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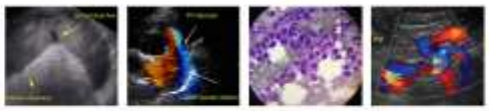

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**PATIENT**

Charli Downing

**SPECIES**

Canine

**BREED**

Fox Terrier Mix

**SEX**

MN

**AGE**

13 years

**WEIGHT**

22.2 lbs

**INTERPRETED BY**

R. McKenzie Daniel, DVM, DABVP (Canine and Feline)

**IMAGING PERFORMED BY**

Pamela Harrigan, RDCS

**HOSPITAL NAME**

Littleton AH

**REFERRING VET**

Christy Cox, DVM

**INVOICE**

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**DATE**

2/10/22

## PRESENTING CLINICAL SIGNS

Hyporexia, vomiting, responsive to Cerenia. Grade II-III/VI syistolic murmur. AST 112; ALT 1297; ALP 821; TBili 0.7. BP: 140-150 mmHg. Having bi-cavity ultrasound exams.

## ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

### *Urinary System*

The urinary bladder, trigone, cystourethral junction, and visible pelvic urethra to a depth of 3.0 cm 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 residual prostate was symmetrically normal in size with uniform parenchyma and slight coarse echotexture measuring 0.72 cm in diameter.

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. Intermittent small cortical cysts were present in the left kidney. Scant pyelectasia was noted in both kidneys. The left kidney measured 5.6 cm in length. The right kidney measured 5.1 cm in length.

### *Adrenal Glands*

The bilateral adrenal glands were normal in size. Mild parenchyma heterogeneity and mild capsule asymmetry were present without suspicion for overt neoplasia. The left adrenal gland measured 0.82 cm width in the cranial pole and 0.78 cm width in the caudal pole. The right adrenal gland measured 0.6 cm width in the cranial pole and 0.48 cm width in the caudal pole.

### *Spleen*

The spleen exhibited generalized mild to potential moderate enlargement yet primarily maintained symmetrical contour with generalized parenchyma heterogeneity exhibiting subjective mild generalized decreased parenchyma echogenicity. Normal splenic vasculature was present. No splenic masses or nodules were noted.

### *Liver/ Gallbladder*

The liver was subjectively normal in size, structure, and contour. The liver parenchyma was mildly nonuniform and hypoechoic to the spleen with a moderate coarse echotexture and moderate generalized parenchymal remodeling. Subtle areas of focal increased hepatic parenchyma echogenicity suggestive of areas of nodular to regenerative hyperplasia or very indistinct lipogranulomas are suspected. The hepatic and portal vasculature were normal in appearance without signs of congestion. The gallbladder was non-distended in size with mildly prominent to echogenic gallbladder walls containing primarily anechoic content with mild nondependent yet nonorganized, nonmineralized luminal debris. The cystic and common bile ducts were normal.

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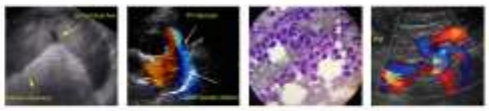

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### *Gastrointestinal*

The stomach presented intact wall layering with a normal wall layer ratio. The lumen of the stomach was empty with no signs of ileus, obstruction, or foreign material.

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 duodenum wall width measured 0.48 cm. The jejunum wall width measured 0.44 cm.

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

### *Pancreas*

The pancreas was mildly prominent in size with mild asymmetrical contour and heterogeneous to subtly hypoechoic parenchyma compared to adjacent omentum. No signs of active inflammation or neoplasia.

### *Free Abdomen*

Scant free fluid was noted around the caudal lateral aspect of the spleen. Subtle generalized echogenic omentum was present. No overt lymphadenopathy was noted.

## ULTRASONOGRAPHIC FINDINGS

- Chronic hepatitis liver pattern with parenchymal remodeling
- Mild cholecystitis with mild luminal debris (non-mucocele)
- Possible chronic to chronic active pancreatitis
- Nonspecific splenomegaly - hyperplasia, hematopoiesis, incidental splenitis, early neoplasia possible
- Bilateral chronic renal changes with small left kidney cortical cysts and bilateral scant pyelectasia
- Suspect mild gastroenteritis
- Minor perisplenic peritoneal free fluid

## INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Assuming normal clotting status, ultrasound guided hepatosplenic FNA using a 25-gauge needle is warranted for screening cytology with potential for identification of suspected inflammatory hepatic cell type and further assessment of the spleen. No overt evidence of hepatic neoplastic criteria was noted, which is considered a less likely differential diagnosis yet cannot be definitively excluded.

