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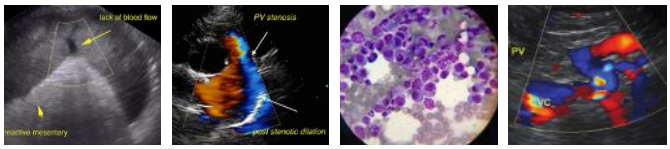

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1-800-838-4268 info@sonopath.com

PATIENT
Cookie Fox

SPECIES
Feline

BREED
DSH

SEX
FS

AGE
8 years

WEIGHT
5.8 pounds

INTERPRETED BY
Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY
Shari Reffi CVT

HOSPITAL NAME
American Animal Hospital

REFERRING VET
Dr. Vogel

INVOICE
10721ag

DATE
06/02/2022

PRESENTING CLINICAL SIGNS

History: Inappetent, 3 lb weight loss in 1.5 years. Elevated liver values. Also has coat change (now oily).
Current meds: Mirtazapine transdermal sid
Abnormal PE/Chem/CBC/UA Results: ALP 206, ALT 660, AST 222. TBILI 0.5, PLT 337, Normal T4, Hct 35%, TP 7.1

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder, trigone, and pelvic urethra presented normal thicknesses and normal tone. The ureters were not visible which is normal. No uroliths or sediment were visualized and anechoic urine was present. No evidence of inflammatory or neoplastic changes were noted. Ureteral papillae were normal.

The kidneys revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some minor age-related loss of curvilinear patterns regarding the capsule and C/M junction. The cortices presented largely uniform texture with some increased echogenicity expected for his age patient. Medullary structure differed distinctly from that of the cortex and no evidence of pelvic dilation was present.

The left kidney measured 3.24 cm in length. The right kidney measured 3.36 cm in length.

Adrenal Glands

Both adrenal glands were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The right adrenal gland measured 0.53 cm.

Spleen

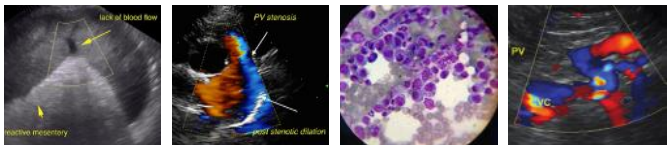
The spleen presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes were noted.

Liver

The liver images submitted revealed slight coarse architecture with diffusely hyperechoic falciform fat. The gallbladder presented acceptably thin walls with primarily anechoic content. The common bile duct was normal. The common bile duct measured 0.26 cm. The cystic duct was slightly tortuous, may be a normal variant. No evidence of post hepatic obstruction was present.

Gastrointestinal

Examination of the gastrointestinal tract revealed a stomach and intestine free of stasis, of normal wall thickness, acceptable curvilinear mural detail, and peristaltic activity. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively. No obstructive or overt infiltrative disease was noted. No associated abnormal lymphatic activity was noted.



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Pancreas

The base and limbs of the pancreas were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

