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CHAPTER 88

Intra-abdominal Infections

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Overview of Bacterial Intra-abdominal and Hepatobiliary Infections

Causes: Most commonly *Escherichia coli, Enterococcus* spp., and anaerobes; less often other gram-negative aerobes, *Streptococcus,* and *Staphylococcus* spp.

Mode of Transmission: Opportunistic invasion by commensal bacteria of the gastrointestinal and urogenital tracts

Major Clinical Signs: Inappetence, fever, vomiting, diarrhea, abdominal pain

Differential Diagnoses: Nonbacterial causes of pancreatitis, peritoneal effusion, hepatitis, or extrahepatic biliary tract obstruction (especially neoplasia and inflammatory disorders but also viral, protozoal, or fungal infections)

Human Health Significance: Enteric bacteria involved may be multidrug-resistant organisms that have the potential to colonize humans.

INFECTIOUS GASTROENTERITIS

Gastroenteritis in dogs and cats may be caused by an enormous array of microorganisms that include viral, bacterial, fungal, and protozoal pathogens (Table 88-1[1-5]). Other parasites (such as nematodes) should also be considered on the differential diagnosis list. Enteropathogenic bacteria cause diarrhea by adhesion to or destruction of enterocytes, secretion of a variety of potent enterotoxins, and stimulation of the host inflammatory response. Many of these microbes can be detected in the feces of apparently healthy animals as well as the feces of those with clinical signs of diarrhea. Therefore, it may be difficult to ascertain the role that these organisms play in disease in a single patient. Clinical signs of diarrhea may be more likely to occur when multiple organisms are present simultaneously ("polyparasitism"). In addition, other host factors (such as nutritional status or age) and bacterial virulence factors influence whether clinical signs develop. When outbreaks occur in dog and cat populations, collection of specimens from multiple affected and in-contact animals may be useful to determine the significance of one or more organisms involved. Use of antibiotics to treat bacterial diarrhea should be reserved for animals with systemic signs of illness such as fever, lethargy, and leukocytosis on the CBC.[6] Other infections are self-limiting, and the mainstay of treatment is proper fluid therapy and supportive care.

The reader is referred to other chapters in this book for detailed information on pathogenesis, clinical signs, diagnosis, and treatment for the organisms that are most commonly involved. References are also provided in Table 88-1 for information on pathogens that are uncommon or rarely identified or that have uncertain pathogenicity.

BACTERIAL PERITONITIS

Etiology and Epidemiology

Peritonitis may be focal or diffuse and can also be classified as *primary* or *secondary peritonitis*. Primary peritonitis is peritonitis that has no identifiable underlying cause. In secondary peritonitis, a reason for bacterial leakage into the abdomen can be identified.

In humans, primary peritonitis has also been referred to as *spontaneous bacterial peritonitis* and most often complicates development of ascites (e.g., secondary to cirrhosis, hepatitis, or congestive heart failure).[7,8] In contrast, conditions that predispose to ascites are rarely present in the history of dogs and cats with primary peritonitis. Some primary peritonitis in dogs or cats may result from hematogenous spread of bacteria to the peritoneum or bacterial translocation from the gastrointestinal tract.[7]

Secondary peritonitis is the most common form of peritonitis in dogs and cats and occurs when bacteria are introduced into the peritoneal space as a result of gastrointestinal perforation, penetration of the abdominal wall, rupture of the genitourinary tract, rupture of intra-abdominal abscesses, gallbladder rupture, or ascending infection of the umbilicus in neonates (Table 88-2).[7,9-15] In dogs, low preoperative total protein or serum albumin concentrations are risk factors for development of bacterial peritonitis after gastrointestinal surgery.[16]

The bacterial species involved in peritonitis reflect the normal flora of the gastrointestinal tract. Mixed aerobic-anaerobic infections, with up to four different bacterial species, occur in more than 50% of affected dogs and cats.[7,10] *Escherichia coli* is the most common isolate from both dogs and cats, followed by *Enterococcus* and *Clostridium* spp.[7,10,11,14,15,17,18] Other isolates include staphylococci, streptococci, *Pseudomonas aeruginosa* or *Acinetobacter* spp., a variety of anaerobes, gram-negative Enterobacteriaceae (*Proteus, Citrobacter, Serratia, Klebsiella,* or *Enterobacter*), *Actinomyces,* or in cats, *Pasteurella multocida*. In one study, a greater proportion of dogs with primary peritonitis had gram-positive bacterial infections than dogs with secondary peritonitis.[7] Uncommonly, *Candida albicans* can be involved, especially if there is a history of antibiotic treatment (see Chapter 67).

The mean age of dogs with peritonitis is around 5 to 7 years, and the mean age of cats is 7 to 10 years, although dogs and cats of any age can be affected.[9,12,19] Age was not found

TABLE 88-1

Examples of Potential Infectious Causes of Enterocolitis in Dogs and Cats

Organism Type	Dogs	Cats
Viruses	Canine parvovirus Canine enteric coronavirus Canine distemper virus Rotaviruses Astroviruses Adenoviruses Caliciviruses	Feline panleukopenia virus Feline coronavirus Feline calicivirus FeLV FIV Rotaviruses Astroviruses Torovirus-like agent Reoviruses
Bacteria	*Salmonella* spp. *Clostridium perfringens* *Clostridium difficile* *Campylobacter* spp. *Helicobacter* spp. *Escherichia coli* *Klebsiella pneumoniae*[1] *Enterococcus* spp.[2] *Yersinia enterocolitica* *Brachyspira pilosicoli*[3] *Mycobacterium* spp. (e.g., *M. avium*) *Leptospira* spp. *Neorickettsia helminthoeca*	*Salmonella* spp. *C. perfringens* *C. difficile* *Campylobacter* spp. *Helicobacter* spp. *Escherichia coli* *Enterococcus* spp.[4] *Y. enterocolitica* *Anaerobiospirillum*[5] *Mycobacterium* spp. (e.g., *M. bovis*)
Protozoa	*Giardia* spp. *Entamoeba histolytica* *Balantidium coli* *Isospora* spp. *Hammondia heydorni* *Cryptosporidium* spp. *Leishmania* spp. *Tritrichomonas foetus*	*Giardia* spp. *E. histolytica* *Isospora* spp. *Cryptosporidium* spp. *Tritrichomonas foetus*
Fungi	*Histoplasma capsulatum* *Cryptococcus neoformans* *Blastomyces dermatitis* *Candida albicans* *Aspergillus* spp. Zygomycetes *Pythium insidiosum* *Prototheca* spp.	*Histoplasma capsulatum* *Candida albicans*
Other parasites	*Toxocara canis* and *Toxascaris leonina* (roundworms) *Ancylostoma* and *Uncinaria* spp. (hookworms) *Trichuris vulpis* (whipworms) Tapeworms (diphyllobothriidean)	*Toxocara cati* and *Toxascaris leonina* (roundworms) *Ancylostoma* and *Uncinaria* spp. (hookworms) Tapeworms (diphyllobothriidean)

TABLE 88-2

Underlying Causes of Secondary Peritonitis in Dogs

Organ System Affected	Disease Process
Gastrointestinal system	Gastrointestinal surgical site dehiscence (e.g., resection and anastomosis) Penetrating abdominal trauma Nonsteroidal or steroidal anti-inflammatory drug toxicity Foreign bodies Gastrointestinal neoplasia Eosinophilic gastroenteritis Torsion Intussusception
Genitourinary tract	Ruptured pyometra Surgical site dehiscence (e.g., ovariohysterectomy) Ruptured prostatic abscess Necrotizing bacterial cystitis
Hepatobiliary system	Gallbladder rupture (especially animals with necrotizing bacterial cholecystitis) Hepatic abscess rupture
Other abdominal organs	Abscess rupture
Umbilicus	Ascending infection in neonates

to relate to development of primary versus secondary peritonitis in dogs or cats.[7] There is no known breed or sex predisposition. In one study, three quarters of affected cats were indoor-only.[10]

Clinical Features

The most common historical signs in dogs and cats with bacterial peritonitis are lethargy, anorexia, vomiting, and diarrhea.[7,10,15] Weakness and collapse can also occur.[14] Diarrhea and vomiting may result from intestinal hypermotility or ileus or may be secondary to underlying intestinal disease.

