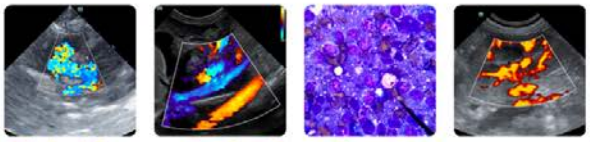


PATIENT

Wembley Barnes

SPECIES

Canine

BREED

Terrier x

SEX

Neutered Male

AGE

9 Years

WEIGHT

14.8 lbs

INTERPRETED BY

Beth Johnson, DVM DACVIM

IMAGING PERFORMED BY

Renee Trionfetti, VMD

HOSPITAL NAME

Blue Pearl Wyomissing

REFERRING VET

Heatherlynn McFarlane, DVM

INVOICE

71948

DATE

11/19/25

PRESENTING CLINICAL SIGNS

AUS to further evaluate regurgitation, diarrhea, and weight loss (3.4 kgs) that started approximately 1 month ago. Currently being managed with IM service. 10/14/25 pDVM BW showed marked panhypoproteinemia (Alb 1.2, Glob 2.4), hypocholesterolemia (64), and total hypocalcemia (Ca 6.4). Tx w/cerenia and 7days of amoxicillin which did not resolve the clinical signs. Recheck BW 10/21/25 showed persistent panhypoproteinemia (Alb 1.3, Glob 2.2), persistent hypocholesterolemia (71), and total hypocalcemia (Ca 6.6). Transitioned to Hills GI Biome. Fecal NPS but treated with 1 dose of Drontal (10/21/25) and 5days of metronidazole. 11/3/ ACTH no supportive of Addisons. Signs have been slowly improving and he is now longer having any episodes of diarrhea and appetite has normalized but recheck BW 11/12/25 neutrophilic leukocytosis (WB 21K, Neut 17K), persistent hypoproteinemia (Alb 1.5, Glob 1.2) but normal chol 108. CXR shows presence of mild amount of fluid within the caudal esophagus

Abnormal PE/Chem/CBC/UA Results: Oct 2025 CBC: WBC 19.2 H, Neut 14.62K H, Lymph 2.04, Eos 1.18, HCT 55.4%, PLT 463 Chem: TP 3.6 -> 3.6 L, Alb 1.2 ->1.3 L, Glob 2.4->2.2 L, Cr 1.1->1.0, BUN 21->17, SDMA 16->14 H, ALT 83->41, ALP 20->24 L, GGT 0->5, TBil <0.1->0.1, Chol 64->71 L, Na 154->151, K 3.8->4.3, Cl 119->120, Ca 6.4->6.6 L, Phos 3.8->3.3 Nov 2025 ACTH Stim: Pre: 2.2, Post: 11.4 CBC: WBC 20.9 H, Neut 17.1H, Lymph 1.8, Eos 1.04, HCT 44%, PLT 525 H Vetscreen: TP 2.7 L, Alb 1.5 L, Glob 1.2 L, Norm LES and renal values, TBil 0.1, Chol 108, Ca 7.1 (9.1 corrected), Glu 84 Texas A&M GI Panel: TLI 20.2-n, PLI 619 H, Cobalamin 292-low norm, Folate 10.2-n CXR: Presence of cd eso fluid, particularly on the RLat, suggests GER +/- esophagitis. Bronchial pattern consistent w/ incidental age-related fibrosis, vs mild bronchitis.

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.

Prostate is normal in size, echotexture and echogenicity for a neutered male.

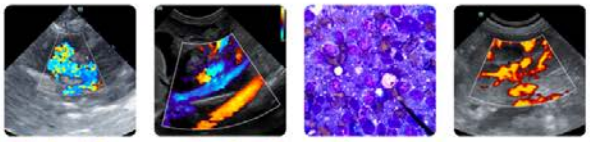
The right kidney is normal is size (5.51 cm), shape and echogenicity. It has smooth peripheral margination. There is a normal 1:3 cortex to medulla ratio with appropriate corticomedullary distinction. There is no evidence of pyelectasia, mineral or infarcts observed.