Empirically, hepatitis / pancreatitis protocol with as-needed gastrointestinal support would be appropriate.

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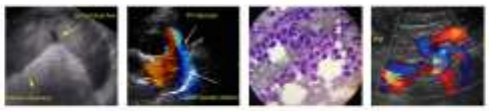

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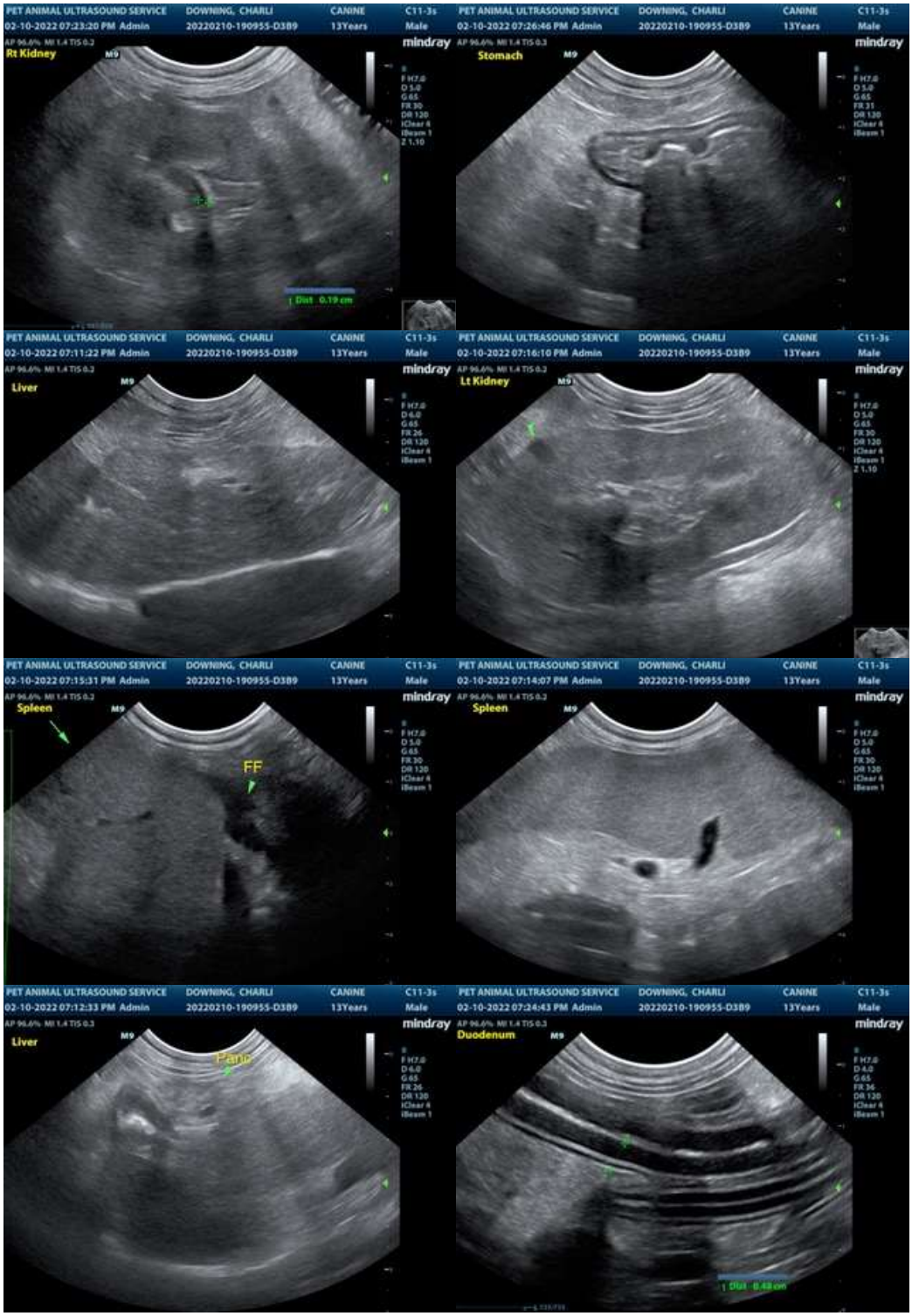
The pyelectasia in both kidneys may be owing to chronic renal changes, potential pelvic scarring possibly owing to previous calculi passage, IV fluid therapy (if applicable). Urine C/S and protein: creatinine ratio on sterile urine sample is recommended.



