Inflammatory Liver Diseases in Cats

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ABSTRACT: Inflammatory liver diseases comprise the second most common cause of liver disease in cats. The two major forms of inflammatory liver disease described in North America are cholangiohepatitis (acute and chronic forms) and lymphocytic portal hepatitis. Signalment, clinical signs, clinical laboratory data, and ultrasonography can be used to establish a presumptive diagnosis of inflammatory liver disease; however, hepatic biopsy is needed for definitive diagnosis. Differences in treatment strategies for cholangiohepatitis and lymphocytic portal hepatitis warrant establishing a definitive diagnosis. Prognosis for long-term survival is good for cats that survive the first 2 to 3 months after diagnosis of either of these diseases.

In North America, major causes of feline liver disease include the following:

- Hepatic lipidosis—50% of cases
- Inflammatory liver disease—25%
- Malignant lymphoma—5%
- Carcinoma—4%

Terminology used to describe inflammatory liver diseases of cats can be confusing. These terms include suppurative or acute cholangiohepatitis, chronic cholangiohepatitis, chronic progressive nonsuppurative cholangiohepatitis, pericholangiohepatitis, chronic lymphocytic cholangitis, progressive lymphocytic cholangitis, sclerosing cholangitis, lymphocytic-plasmacytic cholangitis/cholangiohepatitis, lymphocytic cholangitis/cholangiohepatitis, lymphocytic portal hepatitis, and biliary cirrhosis. Some authors have preferred to use the general term cholangiohepatitis complex or cholangiohepatitis/cholangitis complex. Recent retrospective studies have clarified the classification of these diseases into acute cholangiohepatitis, chronic cholangiohepatitis, and lymphocytic portal hepatitis.

TYPES OF INFLAMMATORY LIVER DISEASE

Although there is no universally accepted classification of inflammatory liver disease in cats, a classification scheme based on histopathologic features is gaining support. Two major types of inflammatory liver disease have been described based on distinct histopathologic features: cholangiohepatitis (acute and chronic forms) and lymphocytic portal hepatitis. Another syndrome that may...
be distinct from cholangiohepatitis and lymphocytic portal hepatitis, called progressive lymphocytic cholangitis/cholangiohepatitis, has been described.\textsuperscript{6,7} Liver fluke infestation can also cause inflammatory liver disease.\textsuperscript{8}

### Acute Cholangiohepatitis

Acute cholangiohepatitis is characterized by infiltration of large numbers of neutrophils into portal areas of the liver and into bile ducts (Figure 1).\textsuperscript{1} Disruption of the periportal limiting plate results in necrosis of hepatocytes adjacent to portal areas and infiltration of neutrophils into hepatic lobules. Acute cholangiohepatitis may begin as an ascending bacterial infection within the biliary tract; however, bacteria have been isolated from the liver or gallbladder in only a few cases.\textsuperscript{2,9} Failure to isolate organisms may be due in part to previous antibiotic therapy and failure to culture for anaerobic organisms. Organisms that have been isolated include Bacteroides, Escherichia coli, Clostridium, and α-hemolytic Streptococcus.\textsuperscript{2} Congenital or acquired abnormalities of the biliary system, including anatomic abnormalities of the gallbladder or common bile duct and gallstones (i.e., choleliths), may predispose cats to cholangiohepatitis. Choleliths appear to be an infrequent cause of inflammatory liver disease.\textsuperscript{2} Conversely, inspissation of bile, which may cause partial or complete obstruction of the common bile duct, gallbladder, or intrahepatic bile ducts, appears to frequently accompany cholangiohepatitis and may require surgical intervention.

### Chronic Cholangiohepatitis

Chronic cholangiohepatitis is theorized to be a later stage of acute cholangiohepatitis.\textsuperscript{1,10} It is characterized by a mixed inflammatory infiltrate in portal areas and bile ducts consisting of neutrophils, lymphocytes, and plasma cells (Figure 2).\textsuperscript{1} Unlike acute cholangiohepatitis, bile duct hypertrophy and portal fibrosis are prominent features of the disease. Other conditions that may be confused with chronic cholangiohepatitis include feline infectious peritonitis virus, Toxoplasma infections, and liver fluke infestation. In liver fluke infestation, eosinophil numbers are usually increased in portal areas, whereas eosinophils are rare or absent in other types of chronic cholangiohepatitis.

