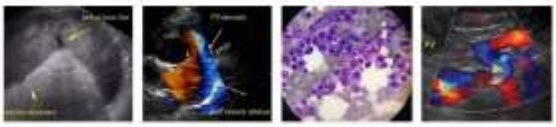

PATIENT

Rosie Springett

SPECIES

Canine

BREED

Bichon

SEX

FS

AGE

13 Years

WEIGHT

5.7kg

INTERPRETED BY

R. McKenzie Daniel, DVM, DABVP

IMAGING PERFORMED BY

Crystall Hill

HOSPITAL NAME

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PRESENTING CLINICAL SIGNS

Jaundiced and unwell. Not really eating. Depressed. Currently on Metacam and Cerenia.
Abnormal PE/Chem/CBC/UA Results: Please see attached bloodwork.

CBC hematocrit 36.4, WBC 20.9 with neutrophilia, minor lymphopenia monocytosis, platelets 702

Chem panel BUN 11.4, Creatinine 146, SDMA 20, Globulin 51, ALT 1849, ALP greater than 2000,

GGT 79, Total bilirubin 180

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder presented uniformly thickened urinary bladder wall isoechoic to the adjacent normal urinary bladder wall. The luminal margin of the thickened urinary bladder wall was mildly asymmetrical in contour. The ventral apical urinary bladder wall thickness measured 0.45 cm width. Mild dependent mineral was present. Potential for focal areas of adhered mineral along the mildly thickened apical luminal wall noted. The urinary bladder, trigone, cystourethral junction, and visible pelvic urethra exhibited normal 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 pathology in the area of the aortic trifurcation.

Normal size and margination was 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. Focal areas of nonobstructive medullary mineral were present. No evidence of pelvic dilation was present. The left kidney measured 3.7 cm in length. The right kidney measured 4.3 cm in length.

Adrenal Glands

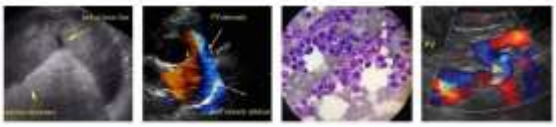
The left adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The left adrenal gland measured 1.8 cm length x 0.58 cm width at the caudal pole. The right adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The right adrenal gland measured 1.4 cm length x 0.60 cm width at the caudal pole. No evidence of hyperplasia or tumors.

Spleen

The spleen exhibited primarily finely textured and homogenous parenchyma which was hyperechoic to the liver and renal cortical parenchyma. Intermittent, variably sized, yet non-expansive echogenic nodules were present throughout the cranial to caudal parenchyma. An example of a splenic nodule measured 0.67 cm width. 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 or neoplastic changes were not noted. The echogenic nodules tend to trend benign and are most consistent with benign hyperplasia or myelolipomas.

Liver

The liver was subjectively normal in size. Normal overall hepatic parenchyma echogenicity with moderate course echotexture and area of increased portal vascular border prominence. Symmetrical hepatic contour was primarily maintained. No hepatic masses or nodules. The hepatic and portal vasculature were normal in appearance without signs of congestion. The gallbladder was non-distended in size with thin walls and primarily anechoic luminal content. The cystic and common bile ducts were normal.



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Gastrointestinal

The stomach presented mildly prominent yet intact wall layering owing to mild gastric mucosal hypertrophy. Mild to moderate retained anechoic fluid was present along with areas of luminal gas. No evidence of retained ingesta, fluid, foreign material, or overt mechanical pyloric outflow obstruction. The pylorus wall measured 0.50 cm width.

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 measured 0.34 cm width. The duodenum wall measured 0.35 cm width.

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

Pancreas

The parenchyma of the left limb, body and right limb of the pancreas presented isoechoic to the adjacent omental fat. A normal curvilinear capsule contour of the pancreas was present. The visible pancreatic duct was normal. No signs of active inflammation or neoplastic disease was evident.

Free Abdomen

No overt lymphadenopathy or peritoneal effusion was present.

ULTRASONOGRAPHIC FINDINGS

- Cystitis pattern with mild luminal mineral.
- Benign splenic nodules - probable benign myelolipomas, nodular hyperplasia, previous infarct, or emerging mineralization possible.
- Hepatopathy with sonographically unremarkable gallbladder and common bile duct.
- Gastritis pattern with gastric stasis.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The overall appearance of the liver was nonspecific yet not overly consistent with neoplastic criteria. Acute on chronic hepatitis (viral, bacterial, leptospirosis, immune mediated, hepatotoxicosis) vacuolar hepatopathy with nonobstructive cholestasis possible with occult hepatic neoplasia considered a less likely differential diagnosis. Further assessment may include hepatic FNA for screening cytology assuming normal clotting status and leptospirosis titers/pcr if clinically indicated.

