

**DATE PRESENTING CLINICAL SIGNS**

2/23/22

Pet has a history of hypoalbuminemia and PLE. Pet managed well on Atopica. Pet was seen for routine exam and bloodwork. At this time hypoalbuminemia was noted again and pet on PE did have a pendulous abdomen. Pet also tested positive for Lyme which was a new finding. After bloodwork results were received noting the hypoalbuminemia, owner was instructed to increase Atopica from EOD dosing to SID until Lyme Quant could be received and to recheck Albumin in 2 weeks time. Once these results were received owner was instructed to return to EOD dosing of the Atopica and start Doxycycline but via voice message. Owner never received this message and only came in for recheck Albumin 2 weeks later. With the new abnormalities, AUS was recommended to better assess pet.

PATIENT

Millie Harris

SPECIES

Canine

BREED

Yorkie

SEX

Spayed Female

AGE

12/20/10

WEIGHT

13 Pounds

INTERPRETED BYBeth Johnson, DVM
DACVIM**IMAGING PERFORMED BY**

Rachel Brilhart RDMS

HOSPITAL NAME

Westminster VH

REFERRING VET

Dr. Hall

INVOICE

35847

Current Medications: Atopica 25mg EOD started 2/2020, Doxycycline 40mg in AM and 20mg in PM started 2/17/22. Gabapentin 50mg PO upon arrival to the hospital
Lab Results: 2/2/22 Reticulocytes 142 (10-110), Eosinophils 1.699 (0.0791.49), Potassium 5.8 (4-5.4), Alb 2.3 (2.7-3.9), Lipase 276 (0-250), CK 342 (10-200). UA- USG 1.034, pH 8.5, Protein 4+, Ketones trace, WBC 2-5/hpf, RBC 15-20/hpf. TT4 1.7 (1.0-4.0), Lyme Quant C6 164. 2/10/22- fecal negative. 2/16/22 HCT 57.6% (38.3-56.5), Reticulocytes 143K (10-110), Platelets 612 (143-448), Alb 2.2 (2.7-3.9, ALT 316 (18-121).
Awaiting urine sample from home for UPC- will forward these when available.
Date of Previous IntraPet Ultrasound: No previous.
Sedation: Not required to complete full diagnostic ultrasound.
Stat Report: Not requested.

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN**Urinary System**

The urinary bladder is moderately 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 right kidney is normal in size (4.76 cm) and shape with smooth peripheral margination. A normal 1:3 cortex to medulla ratio is maintained. The medulla and cortices are uniform in texture with some mild increased echogenicity and mild loss of corticomedullary distinction. There is no evidence of pyelectasia, mineral or infarcts observed.

The left kidney is normal in size (4.34 cm) and shape with smooth peripheral margination. A normal 1:3 cortex to medulla ratio is maintained. The medulla and cortices are uniform in texture with some mild increased echogenicity and mild loss of corticomedullary distinction. There is no evidence of pyelectasia, mineral or infarcts observed. There is a small renal cortical cyst in the caudal pole.

Adrenal Glands

The right adrenal gland is normal in size (1.4 cm long x 0.41 cm at the cranial pole and 0.59 cm at the caudal pole), shape and contour. Corticomedullary structure is unremarkable. Visible surrounding vasculature appears normal.

The left adrenal gland is normal in size (1.8 cm long x 0.40 cm at the cranial pole and 0.78 cm at the caudal pole), shape and contour. Corticomedullary structure is unremarkable. Visible surrounding vasculature appears normal.

Spleen

Spleen is subjectively enlarged in size with rounded margins but intact capsule. Parenchyma is homogeneously coarse/heterogeneous in echotexture, characterized by multifocal discrete hypoechoic nodules of varying sizes, ranging from 0.4 cm to the largest mid body, non-capsule disrupting nodule measuring 1.2 cm, and normal to hypoechoic in echogenicity. Splenic vasculature appears normal.

Liver

Liver is subjectively enlarged. Margins are smooth but round. It has a normal homogenous echotexture. Parenchyma is diffusely hyperechoic characterized by less prominent than normal portal vein walls and increased echogenicity relative to the spleen. No focal lesions are observed. Visible vasculature and biliary tree appear normal without distension or congestion.

The gallbladder is non-distended in size. The wall is smooth without visible thickening. Luminal contents are primarily anechoic. There is no evidence of cystic or common bile duct dilation.

Gastrointestinal

The stomach wall is normal in thickness (canine < 0.5 cm and feline < 0.4 cm) and layering. The lumen of the stomach is empty with no evidence of obstruction, foreign material or infiltrative disease. Pyloric outflow tract appears patent.