Diseases frequently associated with cholangiohepatitis include inflammatory bowel disease and pancreatitis. Of cats with cholangiohepatitis, 83% had concurrent inflammatory infiltrates in the duodenum and/or jejunum and 50% had pancreatic lesions.\textsuperscript{9} How these disorders relate to one another is unclear. Some authors theorize that inflammatory bowel disease causes reflux of bacteria or other constituents of duodenal contents into the common bile duct with resultant pancreatitis and cholangiohepatitis.\textsuperscript{1–5}

### Lymphocytic Portal Hepatitis

Lymphocytic portal hepatitis appears to be distinct from acute and chronic cholangiohepatitis. It is characterized by infiltration of lymphocytes and plasma cells but not neutrophils into portal areas but not into bile ducts (Figure 3). Variable degrees of bile duct hypertrophy and fibrosis are present.\textsuperscript{1,10} Lymphocytic portal hepatitis is a common finding in older cats. In a retrospective study of cat livers obtained at necropsy, 82% of cats over 10 years of age had...
cats in the United States. The incidence of infestation varies by geographic region. The condition occurs frequently in Florida, for example, whereas only one case was identified over 10 years at the University of Minnesota Veterinary Teaching Hospital. Histopathologic features of liver fluke infestation include distended and fibrotic bile ducts and epithelial proliferation of bile duct walls. A mixed inflammatory infiltrate, consisting of macrophages, lymphocytes, plasma cells, eosinophils, and neutrophils, is frequently present in portal areas. The presence of eosinophils is suggestive of liver fluke infestation because eosinophils are rarely seen in other types of inflammatory liver disease.

**DIAGNOSIS**

**Clinical Signs**

Clinical signs associated with inflammatory liver diseases are variable, nonspecific, and frequently similar to those associated with hepatic lipidosis (Table 1). Partial or complete anorexia is the most common (and sometimes the only) clinical sign. Other less frequently observed clinical signs include weight loss, vomiting, diarrhea, and fever. Cats with acute cholangiohepatitis tend to be younger (median age, 3.3 years) than cats with chronic cholangiohepatitis (median age, 9 years), lymphocytic portal hepatitis (median age, 8.2 years), or hepatic lipidosis (median age, 6.2 years). Male cats are more frequently affected with acute cholangiohepatitis than are female cats. Cats with acute cholangiohepatitis are more acutely and severely ill than cats with most other types of liver disease. Prominent clinical signs in acute cholangiohepatitis include fever, depression, and dehydration. Jaundice and altered liver size are frequently the only findings that direct attention to liver disease (Table 1). Jaundice is most easily observed in the sclera but

<table>
<thead>
<tr>
<th>Observation</th>
<th>Acute Cholangiohepatitis</th>
<th>Chronic Cholangiohepatitis</th>
<th>Lymphocytic Portal Hepatitis</th>
<th>Hepatic Lipidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Young</td>
<td>Middle age</td>
<td>Middle age</td>
<td>Middle age</td>
</tr>
<tr>
<td>Gender bias</td>
<td>Males</td>
<td>Males</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Onset of signs</td>
<td>Days</td>
<td>Weeks</td>
<td>Weeks</td>
<td>Days to weeks</td>
</tr>
<tr>
<td>Severity of illness</td>
<td>Severe</td>
<td>Mild to moderate</td>
<td>Mild</td>
<td>Mild to severe</td>
</tr>
<tr>
<td>Anorexia</td>
<td>Frequent</td>
<td>Frequent</td>
<td>Frequent</td>
<td>All cases</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Frequent</td>
<td>Infrequent</td>
<td>Frequent</td>
<td>All cases</td>
</tr>
<tr>
<td>Fever</td>
<td>Frequent</td>
<td>Infrequent</td>
<td>Frequent</td>
<td>Frequent</td>
</tr>
<tr>
<td>Jaundice</td>
<td>Frequent</td>
<td>Frequent</td>
<td>Frequent</td>
<td>Frequent</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>Frequent</td>
<td>Frequent</td>
<td>Frequent</td>
<td>Frequent</td>
</tr>
<tr>
<td>Ascites</td>
<td>Rare</td>
<td>Rare</td>
<td>Rare</td>
<td>Rare</td>
</tr>
</tbody>
</table>
may also be observed in the soft palate or under the tongue. Ecchymotic hemorrhages and/or prolonged bleeding from venipuncture sites may occur. When liver size is evaluated radiographically, approximately half of cats with acute or chronic cholangiohepatitis, lymphocytic portal hepatitis, or hepatic lipidosis have hepatomegaly and the remaining cats have normal-sized livers. Therefore, hepatomegaly is a frequent finding in feline inflammatory liver diseases but cannot be used to differentiate the various causes.