Further renal staging to include urine C/S and protein: creatinine ratio on sterile urine sample may be considered.

Hospitalization with hepatic support and broad spectrum antibiotics with potential coverage for leptospirosis and as needed gastrointestinal support recommended. Recheck sonographic may be considered pending clinical response to medical therapy or if persistent to increasing hepatic enzymes are noted despite therapy.

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Clinical Sonography & Telecytology
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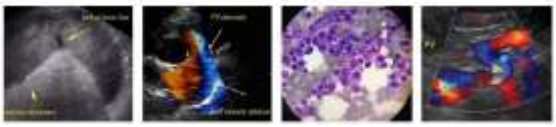

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289-808-2608 info@thefocalzone.com

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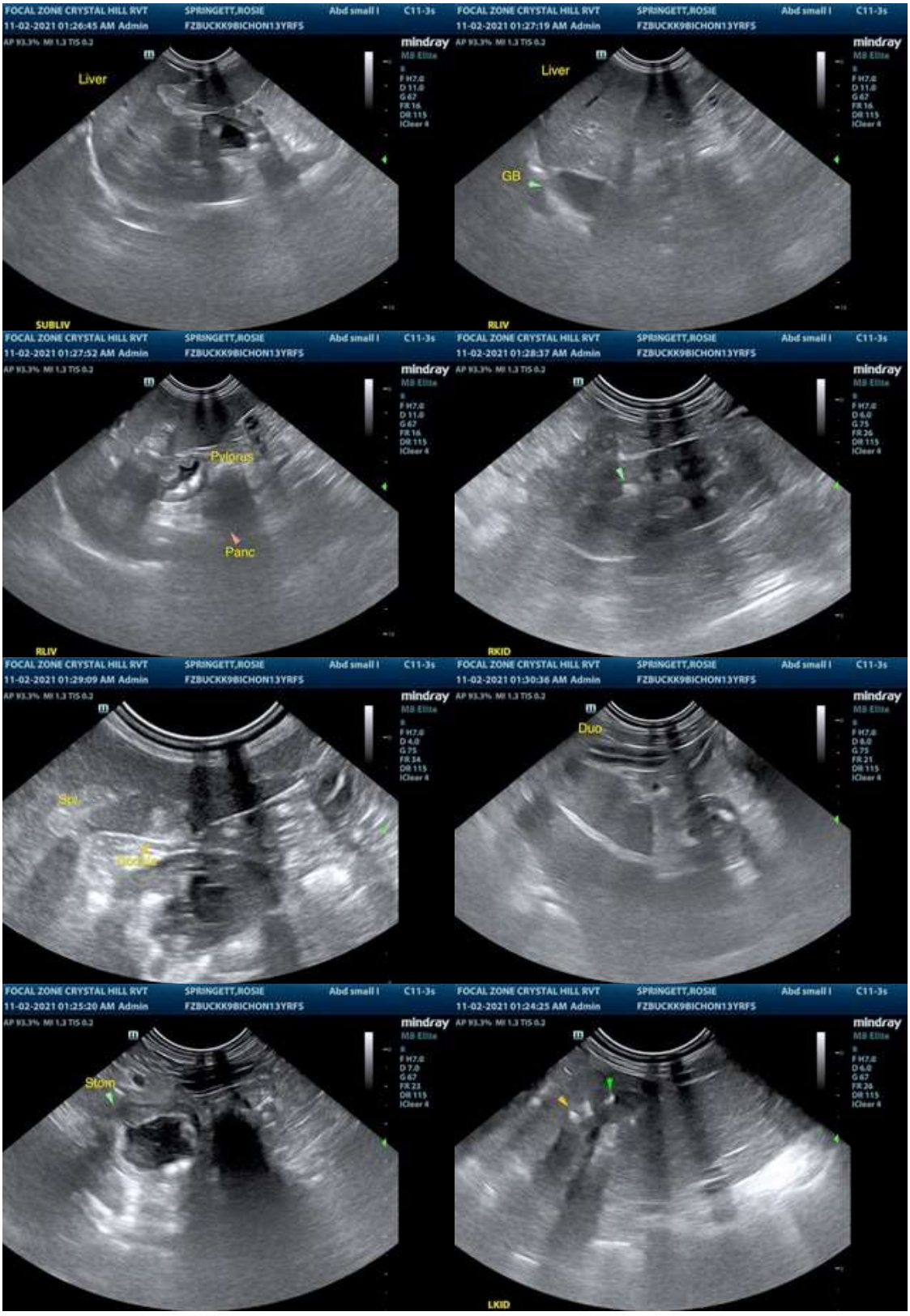

