



PATIENT

Kasey Burke

SPECIES

Feline

BREED

DSH

SEX

Spayed Female

AGE

8 years

WEIGHT

Not Provided

PRESENTING CLINICAL SIGNS

Vomiting, increased ALT and TBili. Abn fpl Current meds: cerenia, buprenorphine

Abnormal PE/Chem/CBC/UA Results: Glu 278, Na 145, K2.8, Cl 98, ALT 266, TBili 4.1

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder, trigone, and cystourethral junction exhibited normal thickness and tone. Anechoic urine was present in the lumen with no uroliths or sediment. The ureteral papillae were normal. The ureters were not visible which is normal. No evidence of inflammatory or neoplastic changes was noted.

The area of the aortic trifurcation was free of pathology.

Normal size and margination were present in the kidneys. A normal 1:3 cortex / medulla ratio was maintained. The medulla and cortices were uniform in texture with some increased echogenicity and mild loss of corticomedullary symmetry and definition expected for the age of the patient. No evidence of pelvic dilation was present. The left kidney measured 3.5 cm in length. The right kidney measured 3.4 cm in length.

Adrenal Glands

The left adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The left adrenal gland measured 0.35 cm width. The right adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The right adrenal gland measured 0.47 cm width.

Spleen

The spleen exhibited a finely textured and homogenous parenchyma which was hyperechoic to the liver and renal cortical parenchyma. The capsule was smooth and regular without apparent expansion. The splenic vasculature at the hilus was normal in volume with no evidence of congestion or thrombosis. Acute to chronic inflammatory, neoplastic, or benign parenchyma changes were not noted. The spleen measured 0.67 cm width.

Liver/ Gallbladder

The liver was subjectively normal in size, structure, and contour. The liver exhibited subtle decreased echogenicity compared to the falciform fat and spleen with a moderate coarse echotexture and evidence of parenchymal remodeling. Subtle lobar biliary tree mineralization was present. The hepatic and portal vasculature were normal in appearance without signs of congestion. The gallbladder presented distended in size with mildly prominent to mildly echogenic walls. Moderate, nondependent yet nonorganized echogenic to mineralized debris was present within the gallbladder extending into the moderately dilated cystic bile duct. The common bile duct exhibited variable dilation with prominent walls and mild proximal common bile duct echogenic to focally mineralized mucus. The common bile duct dilation measured 0.4 cm in the mid common bile duct to 0.7 cm in the proximal common bile duct just distal to the cystic biliary duct. Subtle regional inflammation was noted around the gallbladder and common bile duct with mild, primarily perihepatic free fluid.

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(Canine and Feline)

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Jessica Miller

HOSPITAL NAME

Newton Vh

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Dr. Verhalen

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Gastrointestinal

The stomach presented intact wall layering with a normal wall layer ratio. Minor retained anechoic pyloric fluid was present. The pylorus wall width measured 0.26 cm.

The small intestine presented intact wall layering with 1:3 muscularis/mucosa ratio. The lumen of the small intestine was empty with no signs of ileus, obstruction, or foreign material. The jejunum wall width measured 0.20 cm. The ileocolic wall width measured 0.30 cm.

Normal visible colon wall layers were present with apparent formed feces in lumen.

Pancreas

The parenchyma of the pancreas was hyperechoic to adjacent omental fat with diffuse parenchyma remodeling. The capsule of the pancreas was mildly asymmetrical in contour without evidence of peripancreatic inflammation. These changes may suggest chronic inflammation, fibrosis, or saponification if previous history of pancreatitis. No overt signs of pancreatic neoplasia. Generalized pancreatic duct dilation was present.

Free Abdomen

Focal, pancreaticoduodenal or possible gastric lymph node was present. The lymph node was essentially isoechoic to adjacent omentum without evidence of peripheral inflammation and maintaining a normal width: length ratio (<0.5).