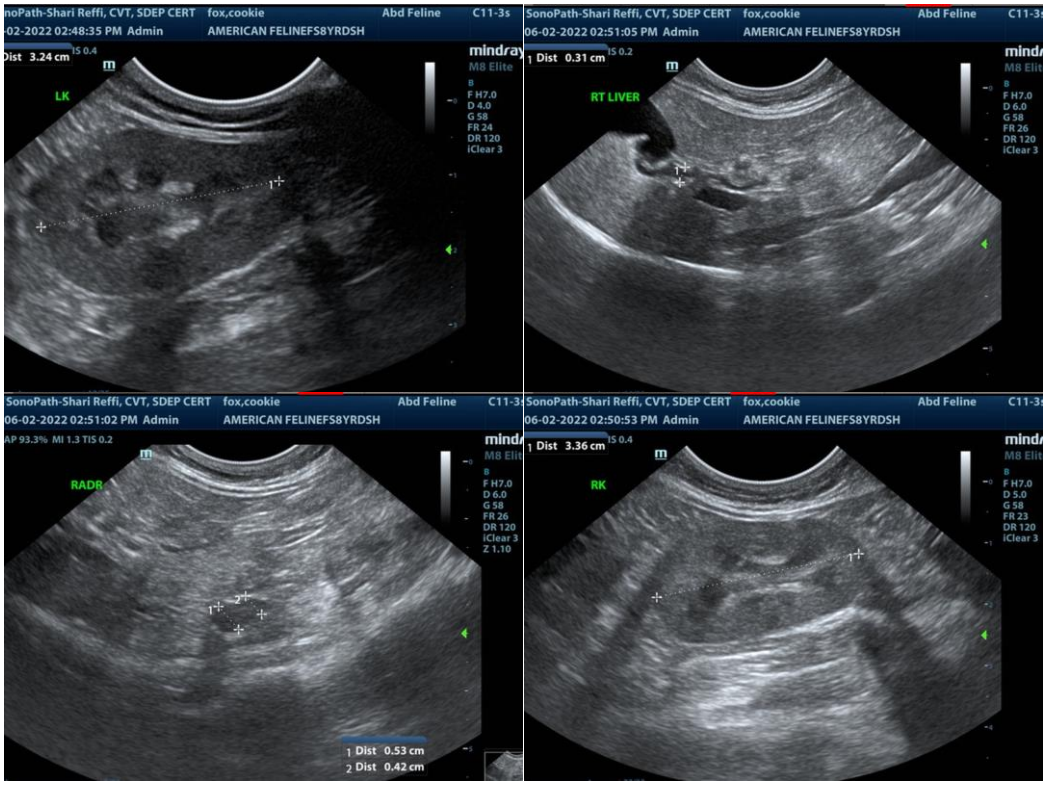
ULTRASONOGRAPHIC FINDINGS

- Hepatic lipidosis/inflammatory hepatopathy pattern

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

A 25g hepatic FNA assuming normal clotting status is warranted for further definition.
Empirical treatment for inflammatory hepatopathy and lipidosis is indicated. Underlying hepatic lymphoma is always a potential in this patient.

Maldigestion panel, three view chest radiographs and full CNS examination is recommended to examine for occult disease that could be responsible for the weight loss. Evaluation for competitive eating environments should also be considered.



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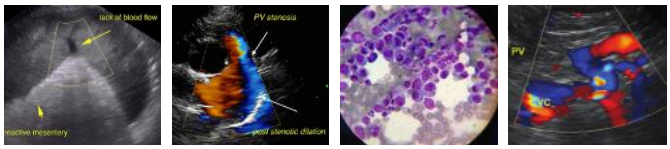

