

**PATIENT**

Piper Normand

**SPECIES**

Canine

**BREED**

Maltese Mix

**SEX**

Spayed Female

**AGE**

10 years

**WEIGHT**

9.3 lbs

**INTERPRETED BY**

Beth Johnson, DVM  
DACVIM

**IMAGING PERFORMED BY**

Pamela Harrigan,  
RDCS, Certified  
Veterinary  
Sonographer

**HOSPITAL NAME**

VCA Palmer Animal  
House

**REFERRING VET**

Dr. Michelle Haroules

**INVOICE**

11114

**DATE**

1/14/2026

**PRESENTING CLINICAL SIGNS**

Routine bloodwork: glob 4.1, AST 87, ALT 399, ALP 233, GGTP 18, BUN 46. No clinical signs.

**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 overall normal in size and shape with smooth peripheral margination. A normal 1:3 cortex to medulla ratio is maintained. The medulla and cortices are uniform in texture with some mild increased cortical echogenicity and mild loss of corticomedullary distinction, expected in this age patient. There is no evidence of pyelectasia, mineral or infarcts observed. Left kidney measures 3.5 cm. The right kidney measures 3.8 cm, and contains a small cortical cyst.

**Adrenal Glands**

The right adrenal gland is normal in size (0.42 cm at cranial pole and 0.42 cm at caudal pole), shape and overall architecture, echogenicity and echotexture. Visible surrounding vasculature appears normal.

The left adrenal gland is normal in size (0.33 cm at cranial pole and 0.48 cm at caudal pole), shape and overall architecture, echogenicity and echotexture. Visible surrounding vasculature appears normal.

**Spleen**

Spleen is subjectively large 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. The spleen is folded upon itself, which is a positional non-pathologic variant.

**Liver**

Liver is subjectively enlarged with mildly irregular margins. Parenchyma is moderately heterogenous characterized by multiple poorly defined hypoechoic nodules within otherwise hyperechoic liver parenchyma. 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. Some of the debris has a mineral appearance with no visible evidence of obstruction noted in these images at this time. In one view, the intraluminal density measures approximately 1.0 cm x 2.0 cm in size with an irregular shape. This density presumably represents debris +/- mineral, although tissue can't be definitively ruled out. 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.



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The visible small intestines are normal in wall thickness and layering. Small intestinal motility appears adequate (1-3 contractions per min). The lumen of the small intestine is empty with no evidence of obstruction, foreign material or infiltrative disease.

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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.

**Pancreas**

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The observed pancreas appears appropriately isoechoic to surrounding omental fat. The capsule is mildly irregular in shape. Parenchyma is mildly heterogenous and coarse. There is no visible pancreatic duct dilation. There is no evidence of active peripancreatic inflammation.

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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.

**PRIMARY FINDINGS**

**WEIGHT**

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- Moderately heterogenous liver – These changes are most consistent with benign processes such as nodular hyperplasia, steroid (vacuolar) hepatopathy, extramedullary hematopoiesis or possibly chronic inflammatory disease and less commonly infiltrative round cell or metastatic neoplasia.
- Subjectively mild splenomegaly– can be associated with congestion caused by sedation (if sedated) but can also be associated with diffuse infiltrative disease. Both benign conditions such as extramedullary hematopoiesis, lymphoid hyperplasia as well as infiltrative neoplastic diseases such as round cell neoplasia should be considered.
- Moderate gallbladder debris - Cholecystic debris is of unknown clinical significance. It can be seen with biliary stasis from fasting or illness. Cholecystic debris is not necessarily related to hepatobiliary disease. Echogenic bile is most commonly an incidental finding in dogs 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. Non-visibly shadowing, non-obstructive mineral and/or even less likely a tissue nodule, as described above, can't be definitively ruled out.

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**SECONDARY FINDINGS**

- Age related kidney changes with a small cortical cyst in the right kidney.
- Pancreatic age-related remodeling/Chronic pancreatitis – Mild irregularities are consistent with benign age-related change. Low-grade smoldering chronic pancreatitis cannot be ruled out and should be suspected in the face of appropriate clinical signs.

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**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**

Given patient's reported azotemia, if not recently evaluated 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 ration is recommended.

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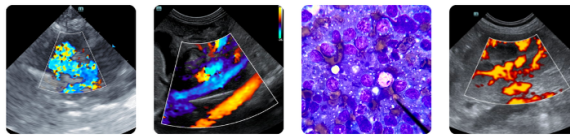
Differentials for a primary hepatocellular injury liver enzyme pattern (increased ALT) depend partially on the level of increase. Mild increases (less than 2 times normal) are often a “reactive hepatopathy” or the liver’s response to an insult elsewhere in the body including, but not limited to, pancreatitis, gastroenteritis, parasitic disease, dental disease, vacuolar or endocrine hepatopathy from diabetes mellitus or hyperadrenocorticism (steroid-induced), hypoadrenocorticism, certain drugs (e.g. phenobarbital, corticosteroids, azathioprine, etc.), and muscle ALT (more likely if AST and CK concurrently increased).