Physical examination findings include dull mentation, fever (up to 108°F or 42°C), dehydration, mucosal pallor, abdominal pain, thin body condition, and/or abdominal enlargement and a palpable fluid wave due to ascites.[7,10,14] Tachypnea or tachycardia may be present as a result of abdominal pain. However, some affected animals, and especially cats, show none of these signs. More than one third of cats lack any evidence of abdominal pain.[10,15] Signs of septic shock may be present, such as tachycardia, tachypnea, weak pulses, and injected mucous membranes in dogs, or hypothermia and bradycardia in cats (see Chapter 86).[10,15]

Diagnosis

Diagnosis of bacterial peritonitis is based on abdominal imaging findings, cytologic examination of peritoneal fluid, and aerobic and anaerobic bacterial culture of the fluid.

Laboratory Abnormalities

Complete Blood Count and Serum Biochemical Tests

Frequent findings on the CBC in dogs and cats with bacterial peritonitis are leukocytosis due to neutrophilia, a mild to severe bandemia, and monocytosis.[7,10,12] Neutrophil toxicity is often present. Some animals are leukopenic (as low as 270 cells/μL in cats and 1000 cells/μL in dogs).[7,10,15] Anemia of inflammatory disease may be identified. Thrombocytopenia may be present in animals with septic shock and DIC. The serum biochemistry panel often shows electrolyte and acid-base abnormalities and/or hypoalbuminemia due to inflammation or third-space losses. Mild to moderate increases in liver enzyme activities, hypoglycemia, or hyperglycemia may be detected; hyperbilirubinemia can be present in cats.[7,10] Assessment of acid-base status in both dogs and cats most often reveals acidemia; hyperlactatemia is present in at least 50% of affected animals.[7,10,15]

Coagulation Profile

Dogs and cats that develop DIC may have prolongations of PT or APTT, increased plasma D-dimer or fibrin degradation product concentrations, and/or decreased plasma antithrombin concentration.

Diagnostic Imaging

Plain Radiography

Abdominal radiographs in dogs and cats with peritonitis may show a focal or diffuse loss of abdominal detail due to variable amounts of peritoneal effusion. In some animals, pneumoperitoneum is identified secondary to rupture of an abdominal viscus or penetrating abdominal trauma. Evidence of gastrointestinal obstruction or intestinal mass lesions may also be present.[10,14] In tachypneic animals, thoracic radiographs should be considered to determine whether a pulmonary problem (such as acute respiratory distress syndrome or pulmonary thromboembolism) is contributing to respiratory distress.

Sonographic Findings

Findings on abdominal ultrasound examination include focal or diffuse hyperechogenicity of the mesentery and the presence of ascites fluid (Figure 88-1). The latter may have an echogenic appearance if it is an exudate. Evidence of underlying disease may be present, such as intestinal mass lesions or foreign bodies. The presence of pneumoperitoneum; dilated, air-filled bowel loops; or severe abdominal pain may interfere with complete ultrasound examination of some patients. CT examination is preferred to ultrasound for evaluation of human patients with bacterial peritonitis,[7,8] but its usefulness for diagnosis and treatment of peritonitis in dogs and cats requires further investigation.

Microbiologic Tests

Cytologic Examination

Specimens for cytologic examination from animals with peritonitis may be collected by blind or ultrasound-guided abdominocentesis or diagnostic peritoneal lavage (DPL). With the increased availability and sensitivity of ultrasound to detect and guide collection of small quantities of undiluted intra-abdominal fluid, DPL is now uncommonly performed in the author's practice. Peritoneal fluid from dogs and cats with bacterial peritonitis is typically a modified transudate or exudate, although fluid from dogs with primary peritonitis may be more likely to be a modified transudate or transudate.[7] Fluid analysis from most animals with peritonitis reveals a high protein concentration and an increased erythrocyte and total nucleated cell count (usually >500 cells/μL and up to 160,000 cells/μL). There is typically a predominance of neutrophils that may have a degenerate appearance, and foreign material and/or intracellular and extracellular bacteria may be seen. The presence of intracellular bacteria is generally considered diagnostic for bacterial peritonitis. Because an absence of bacteria does not rule out bacterial peritonitis, submission of fluid for culture is essential.[10,14] A blood-to-fluid glucose difference greater than 20 mg/dL was 100% sensitive and 100% specific for a diagnosis of bacterial peritonitis in one study of dogs and cats.[18]

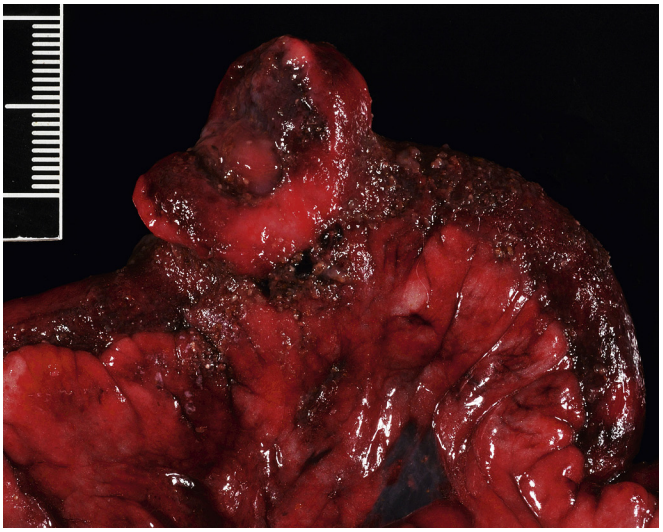

FIGURE 88-1 Necropsy image from a 7-year-old terrier mix with severe bacterial peritonitis secondary to perforated intestinal lymphoma. The dog had acute onset of diarrhea and collapse before being brought to the veterinary clinic in cardiorespiratory arrest. On ultrasound examination there was a large volume of echogenic peritoneal effusion and the mesentery was severely hyperechoic. The stomach, small intestine, and colon were fluid distended, hypomotile, and had thickened, corrugated walls with diminished blood flow. At necropsy, the omentum was discolored dark red to purple, and a segment of jejunum had an intramural mass with a central depression that communicated with the intestinal lumen. (Courtesy University of California, Davis, Veterinary Anatomic Pathology Service.)

Culture

If bacterial peritonitis is suspected, abdominal fluid specimens should be submitted for aerobic and anaerobic bacterial culture and susceptibility testing. Inoculation of blood culture bottles with ascites fluid could be considered if there is to be any delay in transport of the specimen to the laboratory.

