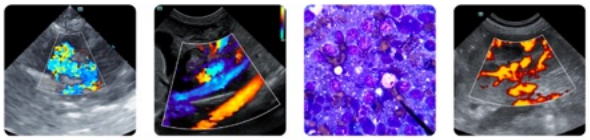


PATIENT

Seamus Martin

SPECIES

Feline

BREED

Domestic Longhair

SEX

Neutered male

AGE

15 years

WEIGHT

4.25 lbs

INTERPRETED BY

Alicia Angosto Guerrero, DMV, PgDip, MSc.

IMAGING PERFORMED BY

Brooke Bennett

HOSPITAL NAME

Viking VH Moscow

REFERRING VET

Dr. Bennett

INVOICE

73749

DATE

3/24/26

PRESENTING CLINICAL SIGNS

Patient presented for weight loss that was noticed about 1 year ago after passing of housemate but recently seemed to loss a lot more rapidly

- 2013 diagnosed with urinary crystals, does not appear that patient was transitioned to urinary diet
- no current medications on presentation, UTD on all vaccines, no known allergies, indoor only
- chronic sneezing has been seen with patient but fluctuates in severity and does not appear to coincide with current symptoms seen
- Patient is still eating very well and no vomiting or diarrhea seen
- PE on 3/22: poor BCS and muscling diffusely, ~7% dehydration, icteric. Chem on 3/22: ALT 448 U/L (12 to 130), ALP 152 U/L (14-111), GGT 6 U/L (0-4), Total Bilirubin 7.3 mg/dL (0-0.9), Cholesterol 226 mg/dL (65-225), Globulin 5.7 g/dL (2.8-5.1) Chem on 3/23: ALT decreased by 2, ALP increased by 19, GGT returned to normal, Total Bilirubin increased by 0.6, Cholesterol unchanged, Globulins returned to normal CBC on 3/22- 22,870 total WBC characterized by mature neutrophilia and monocytosis UA - evidence of bilirubin crystals, USG of 1.022 despite clinical evidence of dehydration T4 - WNL for older cat, below "grey zone" Pancreatic Lipase - WNL

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The bladder lumen is normally distended, and the wall of the urinary bladder appears thin and smooth. The urine is markedly turbid, with abundant suspended echogenic material consistent with sediment. Normal appearance of the bladder neck and proximal urethra. There are no calculi and no evidence of inflammatory or neoplastic changes.

The left kidney is normal in shape and size: 4.31×2.94 cm, and the thickness of the cortex is 0.59 cm in the sagittal plane. The cortex is hyperechoic compared to the liver parenchyma. The corticomedullary ratio is normal and corticomedullary definition is preserved. Mild pyelectasia is present (1.92 mm). No nephroliths or hydronephrosis are identified.

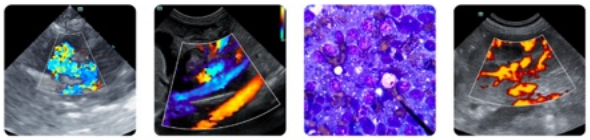
The right kidney is enlarged (5.40×3.37 cm); cortical thickness is not provided. The cortex is markedly hyperechoic. The corticomedullary ratio is normal, but corticomedullary definition is decreased. The renal pelvis is dilated up to 7.57 mm. No nephroliths or hydronephrosis are identified.

Adrenal Glands

Both adrenal glands are not visualized.

Spleen

Splenic thickness is 0.80 cm. The parenchyma demonstrates normal echogenicity and fine homogeneous echotexture without focal parenchymal abnormalities. The splenic capsule is smooth and regular.



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Liver

The liver is subjectively enlarged, with sharp edges and a regular contour. The hepatic parenchyma is diffusely hyperechoic with a fine echotexture. These findings are highly suggestive of hepatic lipidosis. No hepatic lymphadenopathy is observed.

The gallbladder is normally distended (3.39×1.47×0.82 cm: 2.13 mL). Multiple wall-associated structures are identified, including cystic and polypoid-appearing changes, along with a moderate amount of organized biliary sludge. The common bile duct measures 4.90–4.11–2.41–2.08–1.60 mm from proximal to distal (near the duodenal papilla). No clear dilation of intrahepatic bile ducts is identified.

Gastrointestinal

The stomach contains a small amount of ingesta, with mural thickness (2.20 mm) and preserved wall layering.

