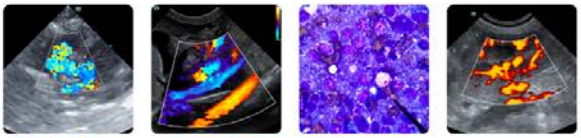

PATIENT

Coco Horn

SPECIES

Canine

BREED

Labrador Retriever

SEX

Spayed Female

AGE

5 Years 5 Months

WEIGHT

86.3 lbs

INTERPRETED BY

Eric Lindquist, DMV, DABVP (CFM), Cert. IVUSS

IMAGING PERFORMED BY

Chloe Lowe, CVT

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PRESENTING CLINICAL SIGNS

Increasing Alt since Jan 2025. Normalpet clinically, worried about Copper Storage Disease. Tried amoxi/metro/denamarin with no reduction in Alt last year. Currently on Hills L/D diet.

Abnormal PE/Chem/CBC/UA Results: Alt 1047, Alp 349

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 pelvic urethra was imaged 2.0 cm beyond the cystourethral junction.

The iliac trifurcation was unremarkable.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. Left kidney measures 6.5 cm. Right kidney measures 6.0 cm.

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. Right measured 2.07 cm x 1.5 cm at the cranial pole and 0.67 cm at the caudal pole. Left measured 1.75 cm x 0.34 cm at the cranial pole and 0.41 cm at the caudal pole.

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** presented mild coarse architecture with slight increased portal markings. Changes were fairly mild to moderate as far as level of remodeling. The gallbladder and common bile duct were unremarkable. No evidence of post-hepatic obstruction.

Gastrointestinal

A minor amount of non-shadowing, non-obstructive ingesta was noted in the **stomach**. Transit of chyme into the small intestine was normal. Curvilinear patterns were maintained throughout the GI tract. No evidence of pathology. 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

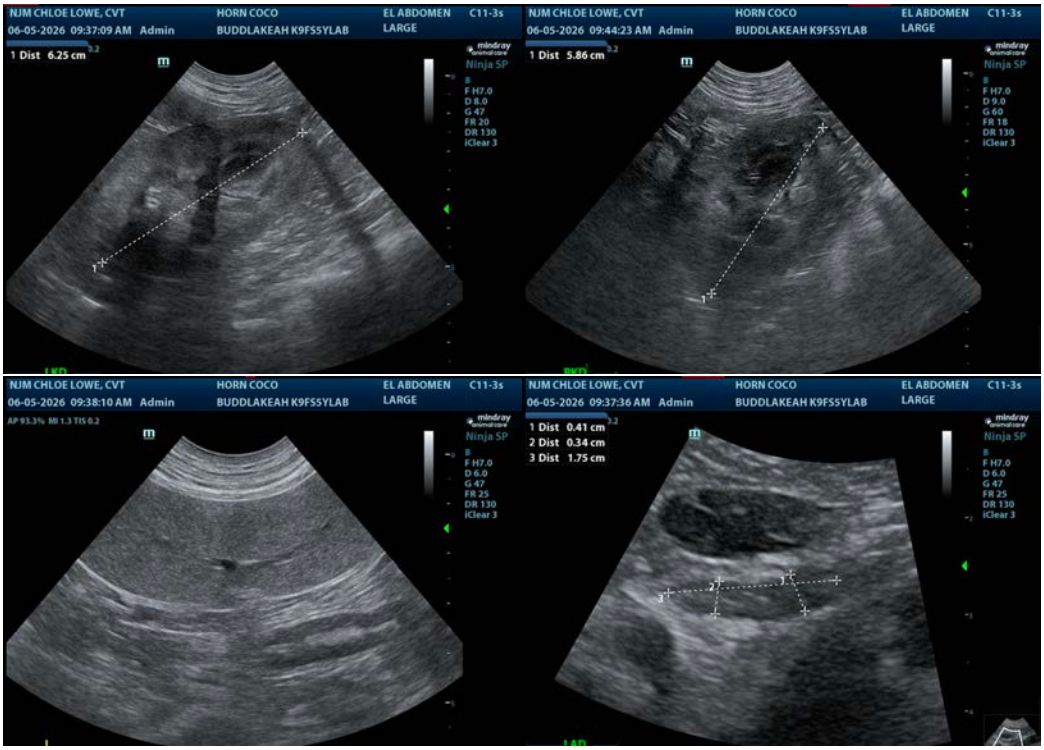
The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