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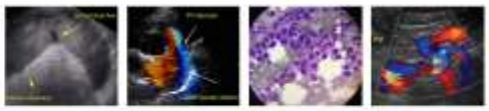

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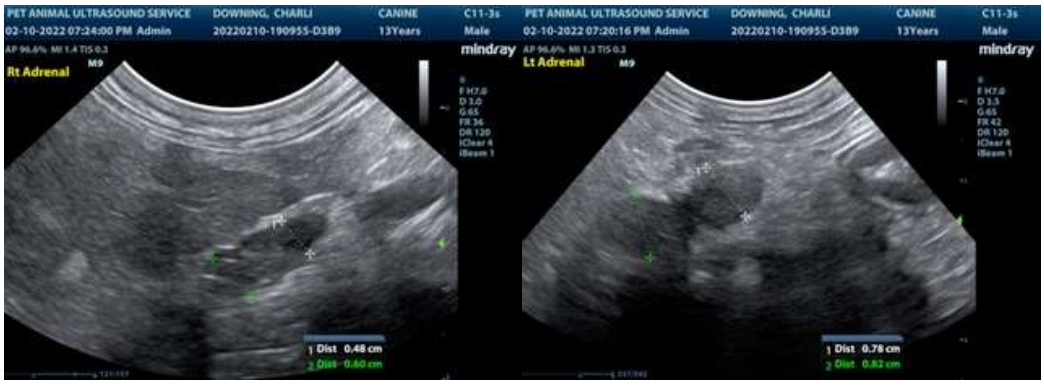


**The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.**

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.

**R. McKenzie Daniel, DVM, DABVP (Canine / Feline Practice)**
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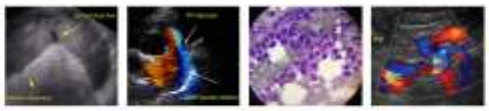
*http://www.sonopath.com/K9LiverDisease*

**Description:** The etiologic causes of canine hepatic disease are vast and varied. Some cases may progress fairly rapidly, while others will remain static for a considerable length of time or even eventually reverse. Regardless of the cause, management is crucial to maintaining and optimizing quality of life. If possible, practitioners should obtain and be guided by a pathologic diagnosis so they can administer a treatment attuned to the underlying disease and arrive at a more exact prognosis.

**Dietary Management:** A lower protein diet to support liver dysfunction should be initiated, especially in cases where hepatic encephalopathy is also present. Since dietary protein is low, the protein quality and bioavailability must conversely be high. It should be noted that a protein-restricted diet is not appropriate in all cases of hepatic disease, especially during the early phases, as protein restriction is unnecessary when there are no signs of significant hepatic dysfunction.

Therapeutic diets, such as Hill's® i/d® and Royal Canin® Hepatic™, are excellent choices and contain enhanced levels of nutrients such as, but not limited to:

- Branched chain amino acids, which bypass liver metabolism and are used directly for skeletal muscle accretion.
- Vitamin E, which helps minimize and reduce oxidative damage and stress from free radicals produced by stressed hepatocytes.
- Vitamin B complex, which helps drive intermediary metabolism.





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- Reduced copper.
- Extremely digestible protein sources with high biologic values, which help minimize the total amount of dietary protein needed and thus reduce blood ammonia levels.
- Carnitine, which helps drive fatty acids into the mitochondria for beta-oxidation and positive cellular energy balance.

**Medical Management:** The following list of medications is commonly used in the management of various hepatopathies or in the face of hepatic failure; however, each patient should be managed as an individual, and not all of the medications listed here are appropriate for each animal. One must always consider the definitive diagnosis of one's patient when developing a therapeutic plan. What follows is an outline of medical management recommendations for cholangiohepatitis and inflammatory hepatopathy/chronic hepatitis.

**Cholangiohepatitis**

*1. Broad-spectrum antibiotics*

a) Amoxicillin: Give 20 mg/kg BID or amoxicillin/clavulanic acid (13.75 mg/kg PO BID) for potential suppurative hepatitis. Options: ampicillin: 20 mg/kg IV TID; cephalexin: 20 mg/kg IV or PO TID; enrofloxacin: 2.5-5 mg/kg PO BID if cholangiohepatitis is present or to decrease ammonia production; gentamycin: 2 mg/kg TID IM or SC for 5-7 days if sepsis or peritonitis is present. Monitor renal function if aminoglycosides are utilized.