The small bowel is normal in thickness with a relatively thick mucosa compared to other layers. Normal wall layering is preserved; however, the mucosa is more echogenic than normal and contains hyperechoic striations perpendicular to the lumen. The lumen of the small intestine is empty with no evidence of obstruction, foreign material or infiltrative disease.

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

Pancreas

The pancreatic parenchyma is appropriately isoechoic to surrounding tissue. Visible capsule is smooth and normal in contour. There is no visible pancreatic duct dilation. There is no evidence of active peripancreatic inflammation.

Free Abdomen

There is no evidence of peritoneal effusion. There is a prominent, hypoechoic hepatic/portal lymph node measuring 0.84 cm x 1.6 cm.

ULTRASONOGRAPHIC FINDINGS

- Nodular splenomegaly – Can be seen with diffuse infiltrative disease including both benign conditions such as extramedullary hematopoiesis, lymphoid hyperplasia, as well as infiltrative neoplastic disease such as round cell neoplasia.
- Hyperechoic hepatomegaly – most consistent with benign steroid (endocrine) hepatopathy or reactive or idiopathic hepatopathy. Infiltrative neoplasia such as round cell neoplasia is also possible, but considered less likely.
- Lymphangiectasia - Small bowel findings are most consistent with lacteal dilation. These findings can be observed with protein-losing enteropathies caused by either primary lymphangiectasia or primary infiltrative inflammatory disease with secondary lymphangiectasia. Infiltrative neoplasia is possible but considered less likely. Histopathology is necessary to definitively determine underlying cause.
- Age related kidney change – This finding is expected/consistent with age-related mild degenerative disease and should be interpreted clinically in combination with laboratory changes.
- Incidental renal cortical cyst left kidney.

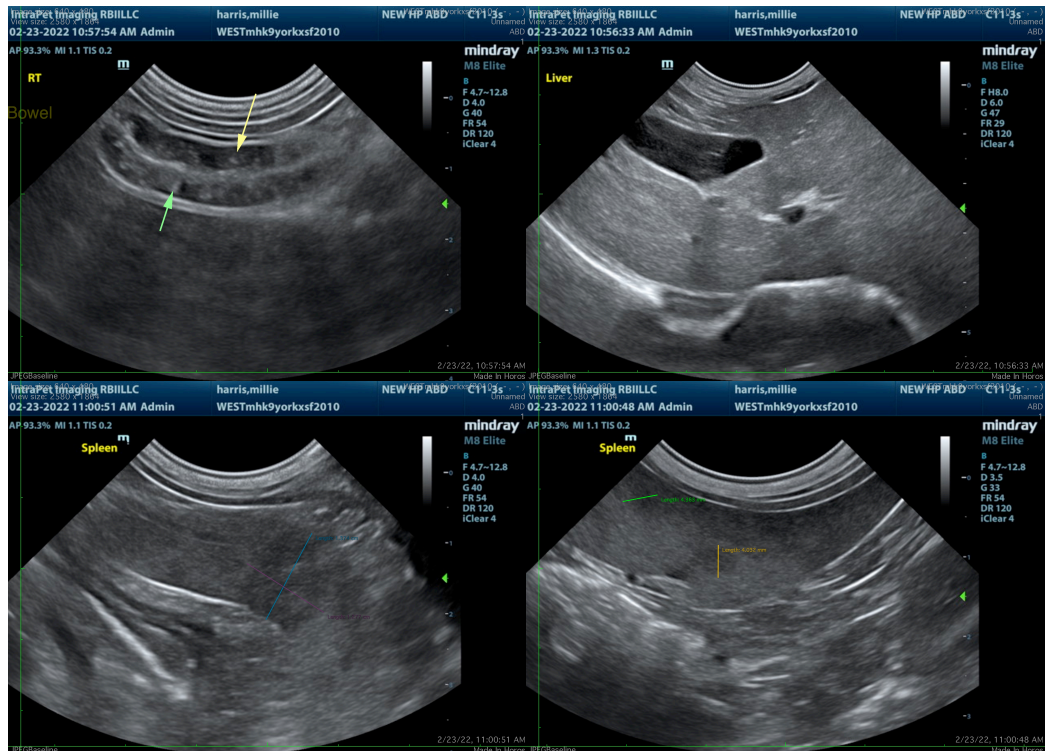
- Prominent hypoechoic hepatic lymph node - Consistent with reactive lymphadenopathy. Infiltrative neoplasia cannot be ruled out but is considered less likely.