Treatment and Prognosis

Treatment of peritonitis involves a combination of antibiotic treatment, supportive care, and surgery. The goal of surgery is to identify and correct the source of leakage and to lavage and drain the abdomen. The latter is critical for effective antibiotic penetration. Cytologic evidence of inflammation with intracellular bacteria in peritoneal fluid specimens and/or the identification of free air within the abdomen (in the absence of a history of surgery or abdominocentesis) are indications for surgical exploration.

TABLE 88-3

Suggested Empiric Antimicrobial Drug Choices for IV Use in Dogs and Cats with Bacterial Peritonitis Pending the Results of Culture and Susceptibility*

Antimicrobial Drug	Spectrum	Comments
Ampicillin-sulbactam *and* a fluoroquinolone (e.g., enrofloxacin, marbofloxacin)	Activity against gram-negative bacteria, some methicillin-resistant staphylococci, streptococci, enterococci, and most anaerobes	Replace the fluoroquinolone with an aminoglycoside (amikacin or gentamicin) if the regional prevalence of fluoroquinolone resistance is high.
Metronidazole *and* a fluoroquinolone	Activity against susceptible gram-negative and gram-positive bacteria and anaerobes	Replace the fluoroquinolone with an aminoglycoside (amikacin or gentamicin) or a third-generation cephalosporine if the regional prevalence of fluoroquinolone resistance is high.
Ticarcillin–clavulanic acid	Activity against susceptible gram-positive and gram-negative aerobes (including *Pseudomonas aeruginosa*) and some anaerobes	Not active against methicillin-resistant staphylococci
Carbapenem (meropenem or imipenem-cilastatin)	Activity against susceptible gram-positive and gram-negative aerobes and anaerobes	Not active against methicillin-resistant staphylococci. Reserve use for when multidrug-resistant gram-negative bacterial infection is suspected.
Piperacillin-tazobactam	Activity against susceptible gram-positive and gram-negative aerobes, including *Pseudomonas aeruginosa,* and some anaerobes	Not active against methicillin-resistant staphylococci. Reserve use for when multidrug-resistant gram-negative bacterial infection is suspected.

*If appropriate, reduce spectrum once the results of culture and susceptibility are available.
Dosages for IV administration (normal renal function). See Chapter 8 for precautions.
Amikacin 15-30 mg/kg q24h (dogs), 10-14 mg/kg q24h (cats)
Ampicillin-sulbactam, 20 mg/kg q6-8h (dose based on ampicillin component)
Gentamicin sulfate, 14 mg/kg q24h (dogs), 8 mg/kg q24h (cats)
Ciprofloxacin hydrochloride, 10 mg/kg q24h
Enrofloxacin, 5-20 mg/kg q24h (dogs); avoid in cats and never exceed 5 mg/kg, see important cautionary notes in Chapter 8
Imipenem-cilastatin, 5 mg/kg q6h (dilute in 100 mL sterile saline and give slowly over 30 min)
Meropenem, 25 mg/kg q8h
Piperacillin-tazobactam, 40 mg/kg q6h
Ticarcillin-clavulanate 50 mg/kg q6h

Antimicrobial Treatment

Parenteral antimicrobial drug treatment should be initiated as soon as possible after diagnosis of peritonitis, because delayed antimicrobial drug treatment in severe sepsis and septic shock may increase mortality (see Chapter 86). The initial choice of antimicrobial drugs in dogs and cats with bacterial peritonitis should include a broad-spectrum combination of antimicrobials with activity against both obligate anaerobes and facultative anaerobes (especially *E. coli* and *Enterococcus*) (Table 88-3). Subsequently, treatment should be adjusted on the basis of cytology and culture and susceptibility results. Because obligate anaerobes may not grow reliably in the laboratory and are commonly involved, continued use of an antibiotic that provides activity against anaerobes is recommended even when anaerobic cultures are negative.

Surgical Treatment

Careful surgical exploration for a site of bacterial leakage is indicated after initial stabilization with fluids, vasopressors, and antimicrobial drug treatment. The underlying cause must be surgically corrected (e.g., intestinal resection and anastomosis), and the peritoneum should be debrided and lavaged thoroughly with warm isotonic saline. The need for subsequent drainage is controversial. Drainage is generally selected when debridement and lavage cannot adequately reduce contamination. Methods of drainage include primary closure with closed suction drains, open peritoneal drainage, and, most recently, vacuum-assisted peritoneal drainage.[10,12,17,20-22] In retrospective studies, no significant differences in survival have been identified among methods, but prospective studies are required. Complications of drainage are ascending nosocomial infection, obstruction of closed suction drains by omentum, and hypoproteinemia. Evisceration and bowel desiccation have the potential to occur with open peritoneal drainage, and fluid production cannot be quantified. Dogs and cats that undergo open drainage are also more likely to require plasma or blood transfusions and may have longer hospitalization times.[17] Frequent sedation or anesthesia for bandage changes is also required.

Supportive Care

Supportive care for cats and dogs with bacterial peritonitis generally includes intensive crystalloid and colloid fluid therapy, administration of vasopressors, and enteral or parenteral nutritional support. Blood transfusions may also be required. Dogs that received early nutritional support had significantly shorter hospitalization times than those for which nutritional support was delayed.[13]

Prognosis

Survival rates of 32% to 67% have been reported in dogs with secondary peritonitis.[7,9,11,12,16,21,22] Survival rates of 44% to

70% after surgery have been reported for cats.[7,10,15] Typically hospitalization is required for 3 to 16 days (median around 5 to 6 days). When death occurs, it usually results from septic shock, DIC, or multiple organ dysfunction. Negative prognostic indicators identified in dogs with bacterial peritonitis include a diagnosis of primary as opposed to secondary peritonitis[7]; preoperative anemia, leukocytosis, or hypoproteinemia[16]; and postoperative administration of glucocorticoids.[16] Low preoperative systolic blood pressure was a negative prognostic factor in a study that included both dogs and cats.[14] Low preoperative median serum ALT activity (56 U/L for surviving cats compared with 179 U/L for non-survivors) was the only positive prognostic indicator in one study of cats with peritonitis.[10] Age, preoperative heart rate, rectal temperature, the results of other laboratory parameters, or the presence of polymicrobial infection did not affect survival. In another study, a high plasma lactate concentration was a negative prognostic indicator in cats with bacterial peritonitis.[15]

HEPATOBILIARY INFECTIONS

Etiology and Epidemiology

Hepatobiliary infections include hepatic abscesses; a variety of viral, bacterial, protozoal, or fungal infections that have spread hematogenously to the liver as part of a systemic infection; ascending infections of the biliary tree; and possibly bacterial translocation from the portal circulation. The focus of this section is bacterial infections of the biliary tree; hepatic abscesses are considered in a separate section that follows.