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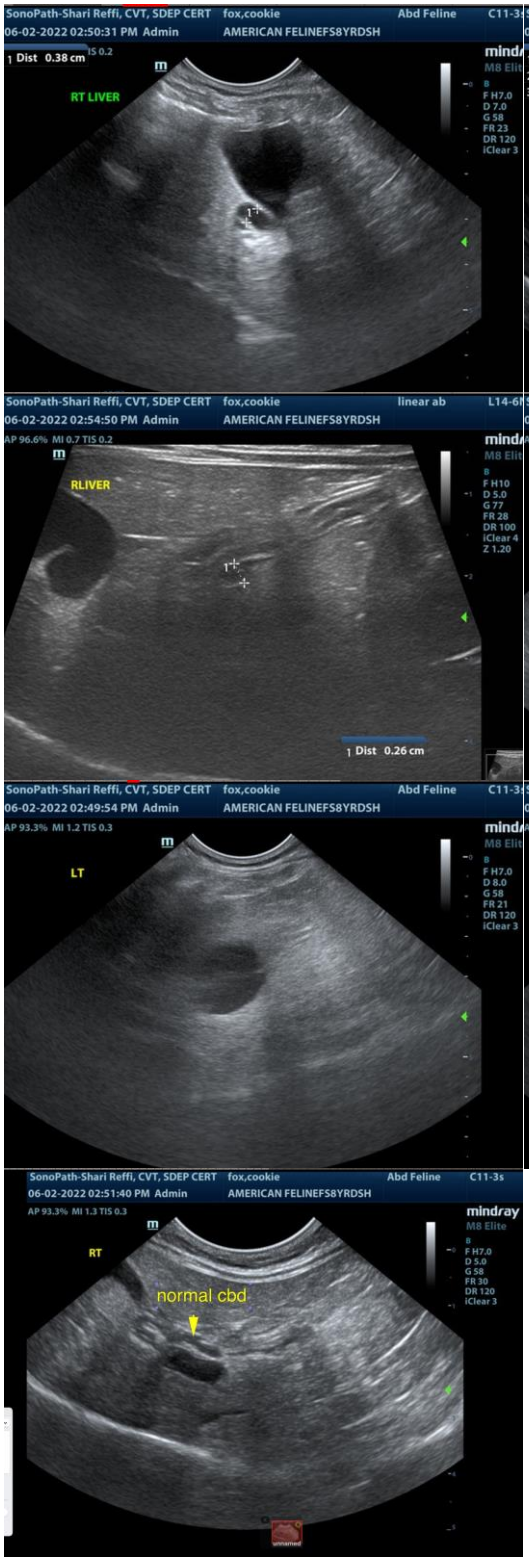
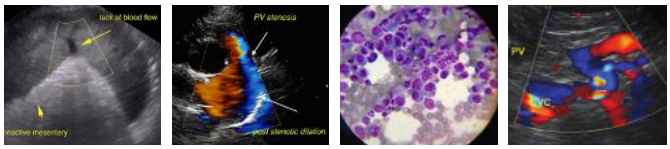


Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.



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Eric.Lindquist@SonoPath.com

Feline Liver Disease & Treatment Recommendations

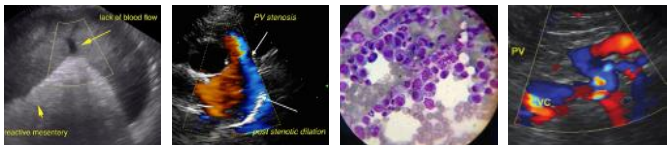
http://www.sonopath.com/FelineLiverDisease

Description: Liver disease is a common clinical condition in cats; however, it can be subdivided into specific disease categories. Cats most often develop feline cholangitis, which is comprised of various forms of inflammatory liver disease or hepatic lipidosis; however, there are other disease processes, including neoplasia, infectious disease, and toxicities, that result in hepatic dysfunction. This chapter will focus predominantly on feline cholangitis and hepatic lipidosis; hepatic neoplasia is discussed in greater detail in a separate chapter.

1. Feline cholangitis complex is one of the most significant diseases in cats. The term “complex” embodies many different disease processes, each with its own signs and treatment protocols. The World Small Animal Veterinary Association (WSAVA) liver Standardization Group classifies feline cholangitis accordingly: neutrophilic cholangitis, which includes both acute and chronic neutrophilic cholangitis (these are likely extensions of the same disease process); lymphocytic cholangitis; and cholangitis associated with a liver fluke infection.

a) Acute neutrophilic cholangitis is a suppurative disease process of the liver and is most commonly seen in young to middle-aged cats. Acute cholangitis is almost always of bacterial origin, with enteric isolates being the most common culprits (these are thought to ascend from the biliary tree). Pancreatitis and inflammatory bowel disease (IBD) are associated disease processes. Histopathologically, this disease is represented by suppurative inflammation within the walls and lumen of the biliary ducts, and may extend into the portal triads and possibly into the hepatic parenchyma.

b) Chronic neutrophilic cholangitis is either neutrophilic or lymphoplasmacytic, and arises from the acute form. Inflammation is centered around the portal region and includes lymphocytes, plasma cells, and neutrophils. Inflammation can also extend into the surrounding parenchyma and is sometimes noted within the lumen of the bile duct. Current research is investigating the role of *Bartonella* species in the development of chronic cholangitis; other postulated etiologies include a *Helicobacter* infection and immune-mediated processes. Biliary hyperplasia occurs secondary to the chronicity of the disease, and fibrosis and/or cirrhosis represent the end-stage manifestation of these foregoing disease processes. Cirrhosis is a rare condition in the cat because most cats either succumb or undergo successful treatment prior to this stage. Cats with chronic neutrophilic cholangitis often have chronic pancreatitis and IBD (triaditis). These other diseases may be responsible for the immune-mediated destruction of the liver that is commonly seen with the chronic form. The fact that the bile and pancreatic ducts are anatomically close to one another in cats may be the reason for coinfection and explain why the diseases occur in tandem.