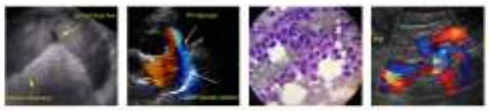
b) Metronidazole: Give 10-20 mg/kg BID in combination with amoxicillin/clavulanic acid or enrofloxacin for cholangiohepatitis because of its efficacy against anaerobic bacteria and/or for its immunomodulating effects. The dose is decreased to 7.5 mg/kg PO TID in the face of hepatic failure and/or encephalopathy. Controls ammonia production in the colon, decreases bacteria absorbed through portal circulation, and reduces cell-mediated immune responses (anti-inflammatory properties).

*2. Hepatic support*

a) S-adenosylmethionine (SAMe): Give 20 mg/kg/day PO on an empty stomach (1-2 hours before feeding). It is available in 90 mg tablets that are not to be broken. SAMe replenishes glutathione and aids in cellular detoxification; it also has anti-arthritic effects. SAMe is an anti-inflammatory and antioxidant. It also promotes hepatocellular regeneration and rectifies RBC membrane abnormalities in dogs with liver disease or oxidative damage.

b) Milk Thistle: Administer as silybin or silymarin extracts (a high-quality supplement is essential). Acts as an antioxidant and free radical scavenger; decreases hepatotoxin binding; improves glutathione concentrations; aids in iron chelating; and promotes choleresis. Give 5-15 mg/kg/day PO.

c) Ursodiol (Actigall): Give 10-15 mg/kg PO once daily, with food, to stimulate bile flow and decrease cholestasis. Tablets (250 mg) or capsules (300 mg) are available; however, ursodiol can





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also be compounded into a liquid to dose small patients. It has immunomodulatory, anti-fibrotic, and choleretic effects, anti-copper storage benefits, and stabilizes mitochondrial function.

d) Vitamin E: Must be coupled with good nutrition and other antioxidants to avoid accumulation of tocopheroxyl radicals. To that end, supplementation with SAMe may help ensure that adequate GSH (mitochondrial glutathione) concentrations are achieved. Give 10-15 IU/kg/day PO (100-400 IU) in a water-soluble form twice daily, as well as with Vitamin C 25 mg/kg/day.

e) Cobalamin and Thiamine (B12 and B1): Give 250ug SC weekly.

**Inflammatory hepatopathy/chronic hepatitis**

*1. Immunosuppressive agents*

a) Prednisone or prednisolone: Administer if inflammatory disease has been diagnosed by biopsy, beginning at 2 mg/kg/day for 2-4 weeks; subsequently reduce to 1 mg/kg/day. Once remission has been achieved, taper to 0.5 mg/kg/day (or to the lowest tolerable dose) over 2-4 weeks. Steroids may be discontinued if a different immunosuppressive medication is effective at controlling inflammation (i.e., azathioprine or cyclosporine) since they are contraindicated with hepatic encephalopathy. Possible negative sequelae of corticosteroids include increased water retention and potentiation of gastrointestinal ulceration. In the face of portal hypertension and ascites, dexamethasone is preferred—it does not exhibit mineralocorticoid activity and thus does not potentiate water retention as compared to prednisone—at 0.2-0.4 mg/kg orally once daily. Taper in a similar manner.

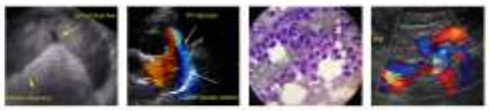
b) Azathioprine (Imuran): Give 50 mg/m²/day or 2 mg/kg/day as a long-term alternative to prednisone. The dose can be decreased to 1 mg/kg and eventually given every other day if there is a positive response. Check CBC and platelet count biweekly for the first 2 months and then monthly thereafter. Taper every 2-4 weeks to the lowest effective dose while monitoring transaminase levels. It can often be dosed on alternate days to prednisone. Possible negative side effects include bone marrow suppression and hepatic necrosis. Cyclosporine has been proposed as an alternative immunosuppressant in the management of chronic hepatitis and may allow one to cease concurrent steroid therapy; however, this has not been thoroughly investigated as of yet.

*2. Hepatic Support*

See medications listed in the previous section.