The left kidney is normal is size (4.88 cm), shape and echogenicity. It has smooth peripheral margination. There is a normal 1:3 cortex to medulla ratio with appropriate corticomedullary distinction. There is no evidence of pyelectasia, mineral or infarcts observed.

Adrenal Glands

The right adrenal gland is normal in size (0.54 cm at cranial pole and 0.39 cm at caudal pole), shape and overall architecture, echogenicity and echotexture. Visible surrounding vasculature appears normal.

The left adrenal gland is normal in size (0.37 cm at cranial pole and 0.29 cm at caudal pole), shape and overall architecture, echogenicity and echotexture. Visible surrounding vasculature appears normal.



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Spleen

The spleen is subjectively normal in size (1.2 cm thick at the hilus) with a normal smooth capsular contour. Parenchyma is appropriately finely textured and homogenous with normal echogenicity relative to surrounding tissue (hyperechoic to liver). No focal nodules or masses are observed. Splenic vasculature appears normal.

Liver

The liver is subjectively normal in size with normal smooth curvilinear peripheral contour. Parenchyma is appropriately hypoechoic to the spleen in echogenicity and appropriately mildly coarse and homogenous in echotexture. 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 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.

Small intestine is diffusely mildly thick 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 or foreign material.

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

Pancreas

The pancreas that is observed appears appropriately isoechoic to surrounding omental fat. Visible capsule is smooth and normal in contour. Visible pancreatic parenchyma is homogenous and unremarkable. There is no visible pancreatic duct dilation. There is no evidence of active peripancreatic inflammation.

Free Abdomen

There are several scant/trace pockets of free fluid and diffusely enhanced hyperechoic mesenteric fat.

There is no apparent pathologic lymphadenopathy noted in these images.

ULTRASONOGRAPHIC FINDINGS

- 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.

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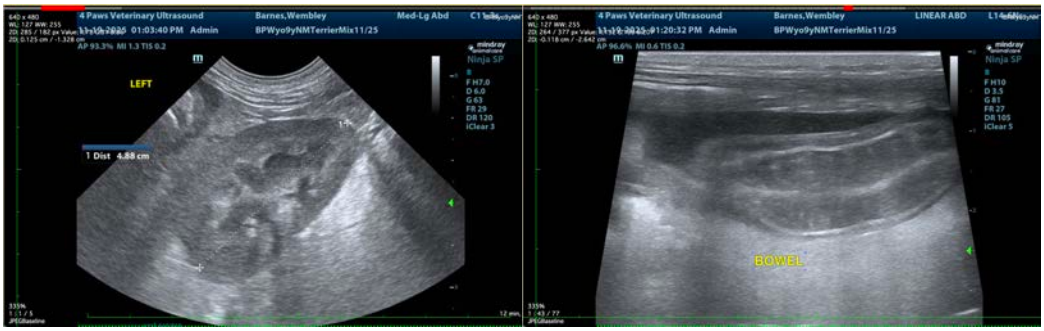
- The pockets of free fluid and diffusely enhanced hyperechoic mesenteric fat are likely secondary to patient's reported hypoalbuminemia and concurrent diffuse gastrointestinal disease. Other pathologic fluid differentials, however, can't be ruled out.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

- 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.
- As is reportedly already assessed, baseline cortisol is recommended. If baseline cortisol is less than 2, a full ACTH stimulation test is recommended to rule out hypoadrenocorticism.
- Ideally, biopsies of the GI tract are recommended to definitively diagnose and therefore manage the infiltrative bowel process.
- If biopsies cannot be obtained safely due to low albumin or patient stability, etc., empirical therapies could include diet change to an ultra-low-fat diet, empirical deworming with a 5-day course of Panacur, cobalamin supplementation (unless cobalamin level is evaluated and supplementation is not warranted) a probiotic and prednisolone (if not contraindicated based on patient contraindications, co-morbidities, etc.).
- Calcium monitoring, and supplementation, if necessary, is also recommended.
- Additionally, if patient's coagulation status is otherwise appropriate, anti-thrombotics such as clopidogrel or low dose aspirin may also be warranted.
- Additionally, 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.
- If not recently evaluated, ruling out proteinuria is also advised via a urinalysis and, if indicated based on urinalysis results, urine culture is recommended. If protein is present in an otherwise quiet sediment, protein quantification with a urine protein to creatinine ratio is recommended.