Bacterial infections of the hepatobiliary system occur occasionally in cats and are rare in dogs. The main mechanism thought to be involved is ascending infections of the biliary tree, although hematogenous spread and bacterial translocation from the portal system are other proposed mechanisms.[23] They have been associated with suppurative cholangitis/cholangiohepatitis, cholecystitis, choledochitis, and cholelithiasis.[24-31] *Cholangitis* is inflammation of intrahepatic bile ducts, whereas *cholangiohepatitis* is inflammation of the bile ducts that has spread to the adjacent liver parenchyma. The use of the term "cholangitis" (rather than "cholangiohepatitis") has been recommended by the WSAVA Liver Diseases and Pathology Standardization Group, because of the variable existence of hepatic involvement, so this term will be used from this point onward. In cats, cholangitis has been classified histologically as *neutrophilic cholangitis, lymphocytic cholangitis,* or *chronic cholangitis associated with liver fluke infestation.* Neutrophilic cholangitis can be further subclassified as acute or chronic depending on the presence of fibrosis, fibroplasia, or bile duct proliferation.[32] Neutrophilic cholangitis is thought to develop as a result of ascending bacterial infection from the gastrointestinal tract, which may occur secondary to inflammatory bowel disease (IBD) and/or pancreatitis (Figure 88-2). It is hypothesized that the common entry of the pancreatic and common bile ducts into the intestine in cats may predispose them to concurrent intestinal, pancreatic, and biliary disease due to pancreatic and hepatobiliary reflux in the face of IBD ("triaditis"). However, not all cats with cholangitis have histologic evidence of associated pancreatitis or IBD. Cats with acute neutrophilic cholangitis tend to be younger than those with chronic neutrophilic cholangitis.[32,33] Retrovirus infection does not appear to be a predisposing factor. The cause of lymphocytic cholangitis is unclear; currently it is thought to be an immune-mediated condition.[34] Chronic cholangitis associated with liver fluke infestation is rare and is primarily reported from regions with subtropical climates such as the southeastern United States and Hawaii, but disease can occur in non-endemic areas in traveled cats. Several different fluke species may be involved, but the most prevalent appears to be *Platynosomum concinnum*.[35]

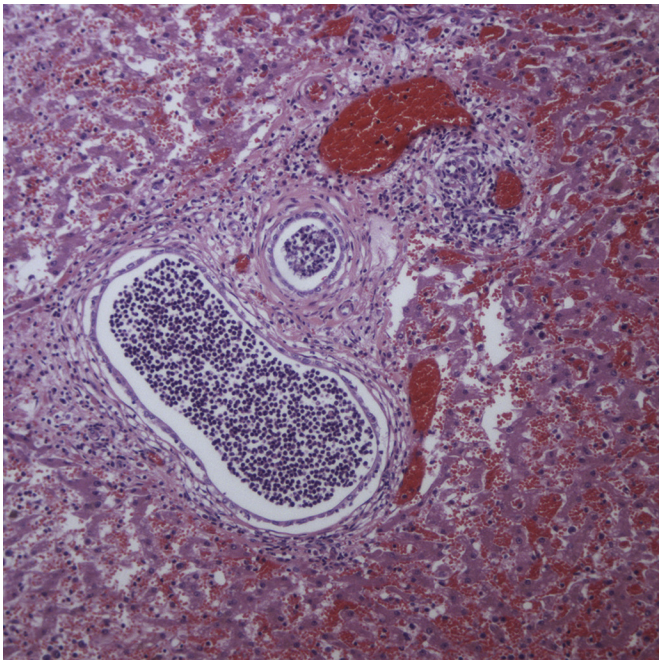

FIGURE 88-2 Histopathology showing severe, acute neutrophilic cholangitis in a 20-year-old female spayed domestic shorthair. Bile ducts are expanded by neutrophils. *Escherichia coli* was isolated from the liver in large numbers at necropsy. Concurrent diseases found at necropsy were pulmonary adenocarcinoma and moderate multifocal glomerulonephritis.

Choledochitis is inflammation of the common bile duct. *Cholecystitis* is inflammation of the gallbladder and may or may not be associated with cholangitis or choledochitis. In some instances, necrotizing and/or emphysematous cholecystitis can develop, which may lead to perforation of the gallbladder wall and septic bile peritonitis. *Cholelithiasis* is stone formation within the biliary tree. In some instances, cholelithiasis or neoplasia of the biliary tree may predispose to cholangitis and necrotizing cholecystitis.[28,29] Alternatively, it has been suggested that bacterial cholecystitis predisposes to stone formation through bacterial deconjugation of soluble bilirubin glucuronide to insoluble unconjugated bilirubin and glucuronic acid.[27] Smaller breed (dachshunds, poodles, miniature schnauzers) female dogs were predisposed to cholelithiasis in one study.[27]

Biliary cultures are more likely to be positive than hepatic cultures in both dogs and cats with hepatobiliary disease. In one large study, approximately 30% of biliary cultures were positive in dogs and cats, whereas hepatic cultures were positive in 14% of cats and 5% of dogs.[31] In cats, more than 80% of biliary cultures yielded a single bacterial species, whereas multiple bacterial species were isolated from approximately 50% of dogs. Bacterial species isolated from cats include obligate anaerobes, a variety of Enterobacteriaceae, *Enterococcus*, *Streptococcus*, or *Staphylococcus* species.[24,31,32] Dogs are most commonly infected with mixtures of *E. coli*, *Enterococcus* spp., and anaerobes, primarily *Bacteroides* and *Clostridium* species.[25-29,31]

Staphylococcus spp., other gram-negative aerobes (*Klebsiella, Enterobacter, Citrobacter,* and *Pseudomonas aeruginosa*), and *Streptococcus* spp. may also be isolated from dogs. *Salmonella* in cats and *Campylobacter* in dogs have also been associated with cholecystitis. Although rare, emphysematous cholecystitis in dogs and cats is usually associated with *Clostridium* spp. and/or *E. coli* infection.[36]

Clinical Features

Clinical Signs and Physical Examination Abnormalities

The most common clinical signs of hepatobiliary infection in both dogs and cats are lethargy, inappetence, vomiting, weight loss, and diarrhea.[24,25,30,32,33] These signs may be chronic, acute, and rarely peracute.[25,37] Peracute disease can occur with necrotizing cholecystitis and rupture of the gallbladder wall and may be associated with clinical signs of septic shock. Hypersalivation may be evident in cats with neutrophilic cholangitis.[24] Physical examination may reveal fever, lethargy, dehydration, icterus, thin body condition, and/or pain on palpation of the abdomen. Hepatomegaly may also be identified.[32] Some animals are tachycardic or tachypneic.

Diagnosis

Laboratory Abnormalities

Complete Blood Count

The CBC in dogs and cats with hepatobiliary infections may show leukocytosis due to a neutrophilia, bandemia, and sometimes monocytosis.[24-26,30,32,33,37] Some cats with cholangitis are leukopenic.[24,32] Lymphopenia and/or anemia may also be present.

Serum Biochemical Tests

Dogs with cholangitis or cholecystitis typically have increased activities of serum ALT, ALP, and GGT and hyperbilirubinemia. At least 50% of cats with cholangitis have mild to moderate increases in the activity of ALT and/or ALP (usually <1000 U/L). Increased activity of GGT may also be present. However, some cats with cholangitis have normal serum ALT, ALP, and GGT activities.[32] Hyperbilirubinemia is present in approximately two thirds of cats. Electrolyte abnormalities may be present secondary to gastrointestinal losses in animals with vomiting or diarrhea.[24,33] Hypocholesterolemia, hyperglobulinemia, and hypoalbuminemia are uncommonly identified. Hypercholesterolemia may be present in dogs with cholestasis or bile duct obstruction but is uncommonly present in cats.[26,28,30]

Urinalysis

Urinalysis in animals with hepatobiliary infections may be unremarkable or show evidence of bilirubinuria.[24,26,38]

Coagulation Profile

Coagulation abnormalities such as prolongations of PT and PTT may be present in dogs and cats with hepatobiliary infections, either secondary to impaired absorption of vitamin K secondary to biliary obstruction, or potentially as a result of liver dysfunction.[25,33]