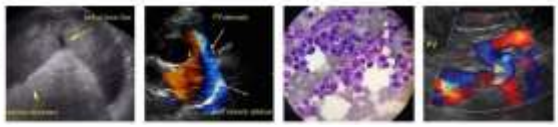

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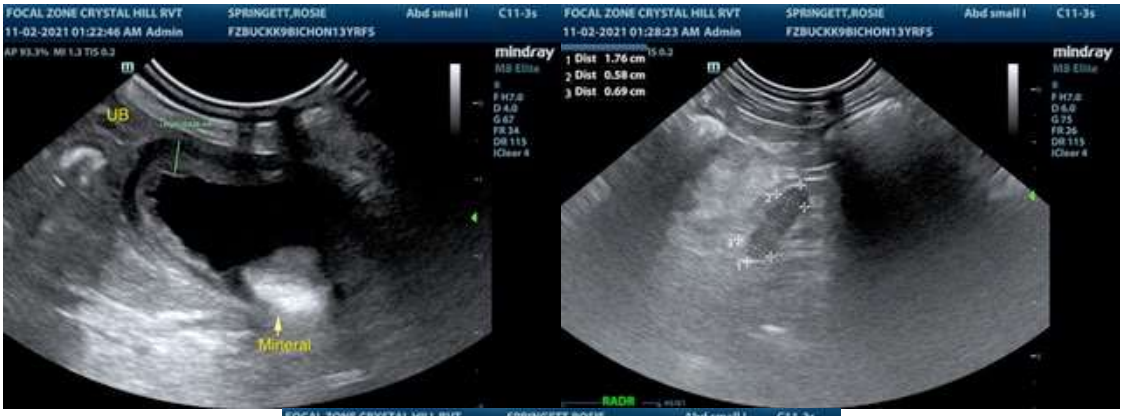

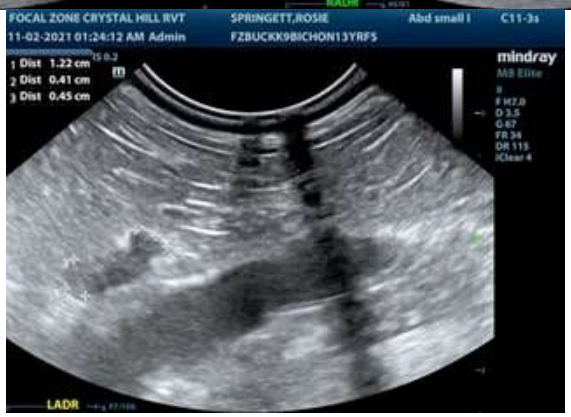

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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

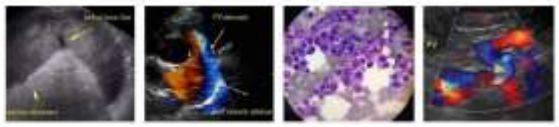
Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

R. McKenzie Daniel, DVM, DABVP (Canine / Feline Practice)

info@SonoPath.com

The following is an applicable excerpt from the *Curbside Guide to Diagnosis & Treatment of Sonographic Disease* offered by SonoPath.com Lindquist, Frank, L and Modler.

An essential quick guide for every general practitioner and sonographer.