**Laboratory Evaluation**

Hematologic and biochemical testing are essential to establish a diagnosis of liver disease (Table 2). Fasting bile acid determination is the test that is most consistently abnormal in all types of inflammatory liver disease. Laboratory changes typically seen with acute cholangiohepatitis include mild neutrophilia and left shift, normal or slightly increased serum bilirubin and serum alkaline phosphatase (SAP) levels, and substantially increased alanine aminotransferase (ALT) level. This profile tends to differentiate acute cholangiohepatitis from chronic cholangiohepatitis, hepatic lipidosis, and hepatic neoplasia. Laboratory changes typical of chronic cholangiohepatitis include substantial increases in serum bilirubin, SAP, and ALT levels. Other associated changes may include mild nonregenerative anemia, hyperglobulinemia, lymphocytosis, and hyperglycemia. Laboratory alterations associated with lymphocytic portal hepatitis include normal to variably increased serum bilirubin, ALT, and SAP levels. When all inflammatory liver diseases are compared with hepatic lipidosis, patients with hepatic lipidosis tend to have higher total bilirubin concentrations and higher ALT and SAP levels. The hallmarks of hepatic lipidosis include jaundice and tenfold or greater increases in ALT and SAP levels without a corresponding increase in γ-glutamyl transferase (GGT). In other forms of liver disease in cats, increases in GGT tend to parallel increases in SAP.

When the clinical chemistry profile reveals evidence of liver disease, hyperthyroidism should be ruled out. Hyperthyroid cats frequently have changes in ALT and SAP levels that may be indistinguishable from those associated with inflammatory liver diseases. The increased enzyme concentrations normalize with treatment of hyperthyroidism. Pathologic changes in the liver associated with hyperthyroidism have not been well characterized.

**Liver Imaging**

Abdominal ultrasonography is often helpful in evaluating extrahepatic disorders associated with cholangiohepatitis. Most cats with acute or chronic cholangiohepatitis or lymphocytic portal hepatitis have variable or no detectable alterations in the echogenicity of the hepatic parenchyma. Conversely, most cats with hepatic lipidosis have hypechoic hepatic parenchyma. Bile duct abnormalities may be observed in cholangiohepatitis. These abnormalities include gallbladder and/or common bile duct distention, choledolithiasis, cholecystitis, and bile sludging (Figure 4). The normal gallbladder is anechoic and appears round in the transverse scan and pear-shaped in the longitudinal scan. It is important to remember that gallbladder filling occurs normally with fasting; therefore, caution must be exercised when interpreting gallbladder enlargement in an anorectic or fasting cat. The common bile duct can usually be seen as an anechoic, tortuous, tubular structure 2 to 4 mm in diameter with an echogenic wall. Distention of the gallbladder and common bile duct (i.e., greater than 5 mm in diameter) occurs as a result of cholecystitis or biliary obstruction. The gallbladder wall may become thickened as a result of inflammation.

**TABLE 2**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Acute Cholangiohepatitis</th>
<th>Chronic Cholangiohepatitis</th>
<th>Lymphocytic Portal Hepatitis</th>
<th>Hepatic Lipidosis</th>
<th>Hepatic Lymphoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neutrophilia</td>
<td>Frequent</td>
<td>Infrequent</td>
<td>Rare</td>
<td>Rare</td>
<td>Frequent</td>
</tr>
<tr>
<td>Left shift</td>
<td>Frequent</td>
<td>Infrequent</td>
<td>Rare</td>
<td>Rare</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Serum bilirubin</td>
<td>Mild increase</td>
<td>Moderate increase</td>
<td>Mild increase</td>
<td>Marked increase</td>
<td>Mild increase</td>
</tr>
<tr>
<td>Alanine aminotransferase</td>
<td>Moderate increase</td>
<td>Moderate increase</td>
<td>Mild to moderate increase</td>
<td>Moderate increase</td>
<td>Moderate to marked increase</td>
</tr>
<tr>
<td>Serum alkaline phosphatase</td>
<td>Frequently normal</td>
<td>Mild to moderate increase</td>
<td>Normal to mild increase</td>
<td>Moderate to marked increase</td>
<td>Moderate to marked increase</td>
</tr>
<tr>
<td>Fasting bile acids</td>
<td>Increased</td>
<td>Increased</td>
<td>Increased</td>
<td>Increased</td>
<td>Increased</td>
</tr>
</tbody>
</table>