Diagnostic Imaging

Plain Radiography

The most common finding on abdominal radiography in dogs and cats with cholecystitis and cholangitis is hepatomegaly, which is variable.[30,33] Uncommonly, single or multiple radiopaque choleliths are visible (Figure 88-3), although choleliths may also be radiolucent.[26-28] In some animals, no abnormalities are detected. Emphysematous cholecystitis is characterized by the presence of gas in the liver in the region of the gallbladder. Loss of abdominal detail may be present if gallbladder rupture and septic bile peritonitis have occurred.[25]

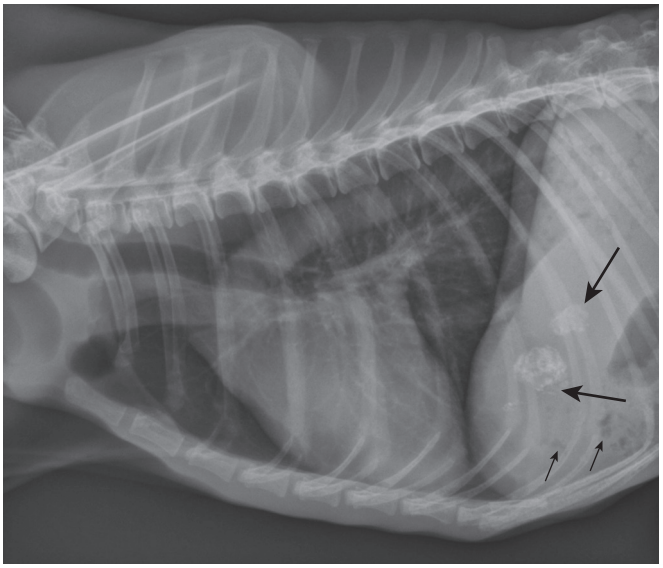

FIGURE 88-3 Lateral thoracic radiograph from a 4-year-old male neutered Chihuahua with a 3-month history of vomiting, lethargy, weight loss, and progressive icterus. Within the viewable abdomen, multiple irregular mineral opacities are present in the region of the gallbladder (*large arrows*). In this region, there are also multiple small irregular gas opacities (*small arrows*). A diagnosis of necrotizing cholecystitis and secondary bile peritonitis was made based on ultrasound and abdominal fluid analysis. Exploratory laparotomy revealed multiple choleliths, and a cholecystectomy was performed. Histopathology revealed severe, subacute to chronic, transmural suppurative and necrotizing cholecystitis.

Sonographic Findings

Abdominal ultrasound findings in dogs and cats with cholangitis are similar and include hepatomegaly, normal or abnormal hepatic echotexture with a homogenous or heterogenous increase in echogenicity, prominent portal vasculature, a thickened and hyperechogenic gallbladder wall, and sediment within the gallbladder.[24-26,30,32,39,40] The gallbladder wall may also appear irregular and contain polypoid mass lesions. Abdominal pain in the right cranial quadrant may be evident during the procedure. Choleliths are occasionally identified within the biliary tract as hyperechoic masses that shadow. Distention of the biliary tract may be evident, sometimes with tortuosity of the cystic and common bile ducts.[24,25,39] Evidence of concurrent pancreatic and/or intestinal wall disease may be present in cats.[32] Gas may be identified in the gallbladder wall of animals with emphysematous cholecystitis (Figure 88-4).

Microbiologic Tests

Cytologic Examination

Cytologic examination of bile collected by percutaneous ultrasound-guided cholecystocentesis (or intraoperative cholecystocentesis) in animals with hepatobiliary infection may reveal increased numbers of degenerate neutrophils, small and/or large mononuclear cells, and in some cases, a mixed or monomorphic bacterial population.[24] Cholecystocentesis carries some risk of

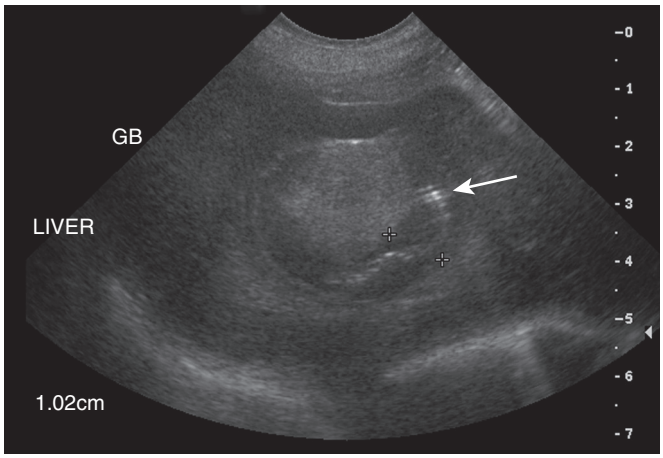

FIGURE 88-4 Abdominal ultrasound image of the gallbladder of a 6-year-old female spayed American Eskimo dog with emphysematous cholecystitis. The gallbladder wall is markedly thickened (1 cm), is diffusely hypoechoic, and the gallbladder contains homogenous sludge. Multiple hyperechoic foci were identified tracking throughout the wall *(arrow)*. The hepatic parenchyma immediately adjacent to the gallbladder was poorly characterized because of a large amount of markedly hyperechoic inflamed mesentery within the cranial abdominal region.

gallbladder leakage or rupture and should be avoided if there is evidence of gas in the gallbladder wall. As much bile should be removed as possible to minimize the chance of leakage. It is possible that percutaneous drainage of infected bile may have a therapeutic benefit, although this requires further study.

Culture

Aerobic and anaerobic bacterial culture can be performed on bile collected by ultrasound-guided or intraoperative cholecystocentesis or liver biopsies. Culture of bile is more likely to yield positive results than culture of liver biopsies. Although it is generally accepted that bile is sterile in healthy dogs and cats,[23,41] transient cytologic and microbiologic evidence of bactibilia has been identified in healthy dogs.[42] Liver tissue from healthy dogs can also harbor a variety of enteric bacterial species.[43] Thus, the results of bile culture must be interpreted in light of clinical and cytologic findings. Antimicrobial drug resistance among bacteria isolated from the bile of dogs and cats with cholecystitis and cholangitis has been described in several studies, and so culture and susceptibility testing should be performed whenever possible. When cholecystocentesis is undesirable because of concerns for gallbladder rupture (see Cytologic Examination), blood cultures may be useful, although the utility of blood culture in dogs and cats with cholecystitis has not been studied. In human patients, bile cultures are positive in 50% to 95% of patients with acute cholecystitis, and blood cultures are positive in 30% to 40% of patients.[44]

Pathologic Findings

On histopathology, neutrophilic cholangitis in cats varies in severity, chronicity, and the extent of associated hepatic parenchymal involvement.[32,38] Concurrent hepatic lipidosis may also be present. In a small percentage of affected cats, complete or partial biliary obstruction is identified as a result of inflammation, neoplasia, or cholelithiasis. Other findings in affected cats include acute or chronic pancreatitis (65% of cats in one study), inflammatory bowel disease and/or intestinal lymphoma (46% of cats). Histopathology of the gallbladder wall in both dogs and cats with cholecystitis may reveal mucosal and glandular hyperplasia and thickening and neutrophilic or lymphoplasmacytic inflammation of the gallbladder wall.[26,30,32]

Choleliths in cats are most often composed of calcium carbonate, although they also may be a mixture of cholesterol, calcium bilirubinate, and calcium carbonate.[26,28] In dogs they are often composed of calcium bilirubinate, bilirubin, or a mixture of bilirubin and cholesterol.