ULTRASONOGRAPHIC FINDINGS

Primary Findings

- Hepatopathy with mild lobar biliary tree mineralization - subjectively acute on chronic
- Distended gallbladder with nondependent, nonorganized, pinpoint mineralized luminal debris
- Variably thickened to distended cystic bile and common bile duct dilation with proximal common bile duct mucus
- Chronic pancreatitis pattern
- Minor retained pyloric fluid - suspect mild metabolic gastric stasis
- Perihepatic to pericholecystic inflammation with minor free fluid

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Acute on chronic cholangiohepatitis / cholangitis and chronic pancreatitis are likely. Potential for emerging post hepatic common bile duct obstruction, given the variable common bile duct distention, degree of gallbladder distention, and total bilirubin elevation is possible. No overt evidence of obstructive CBD or duodenal papilla pathology (calculi, masses, etc) were seen, yet cannot be excluded.

Empirical therapy for acute on chronic cholangiohepatitis and chronic pancreatitis with as-needed gastrointestinal support would be appropriate. However, close monitoring for hepatic response or increasing evidence of cholestasis is recommended. Potential for exploratory laparotomy with gross inspection of the common bile duct, common bile duct flush, bile culture and sensitivity, +/-



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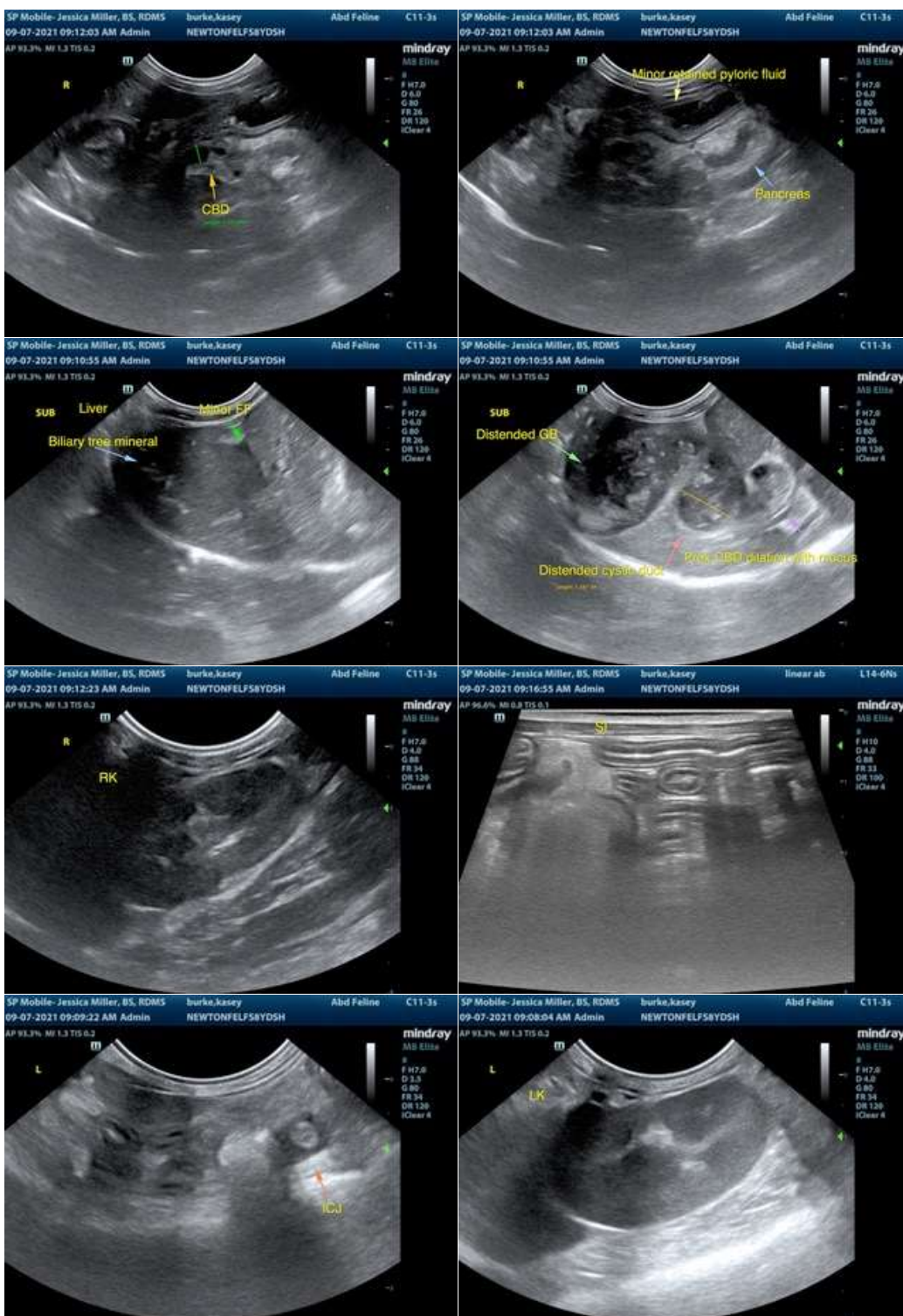
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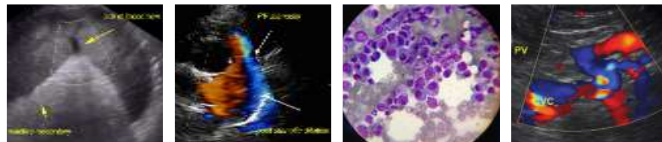
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hepatopancreatic biopsies may be Indicated if persistent or increasing hepatic enzyme elevations or cholestasis is noted.