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Severe lymphocytic cholangitis is defined as chronic inflammation of the biliary tract infiltrated by small lymphocytes. Cats with lymphocytic cholangitis are usually ill for months or years. This disease is more common in Europe than North America, and is manifested as a chronic inflammatory disease, which ultimately leads to fibrosis and cirrhosis. Chronic infections are thought to be the result of *Helicobacter* pylori infections and immune-mediated diseases. There is a greater predisposition in both Persian and younger cats to chronic forms.

c) Lymphocytic portal hepatitis is characterized by mild lymphocytic inflammation around the portal areas, but no inflammation within the bile ducts or hepatic parenchyma. This is a common finding upon biopsy, and although it can be nonspecific and incidental, it is also thought to indicate a reactive hepatopathy secondary to extrahepatic disease.

d) In tropical and subtropical geographic areas, cholangitis is associated with fluke infestation secondary to infection with *Platynosomum* spp. Cats become infected by ingesting the second intermediate host (reptiles and amphibians). The fluke infection results in cystic dilation and bile duct thickening as well as obstruction.

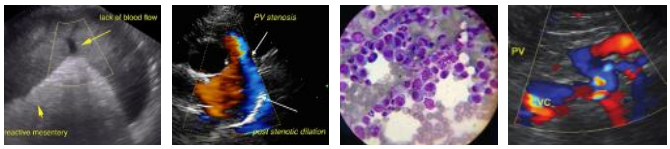
2. Hepatic lipidosis (HL) is one of the most common causes of hepatic disease in cats. It is defined as an accumulation of lipid within the cytoplasm of the hepatocyte. This can be idiopathic (primary) or can occur secondary to other diseases, and results in anorexia and weight loss. The pathophysiology is multifactorial and due to dysregulation of lipid metabolism in a catabolic state, which leads to excess accumulation of intracellular lipid within the hepatocyte.

Other common causes of liver enzyme elevation in cats include hepatocellular thyrotoxicosis, infectious diseases (e.g. *Toxoplasma* and FIP), and primary and metastatic neoplasia. In hepatic toxicosis, thyroid hormones have a direct toxic effect on liver cells and stimulate increased liver enzyme activity. Moreover, increased intestinal motility secondary to hyperthyroidism can cause increased oxygen utilization and thus hepatic hypoxia, which ultimately leads to hepatic dysfunction. Acute cholangitis cannot be fully ruled out without condcuting a biopsy. Owner (and patient) compliance with methimazole should be evaluated. The methimazole dose may need to be increased or other treatment modalities explored, including radioactive iodine, thyroidectomy, and/or dietary therapy with restricted iodine, such as the therapeutic diet, Hill's® y/d®. It should be noted that methimazole can also cause a drug-induced toxicosis and that this can also result in liver enzyme elevation.

Clinical signs:

Cholangitis: Because of the acute suppurative nature of acute neutrophilic cholangitis, clinical signs commonly include pyrexia and jaundice, accompanied by vomiting, diarrhea, and lethargy. Of the four main complex types discussed above, acute cholangitis patients normally present as the most severely ill. A chemistry panel often reveals a moderate increase in ALT, ALP, GGT, bilirubin, and bile acids, while a CBC commonly shows an elevated white blood cell count (WBC) with or without a left shift.

Cats suffering from the remaining types of feline cholangitis normally display less severe signs, but have likely been sick for a longer period of time. Those with chronic neutrophilic cholangitis can



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have intermittent episodes of jaundice and vomiting, which are cyclic and self-resolving. Weight loss, anorexia, and lethargy are common, and one typically observes elevations in ALP, GGT, bilirubin, and bile acids. The degree of elevation in ALT and AST is variable.