Treatment and Prognosis

Treatment of cholangitis and cholecystitis is usually with supportive care and antimicrobial drug administration. Dogs and cats with necrotizing or emphysematous cholecystitis require cholecystectomy. Cholecystectomy could also be considered to prevent recurrence of cholelithiasis in animals with chronic cholecystitis. Cholecystectomy, biliary stenting procedures, and/or biliary diversion surgery may be required for animals with obstructive processes such as cholelithiasis, biliary neoplasia, or obstructive cholangitis.[27,28] Because cholecystotomy or biliary diversion surgery often results in dehiscence and septic bile peritonitis if bacterial cholecystitis or choledochitis is present, cholecystectomy has been recommended for dogs with cholelithiasis as opposed to cholecystotomy.[27] When compared with other surgical biliary procedures, cholecystectomy has been associated with good clinical outcomes in dogs and cats, but any condition that requires biliary surgery carries a guarded prognosis.

Antimicrobial Drug Treatment and Supportive Care

Empirically used antibiotics should target gram-negative enteric bacterial species and anaerobes and be concentrated in bile. Suitable antibiotics include ampicillin-sulbactam or amoxicillin-clavulanic acid, or a fluoroquinolone with metronidazole, although resistance to amoxicillin-clavulanic acid or enrofloxacin has been documented among biliary isolates from dogs and cats.[30] Pradofloxacin also has a favorable spectrum of activity, because of its activity against anaerobes (see Chapter 8). In human patients, specific treatment of *Enterococcus* biliary infections when they are combined with other bacterial infections may not be necessary, since they tend to resolve when the other infections are treated.[44] Supportive treatments that could be considered include intravenous crystalloid fluids with or without colloids, nutritional support by parenteral or enteral means (with a restricted fat diet), ursodiol, *S*-adenylmethionine, antiemetics, antacids, and parenteral vitamin K supplementation.

Prognosis

The prognosis for cats with neutrophilic cholangitis is fair to good, with mean survival times longer than 1 year.[24,33] Poor outcomes may be related to the presence of underlying disease that contributes to mortality such as neoplasia.[28,33] The presence of septic bile peritonitis was associated with mortality in one study of dogs that underwent extrahepatic biliary surgery; more than half of dogs with septic bile peritonitis died.[29] In another study, 9 of 23 dogs with necrotizing cholecystitis died.[25]

HEPATIC ABSCESSES

Hepatic abscesses are uncommon in dogs and very rare in cats.[45,46] They may be associated with ascending bacterial infection of the biliary tree, translocation of portal bacteria, or hematogenous spread of bacteria to the liver, although the source of

bacteremia in some animals is unclear. Because bacteria can be found in the liver of healthy dogs, abscessation may also develop when these bacteria proliferate secondary to compromise of normal defenses (such as hepatic necrosis).[43] Affected animals are typically middle aged to older;[45,46] the mean age of affected dogs or cats is 10 years. The abscesses may be acute or chronic, single or multiple, and macroabscesses or microabscesses may form. Pancreatitis, diabetes mellitus, other hepatobiliary disease, neoplasia, colonic surgery, cholelithiasis, or chronic phenobarbital or glucocorticoid administration have been in the histories of affected dogs.[46-48] Migrating plant awn foreign bodies may also be an underlying cause.[48] Concurrent conditions in affected cats have included IBD, pancreatitis, hepatobiliary neoplasia, chronic cholecystitis, colonic surgery, or congestive heart failure.[45] Many cats have concurrent bacterial urinary tract infections.

The most common bacterial species isolated from hepatic abscesses in both dogs and cats is *E. coli*. In dogs, other species have included *K. pneumoniae, Staphylococcus,* and *Clostridium* spp. In cats, *Bacteroides, Enterococcus,* and *Streptococcus* spp. have been isolated, often in mixed infections with *E. coli*. Blood cultures are also often positive.[45,46] Clinical signs and laboratory abnormalities are similar to those for other hepatobiliary infections, except that severe illness and signs of septic shock are often present. Evidence of bleeding diatheses may be present. Marked neutrophilia or neutropenia, bandemia, toxic neutrophils, anemia, lymphopenia, and thrombocytopenia may be present on the CBC. Hypoalbuminemia and coagulation abnormalities are common.[45,46] Abdominal radiographs may be unremarkable, or they may show hepatomegaly or a hepatic mass effect, poor abdominal detail, and/or pneumoperitoneum if abscess rupture has occurred (Figure 88-5). Thoracic radiography often reveals interstitial, bronchiolar, or alveolar lung infiltrates,[44,46] which may result from associated embolic or aspiration pneumonia. Ultrasonographic examination of the abdomen reveals one or more hypoechoic, anechoic, or heteroechoic hepatic masses, with or without peritoneal effusion. In some cases, gas is evident within the masses.

Antemortem diagnosis of hepatic abscesses usually relies on cytologic examination of ultrasound-guided fine-needle aspirates of the masses, which reveals large numbers of degenerate neutrophils. Abscesses may also be discovered during exploratory laparotomy (e.g., in dogs and cats with bacterial peritonitis). Culture and susceptibility of aspirates of the liver abscesses, free peritoneal fluid, and/or blood should be performed. Effective treatment of solitary abscesses relies on appropriate antimicrobial drug treatment together with complete or partial liver lobectomy, or abscess drainage and omentalization. With the exception of microabscesses, antimicrobial drug therapy alone is often ineffective. Aggressive supportive care in an intensive care unit may be required. Initial antimicrobial drug treatment should be broad