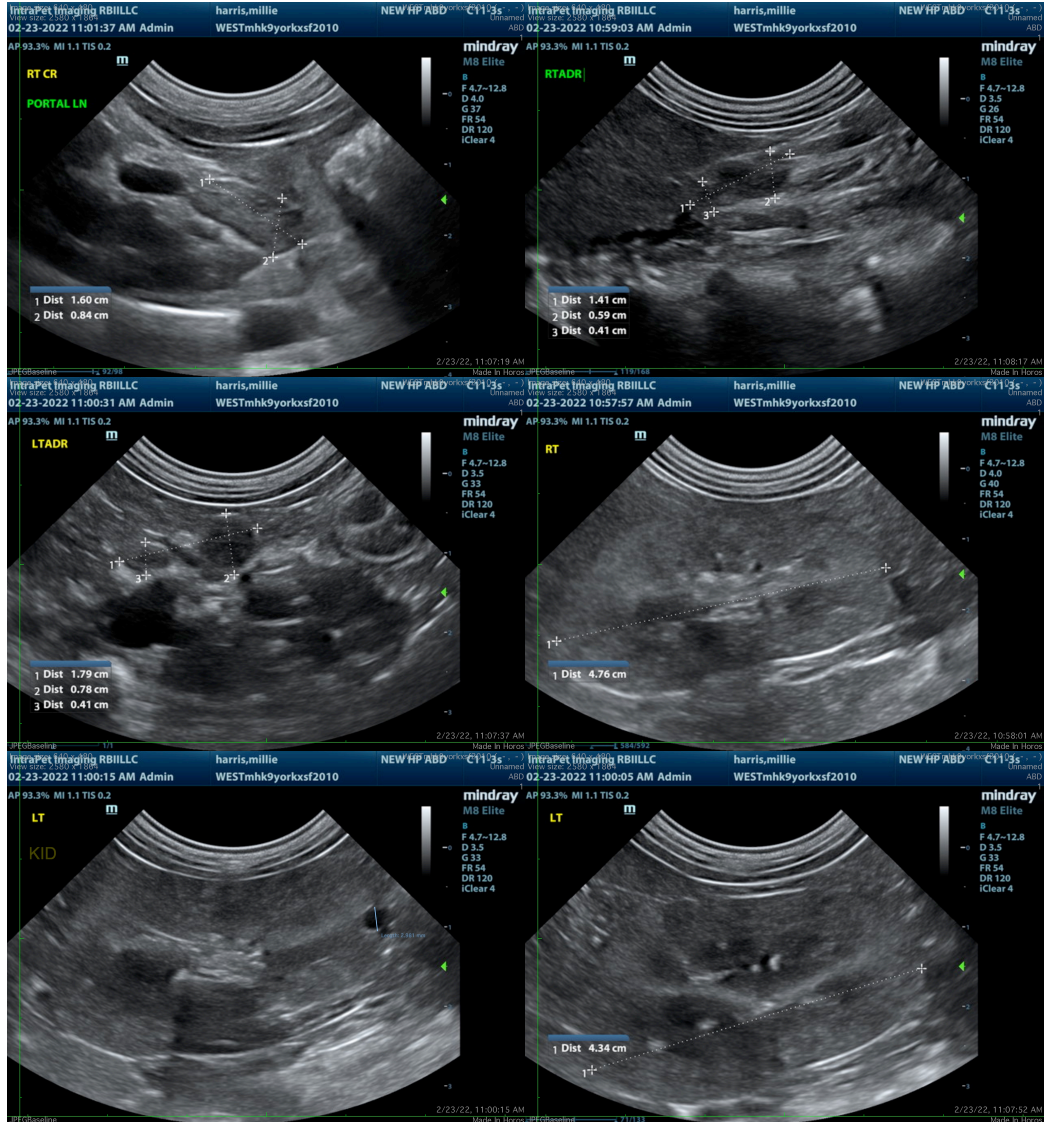
INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Given the recent changes, recommendations include a urine protein to creatinine ratio, which is reportedly planned. Given the mild hyperkalemia and mild eosinophilia in the original lab work, baseline cortisol is recommended to rule out unlikely but possible hypoadrenocorticism. If the baseline cortisol is <2.0 , a follow up full ACTH stimulation test would be recommended.

A fine needle aspirate of the spleen and liver are recommended if patient's coagulation status is appropriate. If treatment of the newly diagnosed lyme disease and management of any concurrent proteinuria does not result in an improvement in the albumin level, and the progressive hypoalbuminemia is deemed consistent with the historically diagnosed protein losing enteropathy, therapeutic recommendations include transition to a low-fat diet if the patient is not already eating a low-fat diet, as well as potentially the addition of steroids to the Atopica that is already in place.

If not recently evaluated, a gastrointestinal malabsorption panel including TLI, PLI, folate and cobalamin to Texas A&M GI laboratory is also recommended to help determine if additional medical management in the form of cobalamin supplementation, etc. may be helpful.





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.

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