ULTRASONOGRAPHIC FINDINGS

- Non-specific mild chronic inflammatory hepatopathy.
- Partially full stomach.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Leptospirosis titers indicated. Bile acid profile indicated. Core liver biopsy would be ideal to assess for structural changes and submit for copper storage disease, which is a potential. FNA would allow to assess inflammatory cell type.



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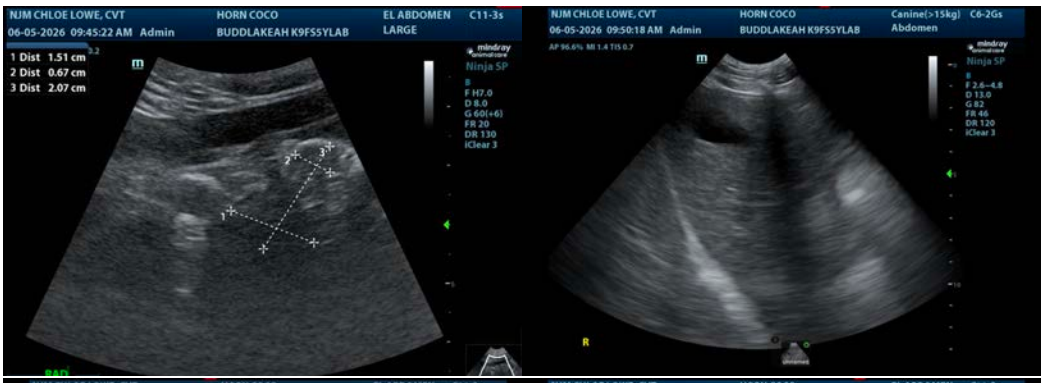
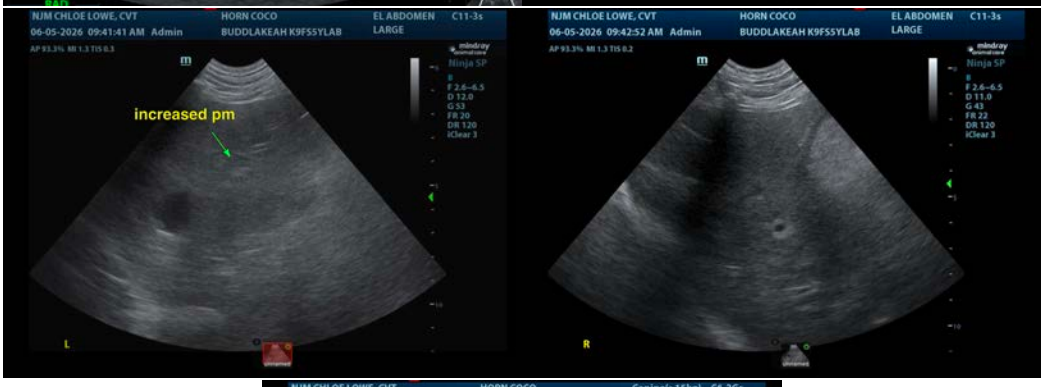
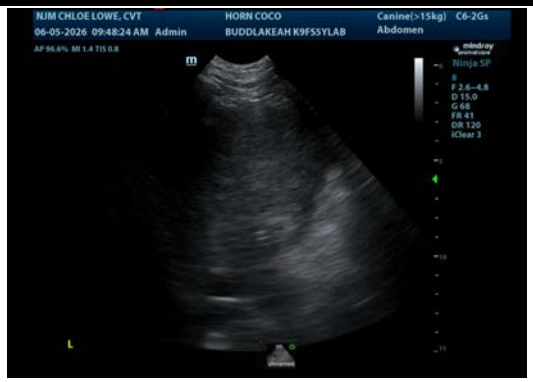