It is a good indicator of active liver damage (cell membrane disruption, cellular necrosis), however, if the value is increased by at least 3-4 times normal. Differentials include infectious disease, including Leptospirosis, inflammatory disease (ie. active hepatitis, copper, other), toxic insult as well as infiltrative neoplasia.

ALT levels vary in cases of vascular anomalies such as microvascular dysplasia and portosystemic shunts (PSS), but are often less significantly increased.

- Testing for Leptospirosis could be considered.
- Bile acids could be considered, if tbili is not increased.
- An empirical course of antibiotics and empirical hepatic nutraceuticals may be tried, with monitoring of ALT for improvement. If improvement is noted, antibiotics should be continued until liver enzymes either normalize or plateau (recheck every 2-3 weeks); however, if improvement is not noted and/or enzyme increase progresses, antibiotics should not be continued long term and liver tissue sampling is recommended.
- FNA of the liver +/- spleen can be performed to assess inflammatory cell type, rule in/out round cell neoplasia, etc. (if patient’s coagulation status is appropriate).
- If round cell neoplasia is not diagnosed, a liver biopsy (including copper level assessment) may be required to definitively diagnose the underlying hepatopathy.





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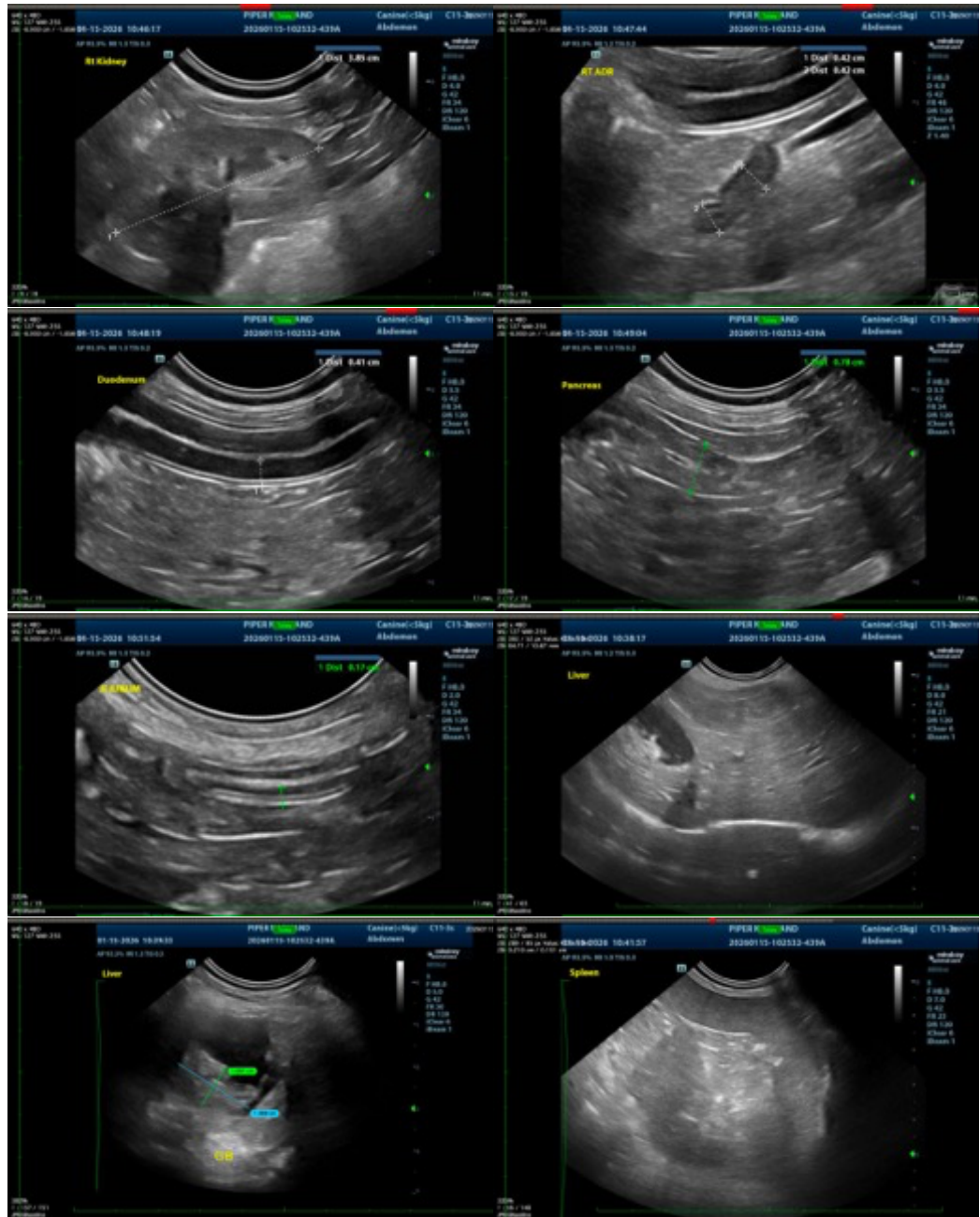
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Beth Johnson, DVM, DACVIM

info@sonopath.com