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or edema. The thickened gallbladder wall has a layered or double-walled appearance. Bile sludge within the gallbladder or common bile duct appears echogenic.

Liver Cytology/Histopathology

Evaluation of pathologic changes in the liver is essential in differentiating inflammatory liver diseases from hepatic lipidosis and neoplasia. Percutaneous fine-needle aspiration; percutaneous tissue biopsy; and key-hole, laparoscopic, and intraoperative wedge biopsy procedures have been described. Fine-needle aspirations can usually be performed without anesthesia. When a 22-gauge needle is used for collection of aspiration specimens, the risk of hemorrhage is minimal; however, the risk is greater if large tissue biopsy needles are used. Therefore, coagulation testing is recommended before a tissue biopsy specimen is collected. Collection of bile from the gallbladder for cytologic evaluation and bacterial culture has the potential to result in rupture with resultant bile peritonitis. Therefore, aspiration of the gallbladder should be attempted only with ultrasonographic guidance or exploratory laparotomy.

The diagnostic utility of liver cytology is controversial. Several reports indicate that cytologic evaluation is highly efficient in identifying hepatic lipidosis and hepatic lymphoma; however, inflammatory liver diseases are more difficult to identify cytologically. Results of another retrospective study, however, indicate poor correlation between liver cytology and histopathology. Hepatic lipidosis is cytologically characterized by clusters of hepatocytes in which the cytoplasm is distended with lipid-filled droplets (Figure 5). Malignant lymphoma cells readily exfoliate and can be diagnosed by cytologic evaluation (Figure 6). Cytologic diagnosis of inflammatory liver diseases is hampered by blood contamination, which introduces variable numbers of blood leukocytes into the samples. Therefore, the cytologist is left to determine whether leukocytes are of blood origin or represent inflammatory lesions within the liver (Figure 7). Cytologic techniques in which samples are collected without aspirating minimize blood contamination and improve the chances that inflammatory lesions can be identified. Use of a 22-gauge needle connected to a 12-ml syringe via an 84-cm flexible extension set (i.e., used for intravenous infusion lines) has been recommended. The needle is inserted into the liver and moved back and forth 8 to 10 times without aspiration.

Ultrasound-guided aspiration and biopsy techniques more consistently produce diagnostic specimens than do blind techniques. Ultrasound-guided fine-needle aspiration can be used to sample bile as well as hepatic...
parenchyma. Such samples can be examined cytologically to look for inflammatory cells and bacteria and can be cultured to confirm bacterial infections.

TREATMENT
Cholangiohepatitis
The major specific therapy for acute and chronic cholangiohepatitis is antibiotics. Surgical intervention has been recommended if discrete choleliths or complete biliary obstruction is identified. When complete extrahepatic bile duct obstruction is identified, surgical decompression and biliary-to-intestinal diversion (i.e., cholecystoduodenostomy or cholecystojejunostomy) are recommended. However, surgery is not indicated for bile duct enlargement or obstruction due to fluke infestation. Bacterial culture and sensitivity testing of liver tissue, bile, choleliths, or gallbladder specimens should be used to select appropriate antimicrobial agents whenever possible. Antibiotics chosen for treatment of cholangiohepatitis should be excreted in the bile in active form and should be active against aerobic and anaerobic intestinal coliforms. Tetracycline, ampicillin, amoxicillin, erythromycin, chloramphenicol, and metronidazole are excreted in the bile in active form; however, several of these agents have significant adverse side effects. Erythromycin is not effective against gram-negative bacteria, tetracycline is hepatotoxic, and chloramphenicol may cause anorexia. As a result, ampicillin or amoxicillin combined with clavulanic acid is frequently used. All are broad-spectrum antibiotics, effective against both gram-negative and gram-positive organisms, and are well tolerated by cats. These drugs may be combined with metronidazole to extend the spectrum to anaerobes and more coliforms. Treatment with antibiotics for 2 months or longer is recommended.