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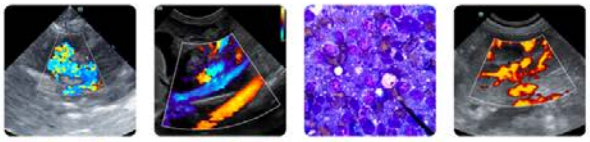

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Educational Teleconsultation Services™

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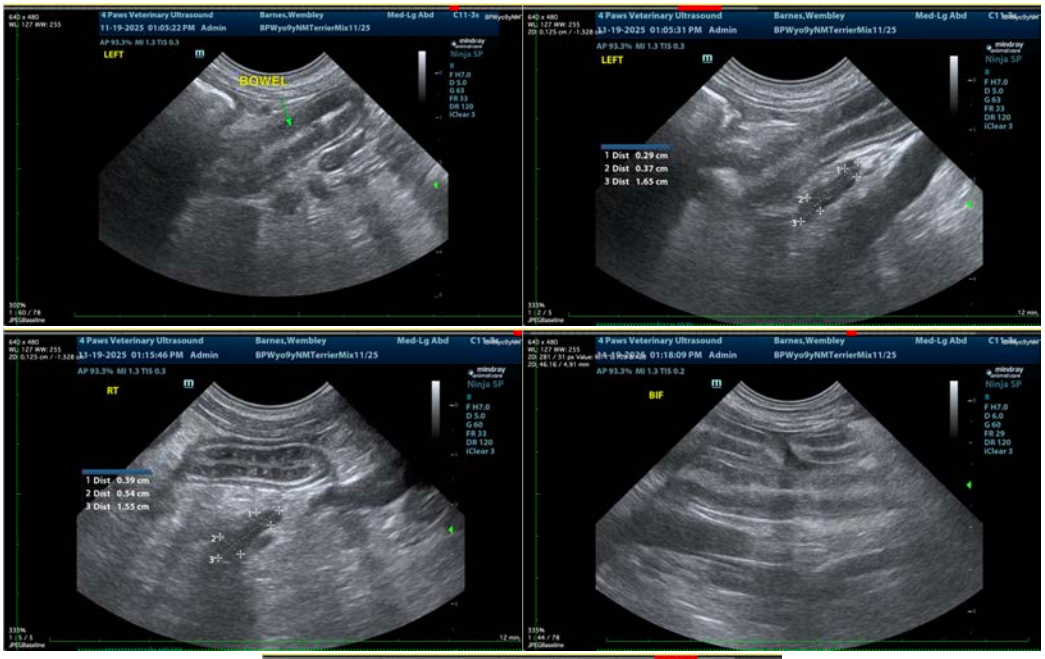

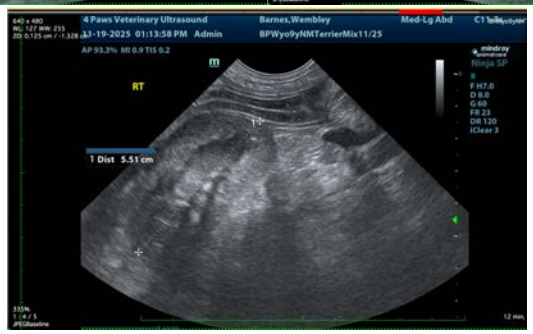

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Beth Johnson, DVM, DACVIM
info@sonopath.com