Duodenum: 1.95 mm. Jejunum: 1.64 mm. Ileum: 1.23 mm. The ileocecal junction is not visualized. All intestinal segments contain ingesta and show increased peristalsis, consistent with a non-fasted state, which limits accurate wall thickness assessment. However, there is no subjective evidence of mural thickening or loss of wall layering.

Colon: 0.92 cm, with abundant formed feces in the lumen.

Pancreas

Pancreatic visualization is suboptimal due to the presence of ingesta within the gastrointestinal tract.

Free Abdomen

A mild amount of anechoic abdominal effusion is present. No sonographic evidence of generalized peritonitis or lymphadenomegaly is identified.

PRIMARY FINDINGS

- Diffuse hepatic hyperechogenicity.
- Gallbladder wall cystic/polypoid changes with biliary sludge. Common bile duct dilation proximally (up to ~4.9 mm)
- Right renal pelvic dilation (7.57 mm).
- Bilateral renal cortical hyperechogenicity.

SECONDARY FINDINGS

- Markedly turbid urinary sediment.
- Mild abdominal effusion.

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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Findings are most consistent with diffuse hepatocellular disease, with hepatic lipidosis strongly suspected, likely secondary to chronic disease or recent metabolic stress associated with weight loss. However, hepatic lipidosis may mask underlying primary hepatobiliary pathology, and concurrent cholangitis/cholangiohepatitis remains a key differential, particularly given the marked hyperbilirubinemia.

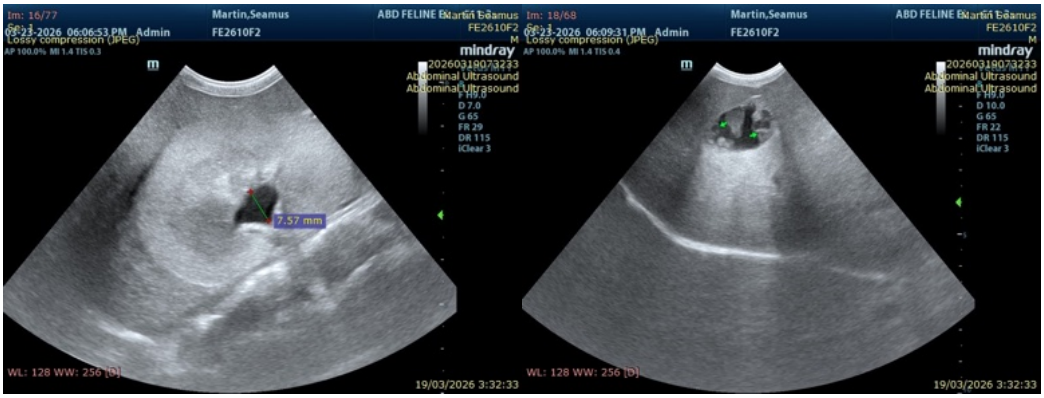
The gallbladder wall changes (multiple cystic lesions and a few polypoid structures) with associated sludge are most consistent with chronic inflammatory disease (chronic cholecystitis and mucosal hyperplasia). However, both inflammatory and neoplastic gallbladder conditions are considered relatively uncommon in cats, and distinction between these entities may be challenging based on ultrasonographic findings alone. The common bile duct is mildly to moderately enlarged proximally (up to ~4.9 mm). In cats, this is above typical reference values (~≤3–4 mm), raising concern for partial biliary obstruction or impaired bile flow, even in the absence of intrahepatic duct dilation. This is clinically significant given the degree of hyperbilirubinemia.

Renal findings are nonspecific but may reflect chronic renal changes or secondary effects of systemic illness.

Mild abdominal effusion is present and likely reactive, potentially secondary to hepatobiliary disease.

Recommendations
Findings support a primary hepatobiliary disease process, most consistent with chronic cholangitis/cholangiohepatitis, with secondary hepatic lipidosis, concurrent chronic gallbladder disease, and suspected partial biliary outflow impairment. Initial management should prioritize aggressive supportive care, including nutritional support and fluid therapy. Medical management targeting hepatobiliary disease is recommended, with specific therapeutic decisions to be made by the attending veterinarian, who can best integrate these findings with the patient's clinical status and ongoing response to treatment.

More invasive procedures, such as gallbladder sampling or liver biopsy, may be considered if there is poor clinical response or once hepatic lipidosis has improved, as current changes may limit diagnostic yield.



