

PATIENT

Mingus Donahue
Nunley

SPECIES

Feline

BREED

Domestic Shorthair

SEX

Neutered male

AGE

15 years

WEIGHT

7 lbs

INTERPRETED BY

Alicia Angosto
Guerrero, DMV,
PgDip, MSc.

IMAGING PERFORMED BY

John Sampson

HOSPITAL NAME

Richboro VH

REFERRING VET

Dr. Sampson

INVOICE

74016

DATE

4/1/26

PRESENTING CLINICAL SIGNS

- Hyperthyroid patient (well controlled) Presented 3/30/26 for significant PUPD, Polyphagia, and yellow color of teeth. Change in behavior went for introvert to extrovert even with strangers, being vocal to get their attention. Very vocal at night crying for food and attention
- Significant icterus throughout body - Hepatomegaly - Attached is bloodwork from 3/30

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder is normally distended. The bladder wall is thin, smooth, and regular. The luminal contents are anechoic. Normal appearance of the bladder neck and proximal urethra. No evidence of urolithiasis or inflammatory or proliferative changes is identified.

The left kidney is normal in shape and size, measuring 3.69×1.97 cm in the sagittal plane. Cortical thickness is 0.43 cm. The right kidney is normal in shape and size; however, measurements were not obtained.

Both kidneys show increased cortical echogenicity compared to the hepatic parenchyma. The corticomedullary ratio is within normal limits, and corticomedullary definition is preserved. There is no evidence of pyelectasia, nephrolithiasis, or hydronephrosis.

Adrenal Glands

Not visualized.

Spleen

Splenic thickness is 0.96 cm. The parenchyma demonstrates normal echogenicity and fine homogeneous echotexture without focal parenchymal abnormalities. The splenic capsule is smooth and regular.

Liver

The liver is subjectively increased in size, with rounded margins and a regular contour. The hepatic parenchyma is homogeneous and isoechoic relative to the falciform fat, with a normal echotexture. No focal lesions or hepatic lymphadenopathy are identified.

The gallbladder is moderately to markedly distended. (4.45×1.52×1.25 cm): 4.42 ml. (normal range 1-4 ml). The wall is thin and regular. The luminal contents are predominantly anechoic, with a very small amount of sludge in the fundus. The common bile duct was difficult to follow and measure due to the absence of Doppler evaluation; however, a segment was identified measuring 2.55 mm in luminal diameter, with a wall thickness of 1.35 mm (subjectively thickened). No clear ultrasonographic evidence of extrahepatic biliary obstruction is identified. No intrahepatic bile duct dilation is observed.



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Gastrointestinal

The stomach is empty and folded, with a wall thickness of 1.99 mm and preserved layering. Duodenum: 2.01 mm. The duodenal papilla measures 5.56×7.23 mm.

Jejunum: 2.04–2.28 mm; mucosa 1.23 mm, submucosa 0.44 mm, muscularis propria 0.51 mm. Ileum: 2.19 mm; mucosa 0.80 mm, submucosa 0.65 mm, muscularis propria 0.74 mm. Wall layering is preserved. The ileocecal junction measures 3.97 mm, with muscularis thickness of 1.50 mm.

Colon: ascending 0.99 mm, transverse 0.73 mm, descending 0.70 mm, containing formed feces.

Pancreas

The evaluated pancreatic areas do not show evidence of overt inflammation or neoplastic disease.

Free Abdomen

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

FINDINGS and INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The liver is enlarged but maintains a homogeneous echotexture without focal lesions, which is most consistent with a diffuse reactive process. In this context, hepatomegaly is most plausibly explained by a combination of hepatocellular swelling secondary to cholestasis, inflammatory infiltration, and parenchymal edema, all of which can increase hepatic volume without significantly altering echogenicity.

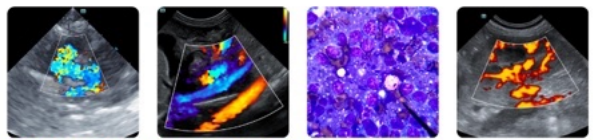
The gallbladder is markedly distended, and although the common bile duct is not clearly dilated throughout and no intrahepatic biliary duct dilation is identified, the presence of a subjectively thickened bile duct wall supports inflammatory involvement of the biliary tree. Importantly, the absence of ductal dilation does not definitively exclude partial extrahepatic biliary obstruction, particularly in feline patients.

The presence of moderate abdominal effusion is most consistent with a modified exudate secondary to hepatobiliary inflammation, likely reflecting a combination of capsular irritation and increased vascular permeability associated with active cholangitis/cholangiohepatitis. Hypoalbuminemia is not sufficient to account for the effusion in this case.

Although less likely, an element of early or developing portal hypertension cannot be completely excluded, particularly given the subjective prominence of splenic and splenorenal vasculature; however, this remains unconfirmed.

Pancreatic evaluation does not reveal overt abnormalities, although subclinical pancreatic involvement cannot be excluded and a triaditis-type process remains a reasonable unifying consideration.

Mild intestinal muscularis thickening at the ileocecal junction is noted and may be compatible with



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chronic enteropathy or low-grade lymphoma, although this finding is subtle and likely incidental relative to the primary hepatobiliary disease.

