



**PATIENT**

Morrolan Sloan

**SPECIES**

Feline

**BREED**

DSH

**SEX**

Neutered Male

**AGE**

14 Years

**WEIGHT**

13 Pounds

**INTERPRETED BY**

R. McKenzie Daniel,  
DVM, DABVP  
(Canine and Feline)

**IMAGING PERFORMED BY**

Amy Mayhew, LVT

**HOSPITAL NAME**

SVS Imaging Michigan

**REFERRING VET**

Cat Care of Rochester

**INVOICE**

33188

**DATE**

12/2/21

**PRESENTING CLINICAL SIGNS**

History: 1. PU/PD, Mild CRI (USG 1.017, kidney values normal) for ~ 12 to 18 months 2. Chronic, mild pancreatitis for several years 3. Weight stable until last month (mild loss, although still on OM dry and canned); mild tartar and gingivitis; MCS normal, BCS 3.5/5 4. Occasional vomiting (not always sure who the culprit is, as there are 3 cats in the home) 5. Bout of inappetance and lethargy the week of Thanksgiving; responded to B12, Cerenia, Famotidine 6. Elevated pLi and liver values on most recent BW (11-11-21) BW notes: 1. MCH 17.6 (H)- Not clinically significant 2. Renal values are normal and stable 3. ALB 4.0 (H)- Rule-out mild hemoconcentration 4. ALT 312 (H)- was 65, 38 AST 104 (H)- was 40 ALP 123 (H)- was 30, 29 Rule-out primary liver disease (infection, inflammation, cancer) v. secondary (thyroid, pancreatitis, etc) 5. CK 538 (H)- Not clinically significant 6. PLI 12.7 (H)- was 10.8, 7.4 Rule-out worsening pancreatitis 7. BNP 41 (N) 8. T4 2.9 (H)- was 2.3, 2.2 Requested free T4 by ED 9. T4 by ED WNL Treatments: 1. Long term meds: a. Daily: 5 mg Famotidine q 24h, 1/8 tsp Miralax, ¼ tsp Benefiber b. Monthly: Revolution Plus 2. Currently on: a. Cerenia 12 mg PO q 24 hrs (replacing famotidine) b. Orbax 39 mg PO q 24 hrs c. Miralax, Benefiber, Revolution Plus

**ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN**

**Urinary System**

The urinary bladder, trigone, cystourethral junction, and visible pelvic urethra to a depth of 2.0 cm exhibited normal thickness and tone. Primarily anechoic urine was present in the lumen. Mild non-dependent particulate sediment was present without evidence of calculus formation, suggestive of minor cellular or crystalline debris. The ureteral papillae were normal. The ureters were not visible which is normal. No evidence of inflammatory or neoplastic mural changes were noted.

The area of the aortic trifurcation was free of pathology.

Normal renal size with asymmetrical margination was present in both kidneys. The renal cortex presented uniformly increased in echogenicity with uniform echotexture. The renal cortex appeared to be hypertrophied resulting in an altered cortex: medulla ratio. Mild loss of corticomedullary distinction was also present. The renal medullary volume was subjectively reduced. The left kidney measured 4.2 cm. The right kidney measured 4.3 cm.

**Adrenal Glands**

The adrenal glands were uniform in size and contour with a uniformly hypoechoic parenchyma. The left adrenal gland measured 0.5 cm in width. The right adrenal gland measured 0.4 cm in width.

**Spleen**

The spleen was normal in size and overall contour with generalized splenic parenchyma heterogeneity with subtle micronodular parenchymal changes. Intermittent, non-expansive discreet echogenic parenchymal nodules noted. The spleen measured 1.0 cm in width.

**Liver**

The liver was mildly enlarged. The parenchyma of the liver was subjectively normal in echogenicity compared to the spleen and renal cortices. The liver parenchyma was uniform with a mildly coarse echotexture. The capsule of the liver was symmetrically rounded to mildly swollen in margination. The hepatic and portal vasculature were normal in appearance without signs of congestion. The gallbladder was non distended in size with mild, echogenic, nonmineralized biliary sludge. The cystic duct and common bile ducts were normal without evidence of dilation.



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**Gastrointestinal**

The stomach presented intact wall layering with a normal wall layer ratio. The lumen of the stomach was empty with no signs of ileus, obstruction or foreign material. Gastric body wall measured 0.25 cm.

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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. Duodenum wall measured 0.32 cm. Jejunum wall measured 0.25 cm. Ileocolic wall measured 0.33 cm.

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

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**Pancreas**

The pancreas was normal in size with isoechoic to heterogeneous parenchyma compared to adjacent omentum and evidence of parenchymal remodeling. Asymmetrical contour noted. No signs of active inflammation or neoplasia.

**Free Abdomen**

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A mildly prominent colic lymph nodes was present, measuring 0.9 cm x 0.3 cm. These lymph nodes were homogenous, mildly hypoechoic and smoothly marginated. A normal width: length ratio was maintained (<0.5). Evidence of perilymphatic inflammation was evident.