When cats with chronic cholangiohepatitis fail to respond to antibiotic therapy alone within 2 to 3 weeks, prednisolone is usually added as an empiric treatment. The antiinflammatory and immunosuppressive properties of prednisolone may be beneficial in limiting hepatocellular injury. Additionally, prednisolone may enhance appetite. An immunosuppressive dose of prednisolone (2.2 to 4 mg/kg q24h) should be used initially. The dosage is slowly tapered to an alternate-day regimen (1 to 2 mg/kg q48h) for long-term maintenance. A schedule commonly used is to start therapy at 2 mg/kg bid for 2 weeks, then progressively reduce the dosage as follows: 2 mg/kg sid for 2 weeks; 1 mg/kg sid for 2 weeks; and 1 mg/kg q48h for 4 weeks. Biochemical values should be monitored prior to each reduction in dosage, and the dosage should be reduced only if substantial improvement in clinical signs and substantial reductions in ALT and SAP levels are observed. Doses as low as 0.5 mg/kg q48h may be sufficient for long-term maintenance. Long-term corticosteroid treatment is well tolerated by most cats, and side effects are usually minimal.
Methotrexate in combination with prednisolone has been recommended for cats that fail to respond to prednisolone therapy. A suggested dose is 0.4 mg per cat divided into three doses and given over 24 hours. The dose is repeated every 7 to 10 days. Because methotrexate has moderate myelosuppressive potential, total leukocyte count should be monitored and treatment should be discontinued if total leukocyte count drops below 3000/μL.

Ursodeoxycholic acid is recommended for cats with all types of inflammatory liver disease. It has antiinflammatory, immunomodulatory, and antifibrotic properties as well as the ability to increase fluidity of biliary secretions. Ursodeoxycholic acid has been safely administered to cats at a dose of 10 to 15 mg/kg PO q24h. However, ursodeoxycholic acid should not be given to cats with complete bile duct obstruction. Efficacy has not been established for any type of feline liver disease, but clinical trials in human patients with hepatitis support improved quality of life. Adverse effects in cats are uncommon and usually limited to mild diarrhea.

Cats with acute cholangiohepatitis require aggressive supportive care. These cats are frequently acutely ill and have fluid and electrolyte derangements that should be corrected. Treatment with injectable vitamin K (5 mg SC q24–48h) can be given if bleeding diatheses develop. Hepatic encephalopathy appears to be uncommon in cats with acquired liver disease and is manifest most frequently by excessive salivation. Hepatic encephalopathy can be managed by giving lactulose (0.5 to 1.0 ml/kg PO q8h) with or without enteric antibiotics (neomycin [20 mg/kg PO q8–12h]). Anorexia must be managed promptly to prevent further deterioration in clinical condition and possible development of concurrent hepatic lipidosis. Assisted oral feeding should be tried for up to 12 hours after which a nasoesophageal tube should be placed. Ensuring adequate intake of a high-energy, high-protein diet is a high priority throughout treatment. Protein is an important nutrient for liver repair and regeneration and should not be restricted unless hepatic encephalopathy occurs.

The response of cats with cholangiohepatitis to therapy should be monitored though use of serial complete blood counts and chemistry profiles. Persistent increases in ALT activity and serum total bilirubin concentration and/or increasing SAP activity suggest that treatment has been inadequate.

Lymphocytic Portal Hepatitis

The approach to treatment of lymphocytic portal hepatitis is based on the hypothesis that the liver injury is immune mediated. Immunosuppressive doses of corticosteroids are used as described above for chronic cholangiohepatitis. Anecdotal reports indicate prolonged improvement in clinical signs with prednisolone treatment. Others, however, report poor control of disease progression with corticosteroid treatment. Azathioprine (0.3 mg/kg PO q48–72h) has been tried, but side effects, including inappetence and leukopenia, limit its use. Low-dose weekly methotrexate therapy has been used in a few affected cats.