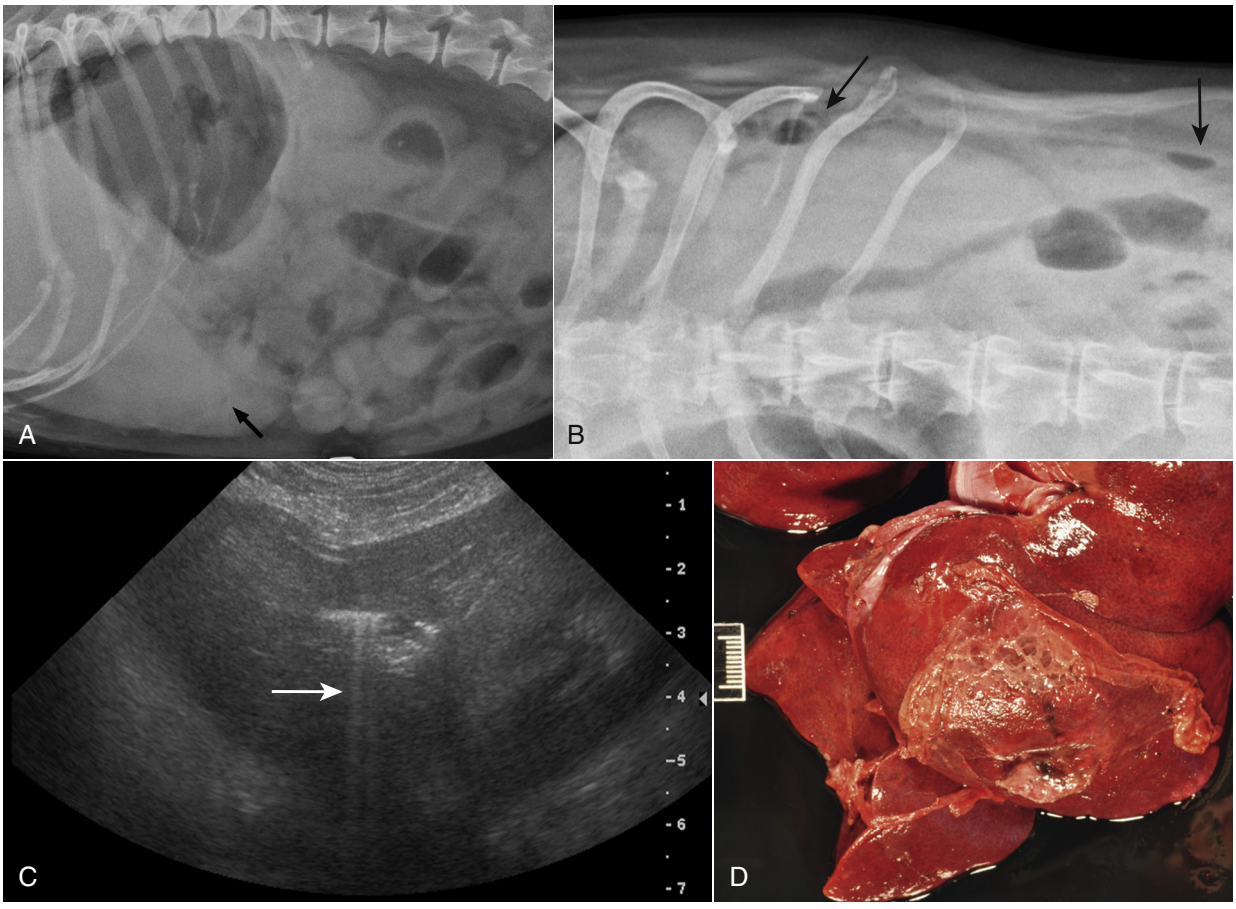

FIGURE 88-5 Ruptured hepatic abscess. **A,** Right lateral abdominal radiograph from a 15-year-old male neutered dachshund with acute onset of vomiting and diarrhea secondary to a ruptured hepatic abscess. There is a diffuse loss of serosal detail as well free peritoneal gas present throughout the abdomen. The liver is enlarged. There are multiple small rounded gas opacities present over the ventral and right aspects of the liver *(arrow).* Multiple small intestinal loops contain gas and fluid, some of which measure at the upper limits or over the upper limit of normal. **B,** Free peritoneal air can be seen clearly *(arrows)* using a horizontal beam view. **C,** Comet-tail artifact *(arrow)* due to the presence of gas within the liver on ultrasound examination. **D,** Hepatic abscess from the same dog at necropsy.

spectrum, after which it should be adjusted based on the results of culture and susceptibility. Mortality rates of 50% and 79% have been reported for dogs and cats, respectively.[45] The prognosis is especially poor when multiple abscesses are present.[45,46]

Pancreatitis and Pancreatic Abscesses

In dogs and cats, pancreatitis and pancreatic abscess formation most often appears to be a sterile process that results from tissue destruction and necrosis by pancreatic enzymes. Experimentally, necrotizing pancreatitis can be complicated by secondary bacterial invasion due to ascending infection from the duodenum or bacterial translocation,[49,50] although the extent to which this occurs in dogs and cats with naturally-occurring pancreatitis remains unclear. Occasionally, pancreatitis results from hematogenous spread of bacteria to the pancreas, usually in addition to other organs (see Table 86-2). Other microorganisms known to cause pancreatitis as a result of systemic infection include *Leptospira* species, *Toxoplasma gondii*, and fungi such as *Cryptococcus neoformans*. In cats, pancreatitis can also result from invasion by flukes *(Eurytrema procyonis)*.[51] When a primary infectious cause of pancreatitis cannot clearly be identified, the use of antimicrobial drugs to treat pancreatitis in dogs and cats is controversial. If used, the primary goal of antimicrobial treatment is to prevent or treat secondary ascending bacterial infection or bacterial translocation; treatment should target enteric bacteria. However, no evidence currently exists that use of antimicrobials alters outcome in dogs and cats with naturally occurring pancreatitis, and it has the potential to select for infection by multidrug-resistant enteric pathogens. In addition, many widely used antimicrobial drugs in veterinary medicine (such as β-lactams and aminoglycosides) penetrate inflamed pancreatic tissue poorly. Suitable choices include fluoroquinolones (for gram-negative aerobes), third-generation cephalosporines, and metronidazole (for anaerobes).

CASE EXAMPLE

Signalment: "George," a 16-year-old male neutered domestic shorthair from northern California

History: George was evaluated for a 2-day history of vomiting and progressive lethargy and inappetence. The owners reported that the vomiting occurred six times at the onset of illness and was associated with apparent abdominal pain but had since ceased. The day he became ill, he was seen at an emergency clinic where a CBC showed only a mild left shift (479 band neutrophils), a chemistry panel showed mild hypokalemia (3.1 mmol/L, reference range 3.6-4.9 mmol/L), and a total serum T4 concentration was increased (10.2 µg/dL, reference range, 1.1-3.3 µg/dL). Pancreatitis was suspected and George was initially treated with subcutaneous lactated Ringer's solution (200 mL SC q24h), mucosal buprenorphine (0.02 mg/kg PO q8h to q12h), and ondansetron (0.2 mg/kg PO q12h) pending the results of laboratory testing. However, because George became inappetent and lethargic, he was returned for reevaluation the following day.

Other medical history: George had been diagnosed with pancreatitis 8 months previously. He was also diagnosed with a biliary cystadenoma at that time. He had been hospitalized for 9 days and treated with jejunostomy tube feeding. Since then, he had been well until this recent onset of illness.

Physical Examination:

Body Weight: 6.1 kg.

General: Quiet, alert, hydrated. T = 103.8°F (39.9°C), HR = 240 beats/min, RR = 32, mucous membranes pink and moist, CRT = 1 s.

Eyes, Ears, Nose, and Throat: Palpable thyroid nodule.

Musculoskeletal: Body condition score was 5/9. The cat was ambulatory with normal gait.

Cardiovascular: A gallop rhythm was present. No murmurs or arrhythmias were auscultated. Femoral pulses were strong and synchronous.

Genitourinary/Gastrointestinal: Soft, mildly painful abdomen. The urinary bladder was small.

Other Systems, Including Peripheral Lymph Nodes: No clinically significant findings.

Imaging Findings:

Abdominal Ultrasound: The margins of the kidneys were irregular, and there was decreased corticomedullary distinction. In the region of the left limb of the pancreas, the mesentery was markedly hyperechoic and hyperattenuating. There was diffuse thickening of the muscularis of the small bowel. There was no evidence of mesenteric lymphadenomegaly. There was a small amount of free anechoic peritoneal fluid.

Thoracic Radiographs: The cardiac silhouette appeared mildly enlarged. There was a mild bronchointerstitial pattern throughout the thorax. There was poor serosal detail within the viewable cranial abdomen.

