without (5) placement of a short-term biliary stent as dictated by patency of the common bile duct and duodenal papilla. The 1 postoperative death, secondary to sepsis of a short-term biliary stent, occurred in a dog that had undergone prolonged medical management prior to surgery. The immediate postoperative mortality rate in this study (1/6) was consistent with that in previous reports of 22% to 40% after cholecystectomy for biliary mucoceles. A second dog was euthanized after the immediate postoperative period because of persistent regurgitation. It has been shown in other studies that dogs surviving the immediate postoperative period after cholecystectomy have an excellent prognosis, consistent with our findings.

Bacterial cholecystitis is commonly associated with hepatitis, accounting for the frequent identification of hepatitis in dogs with bacterial cholecystitis (6/6) in our study population. Because of the limited number of hepatic biopsy specimens, it was not possible to draw any conclusions; however, comparison of hepatic biopsy specimens from dogs with bacterial cholecystitis to samples from dogs with other forms of hepatobiliary disease should be investigated. The routine submission of hepatic biopsies for aerobic and anaerobic bacterial culture should be considered to rule out concurrent bacterial hepatitis in dogs with bacterial cholecystitis. Additionally, submission of gallbladder wall samples for aerobic and anaerobic bacterial culture should be considered because culture of urinary bladder wall samples has been shown to increase diagnostic yield, compared with examination of urine alone, and it is unknown whether testing of gallbladder samples would also provide more information. The use of pooled samples of gallbladder wall, gallbladder content, and hepatic tissue for aerobic and anaerobic bacterial culture warrants prospective examination; although this method would have an advantage in cost savings, it would be expected to preclude determination of the source of infection.

An intriguing finding in the present study was the identification of evidence of IBD in 3 of 4 dogs with bacterial cholecystitis from which full-thickness small intestinal biopsy specimens were obtained. This was also observed in 1 of 3 control dogs for which histologic examination of small intestine samples was performed. An association between IBD and cholangitis has been reported in cats, but a similar association has not been documented in dogs. Further studies are needed to determine whether an association exists between IBD and bacterial cholecystitis in dogs. It has been postulated by other authors that IBD may predispose cats to cholangitis as a result of dysmotility causing reflux of enteric bacteria into the common bile duct. The ultrasonographic identification of immobile biliary sludge in 7 of 10 patients in our study suggested the presence of gallbladder dysmotility in these patients. It is possible, that if dysmotility is present, it could predispose such patients to bactibilia.

The present study had several limitations that must be recognized, including limited case numbers due to the relative rarity of the condition. The retrospective nature of studies of this type generates selection bias, precluding direct comparison of medical and surgical treatments. Additionally, variations in clinician-dictated case management may have been present. Cytologic evidence of bactibilia is not considered equivalent to a diagnosis of bacterial cholecystitis, which requires histologic evaluation. Subclinical bactibilia does occur in a small number of dogs, even though bile is considered sterile, and whether these dogs progress to have clinical biliary disease is unknown. Furthermore, the possibility of sample contamination cannot be definitively excluded. Larger, prospective, multicenter studies are needed to further characterize this disease and discern differences in outcomes between treatment methods.

Our results supported that bacterial cholecystitis and bactibilia should be considered as a differential diagnosis in dogs with signs referable to biliary tract disease. Where none of the evaluated serum biochemical values, CBC variables, or ultrasonographic findings
were pathognomonic for bactibilia, cholecystocentesis to acquire bile for cytologic examination and bacteriologic culture should be considered in dogs with evidence of hepatobiliary disease, especially if immobile sludge is seen on ultrasonographic evaluation. Detection of immobile biliary sludge had 70% sensitivity (95% CI, 34% to 93%) with 100% specificity (95% CI, 88% to 100%) for the diagnosis of bactibilia; however, given the small case numbers, these data should be interpreted with caution, and additional large-scale studies of the diagnostic importance of the visualization of immobile sludge in dogs with hepatobiliary disease are indicated. Dachshunds were overrepresented among case dogs, and this should be investigated prospectively in other populations. Surgical cholecystectomy was an effective therapeutic strategy in this study and allowed concurrent collection of histopathologic samples of the gallbladder, liver, and gastrointestinal tract. Medical management in the form of prolonged antimicrobial and choleretic treatment may also be effective in select cases of bacterial cholecystitis, as the medically managed dogs in our study had good outcomes. Prospective evaluation of patients with suspected hepatobiliary infection is needed to identify potential risk factors for infection.

References