Response to treatment for lymphocytic portal hepatitis is difficult to assess because the disease progresses very slowly. A persistent increase in ALT and/or increasing total serum bilirubin concentration during corticosteroid treatment indicates that the disease is inadequately controlled.

PROGNOSIS

Limited studies of the response of cats with cholangiohepatitis to antibiotic treatment suggest that survival of cats with acute and chronic forms of the disease is similar. Approximately half of the cats die or are euthanized within 90 days after diagnosis. The other half have prolonged survival. Better understanding of the pathogenesis of these diseases and initiation of standard treatment protocols combined, when needed, with surgical correction of bile duct obstruction should increase the number of cats that survive long-term.

Lymphocytic portal hepatitis appears to be a very slowly progressive condition. Mean survival for cats with this condition was reported to be 36.9 months. In three cats with an initial diagnosis of mild lymphocytic portal hepatitis with minimal fibrosis and no bile duct proliferation, necropsies were performed 4 to 7 years after initial diagnosis. Liver sections at necropsy showed marked progression in the severity of lesions, which were characterized by marked lymphocytic infiltrates, severe fibrosis, and severe bile duct hyperplasia. Therefore, the prognosis for cats with lymphocytic portal hepatitis appears to be primarily dependent on the severity of lesions at the time of diagnosis.

REFERENCES

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**ARTICLE #5 CE TEST**

The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. Choose the one best answer to each of the following questions; then mark your answers on the test form inserted in *Compendium*.

1. Dilation of the gallbladder and/or common bile duct is most frequently seen in which of the following feline liver diseases?
   a. cholangiohepatitis  c. lymphocytic portal hepatitis
   b. hepatic lipidosis  d. toxic hepatopathy

2. Histopathologic lesions consisting of infiltration of large numbers of neutrophils into portal areas and bile duct best describes which of the following types of feline liver disease?
   a. acute cholangiohepatitis
   b. hepatic lipidosis
   c. lymphocytic portal hepatitis
   d. hepatic cysts

3. A serum chemistry profile characterized by marked increases in SAP, ALT, and bilirubin levels and normal or slight increases in GGT is most consistent with which of the following feline liver diseases?
   a. acute cholangiohepatitis
   b. hepatic lipidosis
   c. lymphocytic portal hepatitis
   d. hepatic cysts

4. A cat presented with a history of acute onset of severe lethargy, dehydration, fever, and anorexia of 3 days’ duration is most consistent with which of the following types of feline liver disease?
   a. acute cholangiohepatitis
   b. hepatic lipidosis
   c. lymphocytic portal hepatitis
   d. hepatic cysts

5. Finding clusters of hepatocytes in which the cytoplasm contains large, clear vacuoles in a cytologic specimen of feline liver is most consistent with which of the following types of liver disease?
   a. acute cholangiohepatitis
   b. hepatic lipidosis
   c. lymphocytic portal hepatitis
   d. hepatic cysts

6. Which of the following is the most definitive test(s) for differentiating cholangiohepatitis from lymphocytic portal hepatitis and hepatic lipidosis?
   a. SAP and ALT  c. ultrasonography
   b. fasting bile acids  d. liver biopsy

7. Which of the following is the most appropriate antibiotic to use in treating acute or chronic cholangiohepatitis?
8. Which of the following treatment protocols is most appropriate for a cat with chronic cholangiohepatitis and complete bile duct obstruction?
   a. Treat with ampicillin for 3 to 4 months and then reevaluate to see whether bile duct obstruction has resolved.
   b. Surgically correct bile duct obstruction before initiating medical treatment.
   c. Treat with ursodeoxycholic acid to dissolve inspissated bile in the common bile duct.
   d. Treat with a combination of antibiotics and ursodeoxycholic acid and reevaluate bile duct obstruction in 3 to 4 months.

9. The presence of large immature lymphoid cells in portal areas is consistent with which of the following diseases?
   a. chronic cholangiohepatitis
   b. hepatic lipidosis
   c. lymphocytic portal hepatitis
   d. malignant lymphoma

10. Mild neutrophilia and left shift are most frequently associated with which of the following feline liver diseases?
    a. acute cholangiohepatitis
    b. hepatic lipidosis
    c. lymphocytic portal hepatitis
    d. chronic cholangiohepatitis