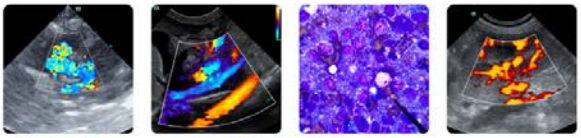




The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Eric Lindquist, DMV, DABVP(CFM), Cert. IVUSS,
CEO, Owner, Founder -- SonoPath.com
info@SonoPath.com



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Excerpt from the Curbside Guide: https://sonopath.com/thecurbsideguide/

Canine Liver Disease

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 eventually even 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 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. However, a lower protein diet to support liver dysfunction should be initiated in cases where hepatic encephalopathy is present. Since dietary protein is low, the protein quality and bioavailability conversely must be high.

Therapeutic diets, such as Hill's® L/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
- 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 β-oxidation and positive cellular energy balance

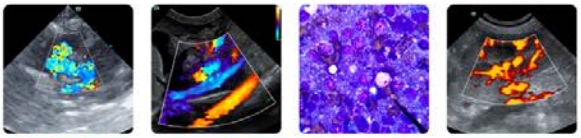
MEDICAL MANAGEMENT The following list of medications is commonly used in the management of various hepatopathies or in the face of hepatic failure; however, each patient should be managed as an individual, and not all of the medications listed here are appropriate for each animal. One must always consider 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

I. 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. This controls ammonia production in the



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colon, decreases bacteria absorbed through portal circulation, and reduces cell-mediated immune responses (anti-inflammatory properties).

II. 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 also be compounded into a liquid to dose small patients. It has immunomodulatory antifibrotic 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 250 µg SC weekly.

INFLAMMATORY HEPATOPATHY/CHRONIC HEPATITIS

I. Immunosuppressive agents

A. 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 as it does not exhibit mineralocorticoid activity and thus does not potentiate water retention as compared to prednisolone. Dose is 0.2–0.4 mg/kg orally once daily. Taper in a similar manner.

B. Azathioprine (Imuran): Give 50 mg/m^2/day or 2 mg/kg/day as a long-term alternative to prednisolone. The dose can be decreasedto 1 mg/kg and eventually given every other day if there is a positive response. Monitor CBC and platelet count biweekly for the first two 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. An alternative immunosuppressant would be cyclosporine which may allow one to cease concurrent steroid therapy.

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See medications listed in the previous section.

III. 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 such as vomiting, diarrhea, and inappetence. Discontinue until clinical signs resolve, reinstitute at a lower dose, and slowly titrate up.

IV. 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: 200–400 mg/day as it is normally synthesized by the liver. L-Carnitine enhances ammonia elimination and is indicated in cases of hepatic encephalopathy and lipidosis.

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

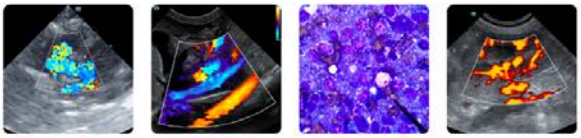
A. D-penicillamine: Give 10–15 mg/kg PO BID on an empty stomach. This is a copper chelator and should only be used based on a quantitative analysis of copper. Possible side effects include vomiting and inappetence. Do not give in conjunction with zinc.

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 10–15 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 µg/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.

VI. 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.



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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 pass effect), numerous medications may 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, NSAIDs, theophylline or chloramphenicol, combinations of cimetidine and metronidazole, and combinations of enrofloxacin and theophylline or cisapride. In cases of hepatic lipidosis, glucocorticoids and anabolic steroids should be avoided as they can exacerbate the hepatic lipidosis as they have an inhibitory influence on mitochondrial matrix acyl-CoA dehydrogenases and ß-oxidation as well as inducing micro-vesicular steatosis and ultrastructural mitochondrial lesions. Glucocorticoids are indicated for cholangitis but only after lymphoma and hepatic lipidosis have been ruled out.

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