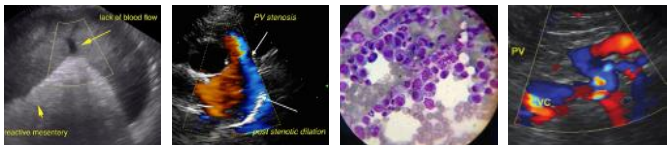
Patients with severe lymphocytic cholangitis exhibit weight loss and anorexia; however, because it is a slow-moving, progressive disease, signs may be chronic and mild. Liver enzyme elevations are generally mild until the chronic phase when icterus occurs as well as ascites. Hypergammaglobulinemia is also a prominent feature of this disease.

Cats with fluke infestations are either asymptomatic or systemically ill with pyrexia, vomiting, anorexia, icterus, and bile duct obstruction. A CBC may indicate marked liver enzyme elevations as well as an eosinophilia. Cats with lymphocytic portal hepatitis are asymptomatic and do not demonstrate laboratory abnormalities.

Hepatic lipidosis: The most common clinical signs are anorexia, vomiting, diarrhea, icterus, lethargy, depression, ptyalism, and weight loss. Since HL occurs in the face of so many other diseases, clinical signs may vary and be due, in part, to the underlying disease process. Likewise, lab work abnormalities can also vary, depending on concurrent disease processes. Hepatic encephalopathy may ensue, resulting in severe weakness, depression, and ptyalism. Common CBC abnormalities include a nonregenerative anemia, stress leucogram, poikilocytosis, and the presence of Heinz bodies. On the serum chemistry, an elevation in ALP is disproportionate to GGT levels, which are usually within normal limits. Serum ALT is variably increased, but typically of a lower magnitude than that of ALP. Bilirubin is increased due to intrahepatic cholestasis. Bile acids are increased, but are a superfluous indicator in the face of hyperbilirubinemia. The BUN and albumin may be normal or subnormal. Coagulation abnormalities occur in cats with HL due to vitamin K deficiency, which is a result of malabsorption in the intestines and decreased production of coagulation factors in the liver due to severe hepatic dysfunction.

Cats with thyroid toxicosis mainly exhibit clinical signs of hyperthyroidism. The chief abnormalities on serum chemistry are a mild to moderate elevation in ALT and elevated T4 levels. In the event of methimazole toxicity, the ALT can also be mild to moderately elevated. Cats with hepatic neoplasia may also demonstrate similar signs to other forms of hepatic disease, display elevations in ALT, ALP, GGT, and AST, and exhibit a leukocytosis and possibly anemia of chronic disease. This condition is discussed in greater detail in the chapter on “Hepatic Neoplasia.”

Diagnostics: Ultrasound and interventional ultrasound are important means of definitively diagnosing hepatic disease in cats. An ultrasound-guided core biopsy of the liver can be performed to acquire both histopathology and aerobic/anaerobic cultures. Alternatively, a larger tissue biopsy can be obtained via laparoscopy or laparotomy. In the event that a laparotomy or laparoscopy is performed, biopsies of the small intestine and pancreas should be obtained. One will also often encounter triaditis. Ultrasound-guided centesis of the gallbladder can be done to collect bile for culture and is preferred over hepatic parenchymal cultures. Ultrasound additionally allows for visualization of choleliths and obstruction to bile flow; it also helps determine whether surgical intervention is required. In the case of HL, the sonographic appearance presents as a uniform, diffuse, dense hyperechoic parenchyma that is hyperechoic to falciform fat and spleen; however, a primary underlying disease may also be present, such as cholangitis or lymphoma. In cats with



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suspected HL, fine needle aspiration (FNA) is the safest way to rule this out and to evaluate for lymphoma, as cytology is especially useful in the diagnosis of both these diseases. Lipidotic livers are friable and hence bleeding can occur as a complication of biopsy due to poor tissue integrity, lack of tissue hemostasis, and possibly compromised systemic hemostasis due to poor hepatic function. Cholangitis and neoplasia can be suspected on cytology, but a hepatic biopsy is preferred in order to define architecture, inflammatory infiltrate, and fibrosis. The clinician must weigh the risks and benefits of obtaining a biopsy in patients that may have a concurrent disease or are unresponsive to conventional therapy for HL. Pretreatment with vitamin K1 and aggressive supportive care may aid in stabilizing a patient for biopsy.