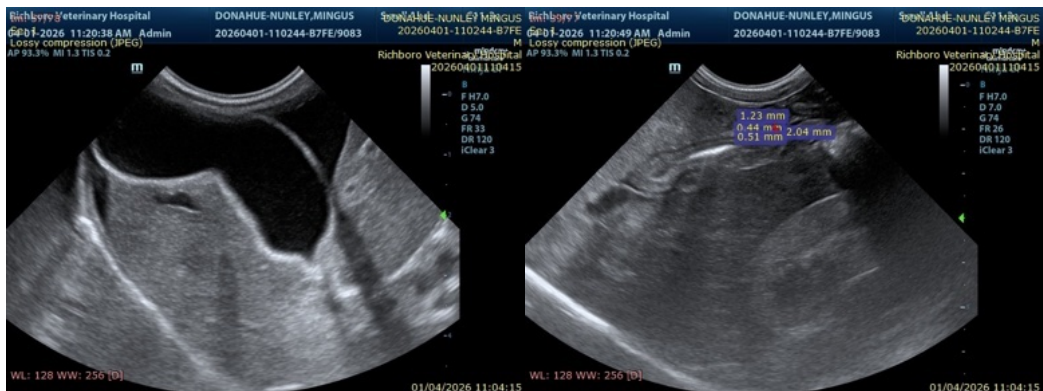
Overall, ultrasonographic findings are most compatible with:

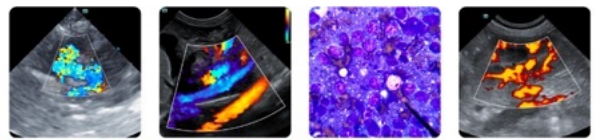
- Severe cholangitis / cholangiohepatitis (highly compatible).
- Possible early or partial biliary obstruction (cannot be excluded).
- Concurrent triaditis (possible, despite inconspicuous pancreas).

Recommendations

- Abdominocentesis and fluid analysis (total protein, cytology) to confirm whether the effusion is consistent with a modified exudate secondary to hepatobiliary inflammation.
- Empirical treatment for hepatobiliary disease.
- Further characterization (as clinically indicated)
 - fPLI (optional, if it will influence management).
 - Sampling may be considered if poor response or clinical deterioration.
- Monitoring
 - Recheck bilirubin and liver enzymes.
 - Adjust therapy based on clinical and biochemical response.

Final diagnostic and therapeutic decisions should be made by the attending veterinarian, who can best integrate these findings with the patient's clinical status.





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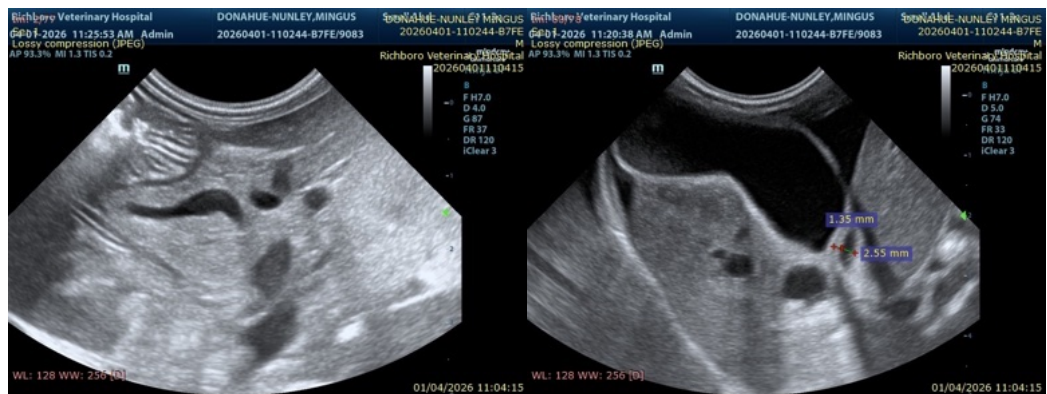
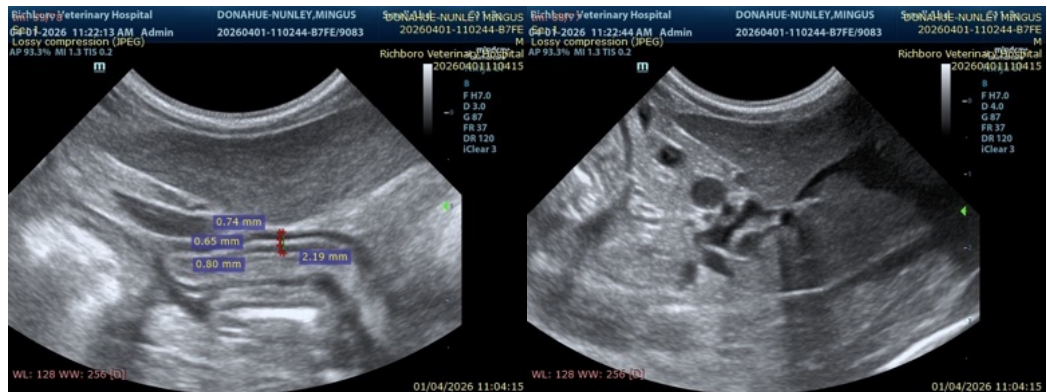
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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