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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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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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.

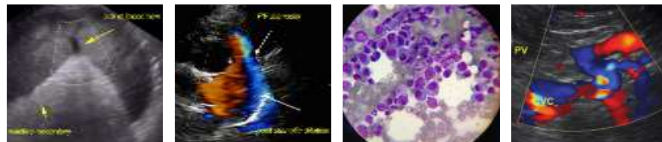
b) Chronic neutrophilic cholangitis is either neutrophilic or lymphoplasmacytic, and arises from the acute form. Inflammation is centered around the portal region and includes lymphocytes, plasma cells, and neutrophils. Inflammation can also extend into the surrounding parenchyma and is sometimes noted 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



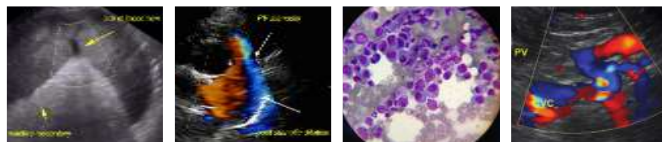
PATIENT	these foregoing disease processes. Cirrhosis is a rare condition in the cat because most cats either succumb or undergo successful treatment prior to this stage. Cats with chronic neutrophilic cholangitis often have chronic pancreatitis and IBD (triaditis). These other diseases may be responsible 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.
Kasey Burke	
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BREED	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 <i>Helicobacter pylori</i> infections and immune-mediated diseases. There is a greater predisposition in both Persian and younger cats to chronic forms.
DSH	
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Spayed Female	
AGE	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.
8 years	
WEIGHT	d) In tropical and subtropical geographic areas, cholangitis is associated with fluke infestation secondary to infection with <i>Platynosomum</i> 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.
Not Provided	
INTERPRETED BY	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.
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HOSPITAL NAME	Other common causes of liver enzyme elevation in cats include hepatocellular thyrotoxicosis, infectious diseases (e.g. <i>Toxoplasma</i> 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 conducting 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.
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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



PATIENT	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.
Kasey Burke	
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Feline	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 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.
BREED	
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Spayed Female	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.
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Not Provided	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.
INTERPRETED BY	
R. McKenzie Daniel, DVM, DABVP (Canine and Feline)	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.
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REFERRING VET	
Dr. Verhalen	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."
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9/7/21	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



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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 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:

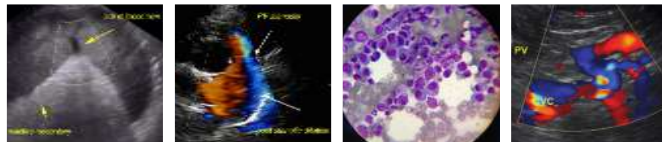
1. Feline cholangitis complex

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).