**WEIGHT**

13 Pounds

No effusion. No other evidence of intraabdominal lymphadenopathy.

**ULTRASONOGRAPHIC FINDINGS**

- Bilateral chronic renal changes exhibiting mild uniformly hyperechoic cortical hypertrophy – Chronic renal disease with potential for interstitial nephritis.
- Heterogeneous to subtly micronodular spleen
- Hepatopathy with mild gallbladder debris – suspect primary versus secondary hepatic parenchymal inflammation or inflammatory hepatobiliary process given the primarily elevated ALT with potential for vacuolar hepatic changes and non-clinical cholestasis. No overt evidence of neoplastic criteria.
- Chronic pancreatitis exhibiting pancreatic parenchymal remodeling
- Possible inflammatory enteropathy

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**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**

The splenic changes are most likely consistent with age related benign nodular hyperplasia. However, potential for early neoplasia (i.e., lymphoma) cannot be entirely ruled out. Assuming normal clotting status, ultrasound guided FNA of the spleen using 25-gauge needle could be considered for screening cytology given the patient's mild weight loss. Otherwise, sonographic monitoring of the spleen with initial recheck in 3-4 weeks would be a more conservative approach.

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No overt evidence of structural gastrointestinal pathology, yet potential triad disease may be a consideration in this patient given the elevated liver enzymes, chronic pancreatitis, and weight loss. Although histology is required for definitive diagnosis, empirical triad disease protocol (which may include continued as needed gastrointestinal support and cobalamin supplementation, hydrolyzed diet trial +/- Prednisolone at lowest effective dose to control clinical signs) could be considered with assessment of clinical response, especially if persistent weight loss is noted in this patient.

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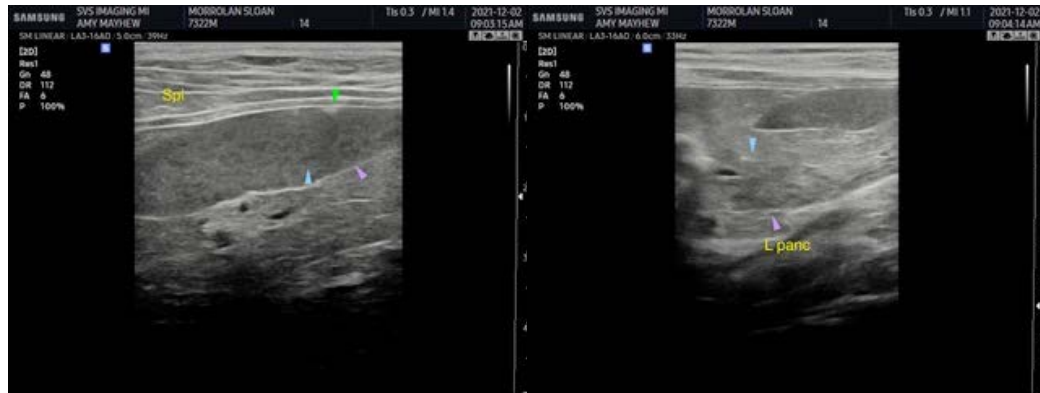
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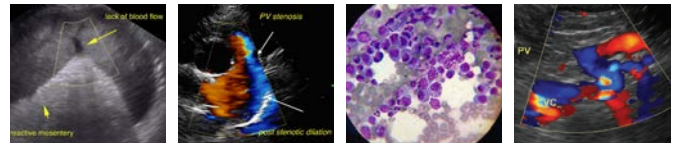
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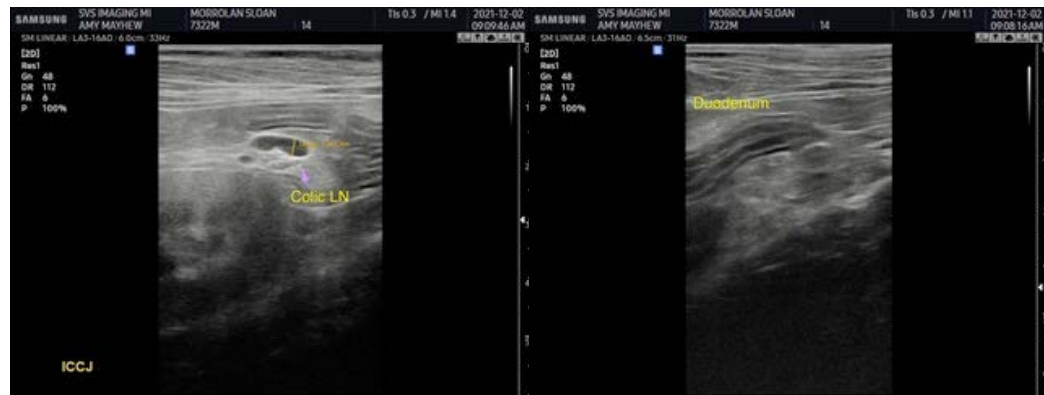
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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)**  
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