Cholangitis associated with liver flukes is uncommon in North America, but if the patient is in a tropical or subtropical location, the diagnosis is either obtained by fecal examination or liver biopsy, which permits observation of the flukes and/or their eggs within the bile ducts.

Treatment: The following medications are suggested in keeping with general guidelines for treatment for feline hepatic disease; however, each patient should be assessed and treated as an individual, and management should be tailored according to a specific diagnosis.

Disease-specific recommendations:

1. Feline cholangitis complex

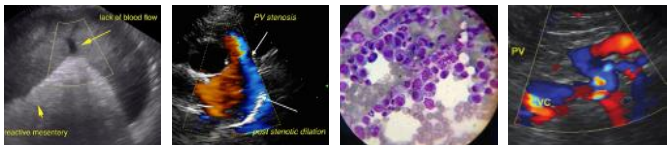
a) Acute neutrophilic cholangitis and chronic neutrophilic cholangitis:

- Antibiotics: Administration of antibiotics should ideally be based on culture and sensitivity. If a culture is not available, one may treat with broad-spectrum antibiotics, such as amoxicillin (10-20 mg/kg PO BID), amoxicillin clavulanic acid (10 mg/kg PO BID or 62.5 mg PO BID), a cephalosporin, such as cefadroxil (20 mg/kg PO BID), or enrofloxacin (5 mg/kg PO once daily). Antibiotics can be used for 4-8 weeks. Metronidazole (11-22 mg/kg PO BID) can be given as an anti-inflammatory medication; it also has an anaerobic spectrum. Metronidazole also decreases ammonia produced by intestinal microbes (administer at 7.5 mg/kg PO BID-TID in cases of hepatic encephalopathy).

b) Chronic neutrophilic cholangitis (lymphoplasmacytic inflammation):

- An anti-inflammatory medication should be given when biopsy reveals that there has been significant infiltration with lymphocytes and/or plasma cells, or if the patient is not responding to antibiotic medication alone (in the absence of a biopsy).
- Prednisolone should be given at 1-2 mg/kg/day. Start at a higher dose, and wean over time to every other day in decreasing dosages every 2-4 weeks following the resolution of signs. Additional immunosuppressant medications are not typically used in cats for this disease.

c) Severe lymphocytic cholangitis:



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- Prednisolone should be dosed at 1-2 mg/kg/day; however, it remains controversial as to whether prednisolone is in fact effective in the course of this disease.
- Ursodiol (Actigall): 10-15 mg/kg PO Q24hr.

d) Liver fluke infection:

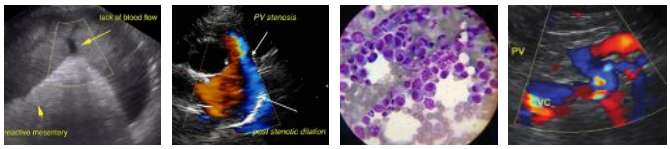
- Praziquantel: Give 20-30 mg/kg PO Q24hr for 3 days.

