

**PATIENT**

Sissy Schultz

**SPECIES**

Feline

**BREED**

DSH

**SEX**

Spayed Female

**AGE**

15 Years 8 Months

**WEIGHT**

6.8 lbs

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

Brandi Kurzowski

**HOSPITAL NAME**

Corfu Veterinary Clinic

**REFERRING VET**

Dr. Kayla Greil

**INVOICE**

72266

**DATE**

12/3/25

**PRESENTING CLINICAL SIGNS**

Weight loss, not eating as much over the past 6 months- p has lost 50% body weight since July 2024. Grade 2/4 PDD. Generalized muscle atrophy.

Abnormal PE/Chem/CBC/UA Results: CBC: Retic 50.5 K/uL (3-50) H ; Eos 0.10 K/uL (0.17-1.57) L Chem 17/lytes: BUN 15mg/dL, Glob 5.3 g/dL, ALP 1315 U/L, TBIL 3.9 mg/dL, T4- 2.3 ug/dL

**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.

Kidneys are bilaterally irregular and diffusely echogenic with decreased corticomedullary distinction and poor visualization of internal architecture. There is no pyelectasia noted and no mineral is observed. The left kidney is small-normal measuring 3.2 cm. A chronic infarct is present in the left kidney. The right kidney is small at 2.8 cm.

**Adrenal Glands**

The areas of the adrenal glands are examined without evident adrenal gland pathology.

**Spleen**

The 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). No focal nodules or masses are observed. Splenic vasculature appears normal.

**Liver**

Liver is subjectively enlarged (swollen contour) without disruption of architecture. 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 and falciform fat. No focal lesions are observed. 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.

The visible small intestine demonstrates areas of moderately 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 empty with no evidence of obstruction or foreign material.



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

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### **Pancreas**

Pancreas is prominent (enlarged) in size, hypoechoic to surrounding tissue and has a mildly irregular undulating contour. Parenchyma is coarse with mixed echogenic remodeling noted. No pancreatic duct dilation is noted.

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### **Free Abdomen**

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

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There is no apparent pathologic lymphadenopathy noted in these images.

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## ULTRASONOGRAPHIC FINDINGS

- Moderate 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, etc. is noted to make lymphoma more probable, but lymphoma cannot be definitively ruled out without tissue sampling.
- Suspect chronic low-grade smoldering pancreatitis.
- Hyperechoic hepatomegaly – This appearance is most consistent with benign hepatic lipidosis or endocrine/DM hepatopathy. Infiltrative disease such as amyloidosis or round cell neoplasia, such as mast cell tumor or less likely, lymphoma, is also possible.
- Moderate gallbladder debris – Cholecystic debris is of unknown clinical significance. It can be seen with biliary stasis from fasting or illness, however, it can also be associated with hepatobiliary disease in cats 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 chronic kidney disease changes including a chronic infarct in the left kidney.

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## INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The appearance of patient's bowel, hepatobiliary system, pancreas, etc. could indicate "Triaditis". Further diagnostic recommendations include:

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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.

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Fine needle aspirates of the liver could be considered if patient's coagulation status is appropriate.

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Ultimately, biopsies of the GI tract, being sure to include ileum, if possible, +/- liver may be necessary for definitive diagnosis and therefore to further guide medical management.

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In the meantime, treatment recommendations include fluid therapy, anti-emetics, gastroprotectants, hepatic nutraceuticals such as ursodiol and/or Denamarin, and broad-spectrum antibiotics. Nutritional support is critical to prevent/manage concurrent hepatic lipidosis, so appetite stimulants and/or, if indicated, feeding tube placement is also recommended.



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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