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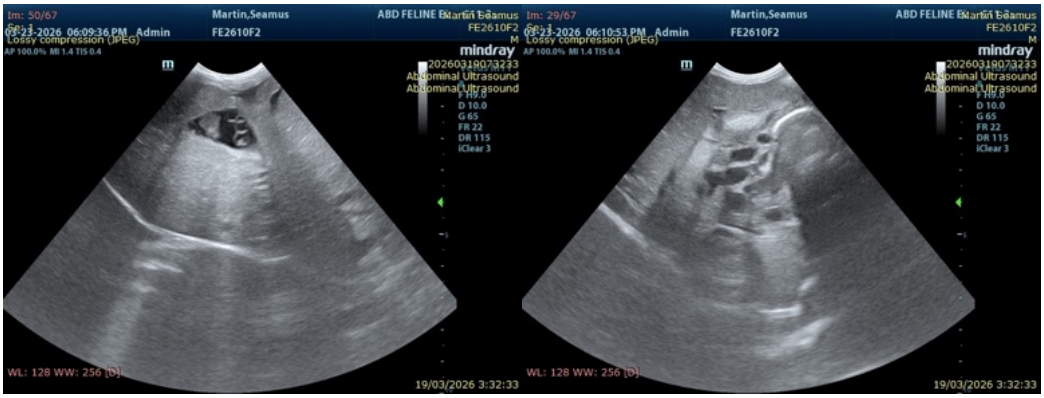

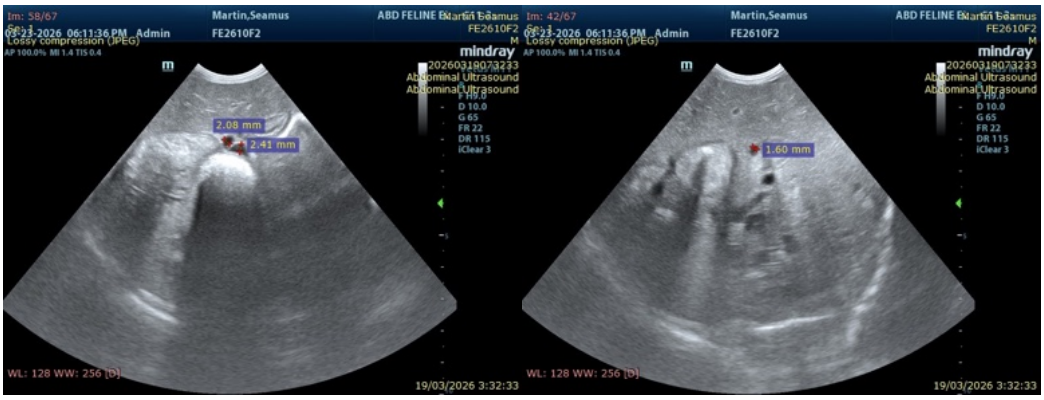

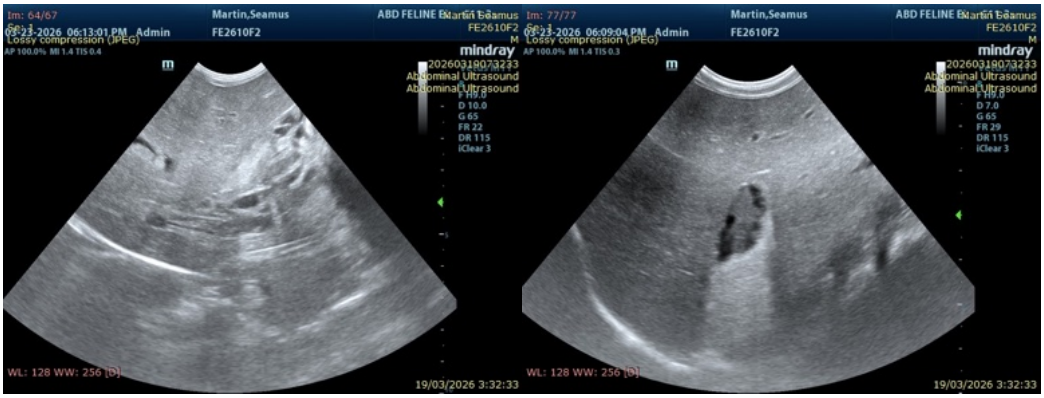

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Alicia Angosto Guerrero, DMV, PgDip, MSc.

info@SonoPath.com