2. Hepatic Lipidosis

- Hyperalimentation is crucial in the management of HL; it may also be necessary in the management of other hepatic diseases in cats that are not eating so as to prevent hepatic lipidosis as a complication. In order to determine the caloric needs of the patient, calculate the basal energy requirement using the formula BER = 70 x BW $kg^{0.75}$. Multiply the BER by an illness energy requirement factor (1.25-1.4 in cats) and then select a therapeutic recovery diet with enhanced protein and fat levels, such as Hill's® a/d®, CliniCare® Liquid Diet (Abbott Animal Health), Royal Canin® Recovery RS™, or Iams® Maximum-Calorie™. Feed small, frequent meals through an esophagostomy tube (E tube), percutaneous endoscopic gastrostomy tube (PEG), or nasoesopahegal tube (NE) tube. Give slowly over 15-30 minutes or trickle feed as a CRI. In cats that have been anorexic for a prolonged period of time, the amount of food should be gradually increased over 3 days' time. The food should be made into a slurry and warmed, and the total amount of food divided into 4-6 feedings per day. Flush the feeding tube with 5-15 ml warm water. Pretreatment with cisapride (1.25-2.5 mg/cat PO or 0.1-0.5 mg/kg PO BID-TID) or metoclopramide (0.2-0.4 mg/kg PO or SC Q8hr) can prove helpful to improve gastric emptying; dosing should occur 30 minutes before feeding.
- Vitamin K1: Give 0.5-1.5mg/kg SQ or IM every 12 hours for a maximum of 2-3 doses if clotting times are increased. (The latter commonly occurs in the face of hepatic lipidosis due to decreased intestinal absorption of vitamin K as well as hepatic failure.)
- L-carnitine: Give 50-100 mg/kg PO Q24hr. L-carnitine is indicated in cats with severe hepatic lipidosis.
- Taurine: Give 250-500 mg PO Q24hr. Taurine can be administered as a supplement; it is an essential amino acid in cats.

3. Hepatic Encephalopathy:

- Lactulose: Give 0.5 ml/kg PO BID-TID to soften the stool. Lactulose helps manage hepatic encephalopathy by combining with ammonium in the GI tract and thus decreasing circulating ammonia levels. It can also be mixed into the slurry during feeding. Lactulose can also be given as a retention enema in an encephalopathic crisis.
- Metronidazole or neomycin: Give metronidazole at 7.5 mg/kg PO BID-TID. This is an antimicrobial, which reduces bacterial counts and reduces ammonia production in the colon. Alternatively, administer neomycin at 20 mg/kg PO BID-TID.



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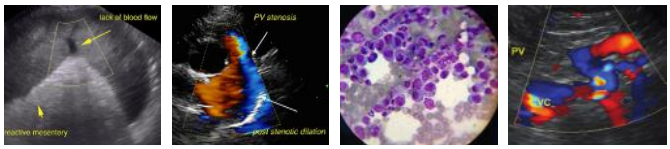
- L-Carnitine: Give 50-100 mg/kg PO Q24hr. Normally synthesized by the liver, L-carnitine enhances ammonia elimination and is indicated in cases of hepatic encephalopathy and lipidosis. Carnitine must be in the L-form.
- Diet: A low-protein diet with high amounts of biologically available protein is recommended in encephalopathic patients to reduce the nitrogen load from the breakdown of amino acids.

General treatment recommendations for cats with either feline cholangitis complex or hepatic lipidosis:

- IV Fluids: Fluid therapy is integral, especially in cats with severe liver disease as they are often inappetant and dehydrated. In the face of hepatic failure, avoid Lactated Ringer's solution (LRS) as lactate is metabolized by the liver. Monitor electrolytes closely. Add potassium in the form of potassium chloride (KCL); 20 mEq/L is a general starting point, but higher doses may be needed in the face of severe hypokalemia (maximum infusion rate is 0.5 mEq/kg/hr). Correct concurrent hypomagnesemia with magnesium sulfate or magnesium chloride at 0.75-1 mEq/kg/day given as a CRI for one day, and then reduce it to 0.3-0.5 mEq/kg/day. Monitor serum phosphorus levels and supplement as needed. Hypophosphatemia can occur following the reinstitution of feeding, especially in previously anorexic patients (re-feeding syndrome). Supplement phosphorus at 0.01-0.06 mmol/kg/hr using potassium phosphate or sodium phosphate.
- Vitamin B1 complex (thiamine) can also be added to the fluids at 1-2ml/liter. Note: Protect the fluid bag from light as the vitamins degrade when exposed to light.
- Vitamin B12 (cyanocobalamin) can be administered at 250 ug SC or IM weekly as needed in cases of HL or in cats with primary gastrointestinal disease.
- Famotidine can be given 0.5 mg/kg PO or IV once to twice daily as an antacid.
- Ursodiol (Actigall): Give 10-15 mg/kg PO Q24hr, with food, to stimulate bile secretion and flow, and decrease cholestasis. It has immunomodulatory, anti-fibrotic, and choleretic effects and anti-copper storage benefits; it also stabilizes mitochondrial function. Ursodiol can be compounded into a liquid formulation for cats.
- S-adenosylmethionine (SAMe): Give 90 mg/cat PO on an empty stomach (1-2 hours before feeding), or a loading dose of 35-60 mg/kg once to twice daily and a maintenance dose of 20 mg/kg PO Q24hr. SAMe replenishes glutathione and aids in cellular detoxification. It is also an anti-inflammatory and antioxidant.
- Antiemetics: these are used to decrease the frequency of vomiting and therefore enable enteral nutrition. A common antiemetic, metoclopramide (0.2-0.5 mg/kg SC TID 30 min before feeding or 0.01-0.02 mg/kg/hr as a CRI) has the beneficial effect of concurrently improving gastric emptying. Alternative anti-emetics include: maropitant citrate (Cerenia), which should be administered at 1 mg/kg subcutaneously once daily for up to 5 days; odansetron (Zofran), which can be dosed at 0.1 mg/kg PO once to twice daily or 0.1-0.3 mg/kg IV BID-TID; or dolasetron (Anzemet), which can be administered at 0.5 mg/kg PO, SC, or IV Q24hr. Silybin-phosphatidylcholine (Marin) (5 mg/kg PO Q24hr) is yet another alternative; however, to date there are no evidenced-based studies in cats on the effects of milk thistle. Nevertheless, it is suggested that it acts as an antioxidant and free radical scavenger, decreases hepatotoxin binding, improves glutathione concentrations, aids in iron chelating, and promotes choleresis.



SonoPath.com
1-800-838-4268 info@sonopath.com

PATIENT
Cookie Fox

SPECIES
Feline

BREED
DSH

SEX
FS

AGE
8 years

WEIGHT
5.8 pounds

INTERPRETED BY
Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY
Shari Reffi CVT

HOSPITAL NAME
American Animal Hospital

REFERRING VET
Dr. Vogel

INVOICE
10721ag

DATE
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- Vitamin E: Give 10-15 IU/kg/day PO (100-400 IU) in a water-soluble form twice daily.

Initially, in the face of hepatic failure and severe icterus, patients are hospitalized and monitored closely. Once stable, the patient can be discharged with a feeding tube if oral intake is not yet sufficient. Weight, appetite, and blood work must be carefully assessed. Specifically, ALT, SAP, serum protein, and serum albumin should initially be evaluated every 2 weeks, then monthly for the first 6 months, and subsequently every 4-6 months. A patient's response to therapy is typically assessed by their clinical condition and laboratory work; however, repeated biopsy or FNA may be necessary if the clinical response proves to be unsatisfactory.

Common drugs to avoid when treating hepatic disease: Halothane; sulphonamides; diazepam; azole antifungals; phenobarbital; tetracyclines; erythromycin or enrofloxacin if combined with theophylline or cisapride; and cimetidine if combined with theophylline, metronidazole, or chloramphenicol. Sedate with caution if lipidosis is present. If anesthesia is necessary for sampling or placing a feeding tube, induction with propofol should be considered with an inhalant, such as sevoflurane or isoflurane.

Conclusion: There are many pathological processes that can result in hepatic disease in the feline patient. Ideally, sampling is performed to obtain a definitive diagnosis, which allows for specific guidance with regards to therapy and prognosis. Adequate nutrition or hyperalimentation is critical for hepatic support when treating HL or preventing it from occurring as a secondary complication. Intensive care and monitoring are also key components of therapy in critically ill icteric cats.

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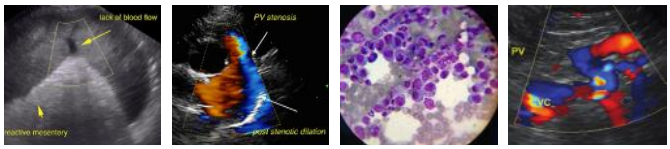

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