Outcome: George was treated supportively for pancreatitis with intravenous fluids (lactated Ringer's solution with 20 mEq/L KCl at 15 mL/hr), buprenorphine (0.01 mg/kg IV q6h), famotidine (0.5 mg/kg IV q12h), and ondansetron (0.5 mg/kg IV q12h). His temperature normalized, but he showed no interest in food or his surroundings. Cytologic examination of an ultrasound-guided liver aspirate on day 2 of hospitalization showed moderate hepatic lipidosis and mild mixed inflammation. Laboratory testing was reevaluated and an echocardiogram was performed on the third day of hospitalization in preparation for anesthesia and placement of an esophageal feeding tube. The echocardiogram was unremarkable; the gallop rhythm was suspected to result from a systolic click. New findings on the CBC were mild, nonregenerative anemia (HCT 29.9%, RR 30%-50%), with a WBC of 11,580 cells/µL (4500-14,000 cells/µL), 4053 neutrophils/µL (2000-9000 cells/µL), bandemia (3242 cells/µL), and circulating metamyelocytes (232 cells/µL). Findings on the serum biochemistry panel included a bicarbonate of 24 mmol/L (15-21 mmol/L), calcium of 8.3 mg/dL (9.0-10.9 mg/dL), BUN of 20 mg/dL (18-33 mg/dL), creatinine of 0.8 mg/dL (1.1-2.2 mg/dL), glucose of 114 mg/dL (63-118 mg/dL), total protein of 5.3 g/dL (6.6-8.4 g/dL), albumin of 2.0 g/dL (2.2-4.6 g/dL), globulin of 3.3 g/dL (2.8-5.4 g/dL), ALT of 57 U/L (27-101 U/L), AST

of 46 U/L (17-58 U/L), ALP of 26 U/L (14-71 U/L), GGT of <3 U/L (0-4 U/L), cholesterol of 109 mg/dL (89-258 mg/dL), and total bilirubin of 0.4 mg/dL (0-0.2 mg/dL). Urinalysis showed a specific gravity of 1.015; pH 7.0, protein 25 mg/dL, no bilirubin, 25 erythrocytes/µL hemoprotein, 1-3 WBC/HPF, 1-6 RBC/HPF, and many lipid droplets. Serum vitamin B_{12} and folate concentrations were 383 ng/L (279-1254 ng/L) and 7.5 ng/mL (10.4-20.7 ng/mL), respectively.

Because of the severely left-shifted neutrophil count and mild hyperbilirubinemia, bacterial cholecystitis was suspected. Findings on abdominal ultrasonography were unchanged from the previous examination. Ultrasound-guided cholecystocentesis was performed while George was under anesthesia for esophagostomy tube placement, and a small volume of the scant peritoneal fluid present was also collected.

Microbiologic Testing:

Cytologic Examination (Bile): Smears had light blue backgrounds and contained a moderate amount of unidentified material. Nucleated cells were not found, but many short, plump coccobacilli were seen. Most of the bacteria were in large clusters, but a few groups were in chains.

Peritoneal Fluid Analysis: Grossly the fluid was pink and cloudy, and it had a yellow, clear supernatant. The total protein concentration was 2.8 g/dL and there were 100,000 RBC/µL. There were 5380 nucleated cells/µL, with 70% nondegenerate neutrophils, 6% small and well-differentiated lymphocytes, 7% foamy macrophages, and 17% eosinophils. No infectious agents were identified.

Aerobic and Anaerobic Bacterial Culture (Bile): Large numbers of *Enterococcus faecium*. No anaerobes were cultured. The isolate was susceptible to amoxicillin-clavulanic acid (≤4 µg/mL), ampicillin (≤0.25 µg/mL), penicillin (0.12 µg/mL), chloramphenicol (≤4 µg/mL), doxycycline (≤2 µg/mL), imipenem (≤1 µg/mL); had intermediate susceptibility to erythromycin (1 µg/mL); and was resistant to rifampin (>2 µg/mL).

Aerobic and Anaerobic Bacterial Culture (Peritoneal Fluid): Very small numbers of *Enterococcus faecium*. The antibiogram was identical to the isolate from the bile.

Diagnosis: Peritonitis and suspected bacterial cholecystitis caused by *E. faecium*; pancreatitis and secondary hepatic lipidosis.

Treatment: Medical treatment with ampicillin-sulbactam (20 mg/kg [ampicillin component] IV q8h) and enrofloxacin (5 mg/kg slow IV q24h) was commenced on day 3 of hospitalization, before the results of culture and susceptibility were available. Treatment was also continued with intravenous fluids, buprenorphine, famotidine, ondansetron, and nutrition through the esophageal feeding tube with a slurry of a commercially available gastroenteric diet (Purina EN Feline Formula, q4h). Surgery was considered, but because George was stable and the amount of free peritoneal fluid was scant, a decision was made to continue medical treatment with close observation and serial CBC and abdominal ultrasound examinations. Within 12 hours of antibiotic treatment, George was brighter. A repeat abdominal ultrasound showed no changes except that mildly enlarged and hypoechoic mesenteric lymph nodes were identified. The CBC showed persistent nonregenerative anemia (HCT of 26.4%), 10,181 neutrophils/µL, 5553 bands/µL, 185 metamyelocytes/µL, and a normal platelet count (305,000/µL) with moderately toxic neutrophils. On day 5 of hospitalization, ultrasonography showed a decrease in the amount of peritoneal fluid. The enrofloxacin was discontinued when the results of culture and susceptibility became available. On day 6, the CBC showed a stable HCT, 9366 neutrophils/µL, 2810 bands/µL, moderately toxic neutrophils, and no metamyelocytes. A serum chemistry panel was unremarkable, and the total bilirubin was 0.1 mg/dL. On day 8, George began eating a small amount of food on his own, and the CBC showed further improvement with 6048 neutrophils/µL, 336 bands/µL, mild monocytosis (896 cells/µL), and no toxic neutrophils. He was discharged from the hospital the following day with instructions to continue tube feeding, ondansetron (0.3 mg/kg PO q24h), famotidine (0.5 mg/kg PO q12h), and amoxicillin–clavulanic acid (15 mg/kg PO q12h). At a recheck 1 week later, the owner reported that George had been doing well at home, although he had vomited three times over the course of the week. Physical examination was unremarkable, apart from the thyroid nodule and mild weight loss (body weight, 5.6 kg compared with 5.9 kg at discharge). A CBC showed a HCT of 30%, 10,592 neutrophils/µL, and 132 bands/µL. A serum chemistry panel was within normal limits, and the total T4 concentration was 8.2 µg/dL. Treatment with methimazole (1.25 mg q12h) was initiated for the hyperthyroidism, but because of continued vomiting, this was discontinued after three doses. George continued to improve. A week later he was eating canned and dry food on his own with no vomiting, a CBC showed no abnormalities, and the antibiotics were discontinued. The esophageal tube was removed 3 weeks later, and methimazole was reinstituted, after which his T4 normalized. Six months later George was still doing well.

Comments: In this cat, bacterial peritonitis and suspected cholecystitis were diagnosed based on cytologic and microbiologic analysis of bile and peritoneal fluid. However, there were no ultrasonographic abnormalities within the biliary tract, and cytologic evidence of inflammation in the bile was absent. Pancreatitis may have predisposed the cat to ascending infection of the biliary tract, and/or infection may have resulted from bacterial translocation. The lack of an inflammatory reaction in the bile was unusual, but it may have resulted from marked neutrophil degeneration in a toxic environment. Isolation of the same organism from both the bile and the peritoneal fluid and the rapid clinical response to antibiotic treatment suggested that *Enterococcus* played a role in disease in this cat, although some of the clinical signs and hematologic abnormalities may also have resulted from severe pancreatitis and hepatic lipidosis. Surgery was postponed with careful monitoring because the peritonitis appeared to be very low grade and there was concern it might exacerbate pancreatitis. Fortunately, the cat made a complete recovery. See Chapter 8 for precautions regarding administration of enrofloxacin to cats.

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Callahan Clark JE, Haddad JL, Brown DC, et al. Feline cholangitis: a necropsy study of 44 cats (1986-2008). J Feline Med Surg. 2011;13:570-576.

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