



PATIENT

Rocky Demott

SPECIES

Canine

BREED

Yorkshire Terrier

SEX

MC

AGE

14 years 2 months

WEIGHT

9.6

INTERPRETED BY

Beth Johnson, DVM
DACVIM

IMAGING PERFORMED BY

Christensen

HOSPITAL NAME

Tranquility VC

REFERRING VET

Dr. House

INVOICE

11462

DATE

3/11/2026

PRESENTING CLINICAL SIGNS

- Chronic proteinuria dog. UPC increase to 5.5 after recent diet change to Hills PD multi-organ.
- History of IBD.
- Current medications include Telmisartan 10mg - 1 tab PO SID, Apoquel 5.4mg - 1/2 tab PO SID, Metronidazole 50mg - 1 tab PO SID, Budesonide 1mg - 1 tab PO SID.

Abnormal PE/Chem/CBC/UA Results: Diagnostic abnormalities: _UPC 5.5, SDMA 16, lymphs 0.8 K/ul, eos 0.04 K/uL, otherwise CBC/Chem/T4/4DX/OP wnl. BP= 168/127, MAP= 134.

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder is adequately distended with anechoic contents. No masses, inflammatory changes, echogenic sediment or cystoliths are observed. The urinary bladder, trigone and visible pelvic urethra are normal in thickness with a smooth mucosal surface.

The area of the prostate is examined without evident prostatic pathology.

Kidneys are bilaterally normal in size, irregular and diffusely echogenic with decreased corticomedullary distinction and poor visualization of internal architecture. There is no mineral observed. Left kidney measures 3.54 cm and contains mild pyelectasia. Right kidney measures 3.57 cm.

Adrenal Glands

The adrenal glands are unable to be visualized.

Spleen

Spleen is subjectively normal in size with a normal smooth capsular contour. Parenchyma is appropriately finely textured and homogenous with normal echogenicity relative to surrounding tissue (hyperechoic to liver). Multifocal well-demarcated hyperechoic homogenous nodules are noted. Splenic vasculature appears normal.

Liver

Liver is subjectively enlarged with mildly irregular margins. Parenchyma is mildly heterogenous characterized by multiple poorly defined hypoechoic nodules within otherwise hyperechoic liver parenchyma. Visible vasculature and biliary tree appear normal without distension or congestion.

Gallbladder is moderately distended with anechoic bile as well as suspended and gravity dependent echogenic debris. The wall is smooth without visible thickening. There is no evidence of cystic or CBD dilation. There is no evidence of effusion or inflammation.

Gastrointestinal

The visible stomach wall is normal in thickness and layering. The lumen of the stomach is empty with no evidence of obstruction, foreign material or infiltrative disease. Pyloric outflow tract appears patent.



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The visible small intestine demonstrates areas of mildly thick muscularis layer relative to mucosa (disruption of the normal 1:3 muscularis:mucosa ratio). Small intestinal submucosa is slightly irregular, thick and hyperechoic, without evident loss of layering appreciated. The lumen of the small intestine is mildly distended with very echogenic reverberation artifact from intraluminal gas. There is no evidence of obstruction, foreign material, or infiltrative disease; however, visualization is partially inhibited by gas.

The visible colon is normal in wall thickness (< 0.2 cm) and layering. Contents are consistent with normal formed feces and gas.

Pancreas

The observed pancreas appears appropriately isoechoic to surrounding omental fat. The capsule is mildly irregular in shape. Parenchyma is mildly heterogenous and coarse. There is no visible pancreatic duct dilation. There is no evidence of active peripancreatic inflammation.

Free Abdomen

There is no visible free peritoneal effusion noted in these images.

There is no apparent pathologic lymphadenopathy noted in these images.

PRIMARY FINDINGS

- Mild inflammatory bowel disease (IBD) pattern – Thick muscularis has been reported with infiltrative bowel disease including both benign inflammatory disease as well as infiltrative neoplasia such as lymphoma. No loss of layering or distinct characteristics of malignancy are present. Therefore, differentials cannot be further ranked without tissue sampling.
- Pancreatic age-related remodeling/Chronic pancreatitis – Mild irregularities are consistent with benign age-related change. Low-grade smoldering chronic pancreatitis cannot be ruled out and should be suspected in the face of appropriate clinical signs.
- Mildly heterogenous liver – These changes are most consistent with benign processes such as nodular hyperplasia, steroid (vacuolar) hepatopathy, extramedullary hematopoiesis or possibly chronic inflammatory disease and less commonly infiltrative round cell or metastatic neoplasia.
- Moderate gallbladder debris - Cholecystic debris is of unknown clinical significance. It can be seen with biliary stasis from fasting or illness. Cholecystic debris is not necessarily related to hepatobiliary disease. Echogenic bile is most commonly an incidental finding in dogs and should be interpreted in combination with clinical signs such as nausea, inappetence, cranial abdominal discomfort and/or laboratory changes such as increased ALP and/or increased Tbili.
- Mild to moderate chronic kidney disease changes bilaterally with mild left sided pyelectasia.

SECONDARY FINDINGS

- Hyperechoic splenic nodules – most consistent with benign myelolipomas. Other differentials such as fibrosis or calcification caused by old hematomas or infarcts, chronic inflammation, granulomatous disease or metastatic disease cannot be ruled out, but are considered less likely.



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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Given patient's GI and pancreatic changes described above, combined with patient's history, if not recently evaluated, a gastrointestinal malabsorption panel (including cobalamin, folate, TLI and PLI) to Texas A&M GI Laboratory is recommended for further evaluation of GI and pancreatic function.

A fecal enteropathogen PCR panel to Texas A&M GI Laboratory could be considered for further evaluation of possible infectious disease. Contact lab for recommendations on how long to discontinue antibiotics (if indicated) prior to obtaining a stool sample for submission.

In the meantime, continued evaluation for possible contributing underlying causes to patient's reported proteinuria is recommended. If none are found, progressive protein losing nephropathy or kidney disease could be the cause and more aggressive medical management may be indicated. Full consultation with, and/or referral to a veterinary internist could be considered.



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.



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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.

Beth Johnson, DVM, DACVIM
info@sonopath.com