PATIENT

Kasey Burke

- 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.

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Feline

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.

SEX

Spayed Female

d) Liver fluke infection:

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

AGE

8 years

2. Hepatic Lipidosis

WEIGHT

Not Provided

- 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 $BER = 70 \times BW \text{ kg}^{0.75}$. 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 nasoesophageal 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.

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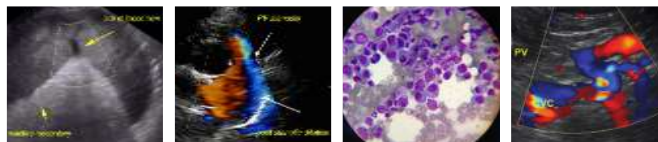
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3. Hepatic Encephalopathy:

- 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



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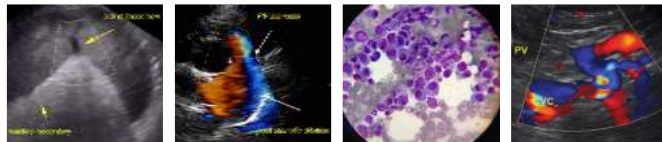
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circulating ammonia levels. It can also be mixed into the slurry during feeding. Lactulose can also be given as a retention enema in an encephalopathic crisis.

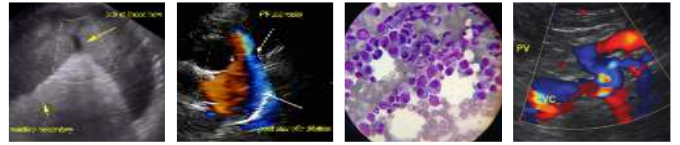
- Metronidazole or neomycin: Give metronidazole at 7.5 mg/kg PO BID-TID. This is an antimicrobial, which reduces bacterial counts and reduces ammonia production in the colon. Alternatively, administer neomycin at 20 mg/kg PO BID-TID.
- L-Carnitine: Give 50-100 mg/kg PO Q24hr. Normally synthesized by the liver, L-carnitine enhances ammonia elimination and is indicated in cases of hepatic encephalopathy and lipodosis. 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 lipodosis:

- 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 reinstatement 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 choleric 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,



PATIENT	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.
Kasey Burke	
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Feline	<ul style="list-style-type: none"> Vitamin E: Give 10-15 IU/kg/day PO (100-400 IU) in a water-soluble form twice daily.
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HOSPITAL NAME	Armstrong PJ, Blanchard G. Hepatic lipidosis in cats. <i>Vet Clin North Am Small Anim Pract</i> 2009;39(3):599-616.
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Dr. Verhalen	
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PATIENT

Kasey Burke

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BREED

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8 years

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INTERPRETED BY

R. McKenzie Daniel,
DVM, DABVP
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IMAGING PERFORMED BY

Jessica Miller

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HOSPITAL NAME

Newton Vh

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Dr. Verhalen

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INVOICE

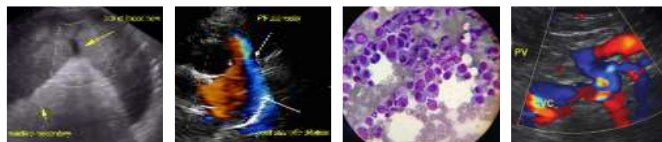
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PATIENT

Kasey Burke

SPECIES

Feline

BREED

DSH

SEX

Spayed Female

AGE

8 years

WEIGHT

Not Provided

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