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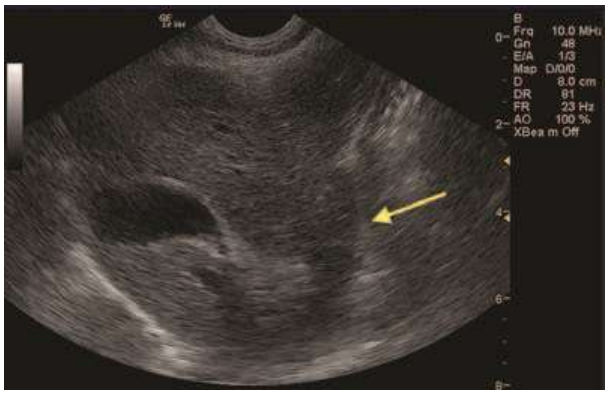
https://sonopath.com/products/curbside-guide-editing-due-release-12012015

Canine Liver Disease & Treatment Recommendations

http://www.sonopath.com/K9LiverDisease



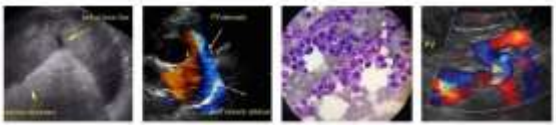
Long axis image of the liver showing a slightly echogenic and thickened gall bladder with mild coarsely echogenic portal markings. The parenchyma is hypoechoi to falciform fat in the near field and mild lobar swelling (arrow) is noted suggestive for an acute process. Diagnosi Acute on early chronic cholangiohepatitis. The patient wa leptospirosis positive and responded to Ampicillin therapy

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.
- Reduced copper.



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- 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.

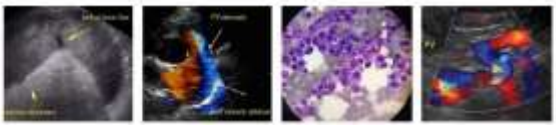
b) Metronidazole: Give 10-20 mg/kg BID in combination with amoxicillin/clavulanic acid or enrofloxacin for cholangiohepatitis because of its efficacy against anaerobic bacteria and/or for its immunomodulating effects. The dose is decreased to 7.5 mg/kg PO TID in the face of hepatic failure and/or encephalopathy. 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 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.



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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.

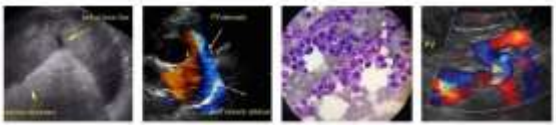
2. Hepatic Support

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.

4. Hepatic Encephalopathy



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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.

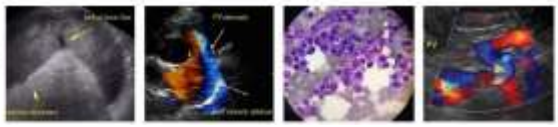
c) Zinc gluconate, acetate, or sulfate (acetate is best tolerated): Give 15-10 mg/kg elemental zinc divided BID for 2-6 months as a loading dose. Administer on an empty stomach (30-60 minutes before eating). Reduce to half the dose during the maintenance phase. A low copper diet is preferred (i.e., therapeutic 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 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



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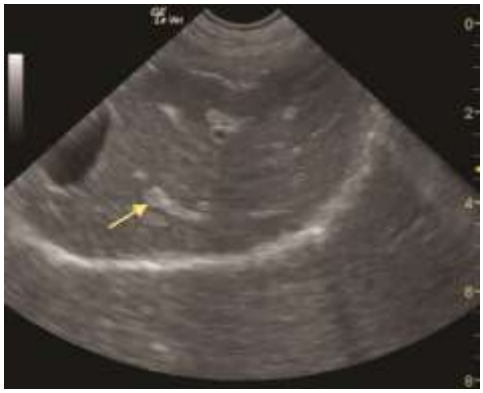
combinations of enrofloxacin and theophylline or cisapride. In cases of hepatic lipidosis, glucocorticoids, anabolic steroids, and lipotrophic agents containing methionine should be avoided as they result in the production of encephalopathic toxins (metacarpans). Glucocorticoids are indicated for cholangitis, but only after lymphoma and hepatic lipidosis have been ruled out.



Short axis of the liver in a dog with leptospirosis and chronic hepatitis. Note the overall increase in echogenicity typically seen in chronic disease. Multifocal hyperechoic patches and increased portal markings (arrow) are present.

References:

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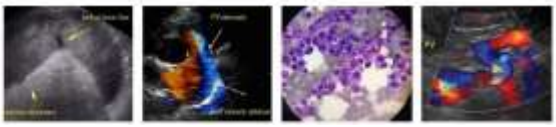

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Hackett T, Twedt D, Gustafson D. Milk thistle and its derivative compounds: a review of opportunities for treatment of liver disease. *J Vet Intern Med* 2013;27(1):10-16.

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