*3. Anti-fibrotics*

a) Colchicine: Give 0.03 mg/kg/day. Colchicine acts as an anti-inflammatory agent, stabilizes membranes, and stimulates collagenase production, thereby diminishing fibrosis. Colchicine should be used to treat hepatic fibrosis based on biopsy results; however, it can also be considered when ascites is present, and when hepatic fibrosis and cirrhosis are highly suspected based on sonographic appearance and clinical findings. It can result in adverse effects, including vomiting, diarrhea, and inappetance. Discontinue until clinical signs resolve, and reinstitute at a lower dose and up-titrate slowly.





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*4. Hepatic Encephalopathy*

a) Lactulose: Give 0.5 ml/kg orally 2-3 times daily to soften the stool. It helps manage hepatic encephalopathy by combining with ammonium in the GI tract and thus decreasing circulating ammonia levels. Use in conjunction with low dose metronidazole. Lactulose can also be given as a retention enema in an encephalopathic crisis.

b) Metronidazole: Give at 7.5 mg/kg PO TID. Neomycin is an alternative and can be administered at 22 mg/kg PO BID-TID.

c) L-Carnitine: Give 200-400 mg/day. 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.

*5. Copper Chelation*

Use chelation when copper toxicity has been documented on biopsy and quantification has been performed to confirm toxic levels.

a) D-penicillamine: Give 10-15 mg/kg PO BID on an empty stomach. This is a copper chelator and should only be used based on a quantitative analysis of copper. Possible side effects include vomiting and inappetance. Do not give in conjunction with zinc.

b) 2,3,2 Tetramine (Syprine, Cuprid): Give 5-7 mg/kg PO BID on an empty stomach (1-2 hours before eating). An alternative to D-penicillamine for those dogs that are intolerant.

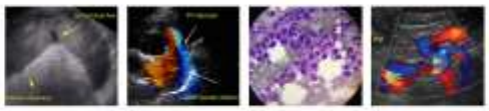
c) Zinc gluconate, acetate, or sulfate (acetate is best tolerated): Give 15-10 mg/kg elemental zinc divided BID for 2-6 months as a loading dose. Administer on an empty stomach (30-60 minutes before eating). Reduce to half the dose during the maintenance phase. A low copper diet is preferred (i.e., therapeutic diets, such as Hill's l/d® or Royal Canin® Hepatic™, are advisable). Zinc binds with intestinal copper to avoid absorption in the gastrointestinal tract and may be used alone in mild cases of copper toxicity. The goal is to reach zinc serum levels of 200-600 ug/dl; levels should initially be measured every 2-3 months. Give this medication on an empty stomach or with tuna fish to avoid vomiting. Zinc is not as effective as D-penicillamine and is only used in mild cases. It is not used in conjunction with D-penicillamine.

*6. Portal Hypertension and Ascites*

a) Spironolactone: If ascites is present secondary to portal hypertension, spironolactone can be dosed at 1-2 mg/kg PO BID; it is the diuretic of choice. Alternatively, spironolactone can be used in conjunction with furosemide (0.5-1 mg/kg PO BID) or hydrochlorothiazide; one should administer 1 mg/kg PO BID if given in conjunction with another diuretic. Monitor renal function and electrolytes diligently.

b) Famotidine: Give 0.5 mg/kg PO BID in cases of portal hypertension that result in gastrointestinal bleeding/melena.

**General Notes on Therapeutic Management:** Given that a primary function of the liver is to metabolize oral medications via the portal system (first past effect), numerous medications may





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result in higher systemic exposure to parent compounds in the face of hepatic insufficiency or failure. Drugs that are inactivated by the liver, produce hepatic damage, or require hepatic metabolism should be avoided. These include: lincomycin, clindamycin, streptomycin, chloramphenicol, sulfonamides, erythromycin, hetacillin, phenobarbital, diazepam, oxy- or chloro-tetracyclines, azole antifungals, nonsteroidal anti-inflammatory drugs (NSAIDs), theophylline or chloramphenicol, combinations of cimetidine and metronidazole, and combinations of enrofloxacin and theophylline or cisapride. In cases of hepatic lipidosis, glucocorticoids, anabolic steroids, and lipotrophic agents containing methionine should be avoided as they result in the production of encephalopathic toxins (metacarpans). Glucocorticoids are indicated for cholangitis, but only after lymphoma and hepatic lipidosis have been ruled out.

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