



PATIENT

Leo Padovani

SPECIES

Canine

BREED

Havanese

SEX

NM

AGE

7 years

WEIGHT

6.14 kg

INTERPRETED BY

Beth Johnson, DVM
DACVIM

IMAGING PERFORMED BY

Renee Trionfetti, VMD

HOSPITAL NAME

Blue Pearl Wyomissing

REFERRING VET

Dr. Heatherlynn
McFarlane

INVOICE

11161

DATE

1/20/2026

PRESENTING CLINICAL SIGNS

AUS to further evaluate acute onset of excessive vomiting, lethargy, loss of appetite that started 1/17/26. Acute marked liver enzyme elevations and marked GGT elevation. Currently seeing IM for a relapse of ITP that occurred Dec 2025 (ITP is back in remission, currently on prednisolone 7.5mg AM, 5mg PM, cyclosporine 25mg q12hr). Saturday, 1/17/26, began to vomit excessively, stopped eating, very lethargy. No history of dietary changes/indiscretion, access to medications, other toxins, plants, etc.

Exam findings: QAR to dull, dehydrated ~7%, mildly incr RR/RE but clear lung sounds, non-repeatable abdominal discomfort, weight loss of 0.6kgs in 2 weeks.

Chronic medications: Prednisolone 7.5mg AM, 5mg PM; Cyclosporine 25mg q12hr.

Current hospitalization: IVF (LRS and Plasmalyte to slowly incr Na) with KCl supplementation, Dexamethasone equivalent to prednisolone, Ondansetron, Buprenorphine.

Abnormal PE/Chem/CBC/UA Results: - CBC: WBC 24.88K (H), Neut 23.55K (H), Lymph 0.68K (L), Eos 0.04K, HCT 39.8%, PLT 554K - Blood smear: 28-31 PLT/HPF, one clump seen on feather edge, neutrophilia with no significant abnormal WBC morphology. Normal RBC morphology. - PCV/TS: 40%/9.2g/dL (H) - Chem: TP 7.7 (H), Alb 4.2 (H), Glob 3.5, Creat 0.7, BUN 32.8 (H), ALT 491 (H), ALP >993 (H), GGT >1200 (H), TBil 0.7 (H), Chol >450 (H), Ca 8.7 (L), Glu 535 (H), Phos 5.2 (H) - GGT (x2 dilution): 1904 (H) - ALP (x2 dilution): >1980 (H) - EPOC: pH 7.115 (L), PCO2 35.6, HCO3 11.4 (L), Creat 1.09, BUN 29 (H), Na 125 (L, corrected for hyperglycemia 131), K 1.6 (L), Cl 102 (L), iCa 1.16, Glu 535 (H) - IH cPLI: >2000 (H).

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

Urinary bladder is adequately distended. It has a normal uniform wall thickness. Contents include primarily anechoic fluid with moderate of echogenic non-shadowing debris, most consistent with exfoliated cells, mucous and/or small blood clots. Both sterile inflammation as well as urinary tract infection can present with echogenic debris. No masses or cystoliths are observed. The trigone and visible pelvic urethra are normal in thickness with a smooth mucosal surface.

Prostate is normal in size, echotexture, and echogenicity for a neutered male.

The right kidney is normal is size (5.35 cm), shape and echogenicity. It has smooth peripheral margination. There is a normal 1:3 cortex to medulla ratio with appropriate corticomedullary distinction. There is no evidence of pyelectasia, mineral or infarcts observed.

The left kidney is normal is size (4.87 cm), shape and echogenicity. It has smooth peripheral margination. There is a normal 1:3 cortex to medulla ratio with appropriate corticomedullary distinction. There is no evidence of pyelectasia, mineral or infarcts observed.

Adrenal Glands

The right adrenal gland is normal in size (0.43 cm at cranial pole and 0.41 cm at caudal pole), shape and overall architecture, echogenicity and echotexture. Visible surrounding vasculature appears normal.



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The left adrenal gland is normal in size (0.42 cm at cranial pole and 0.5 cm at caudal pole), shape and overall architecture, echogenicity and echotexture. Visible surrounding vasculature appears normal.

Spleen

The spleen is subjectively normal in size (0.9 cm thick at the hilus) 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 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. The wall of the gallbladder appears as a thin hyperechoic/calcified rim casting a distinct distal acoustic shadow. Some mineral/sand debris is suspected with no visible evidence of obstruction noted in these images at this time. 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 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.

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

Pancreas

The observed pancreas is prominent (enlarged) in size, hypoechoic to surrounding tissue and irregular in shape with a swollen undulating contour. Enhanced hyperechoic ill-defined surrounding fat is noted.

Free Abdomen

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

There is no apparent pathologic lymphadenopathy noted in these images.

ULTRASONOGRAPHIC FINDINGS

- The pancreatic changes are very mild/subtle but given patient's history, clinical signs, etc., mild to moderate or potentially emerging acute or acute on chronic low grade smoldering pancreatitis is suspected.
- Moderate gallbladder debris with porcelain gallbladder wall - Cholecystic debris is of unknown clinical significance. It can be seen with biliary stasis from fasting or illness. Cholecystic debris



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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. Porcelain (calcified) gallbladder is an uncommon finding in companion animals and has been observed as both an incidental finding and associated with biliary neoplasia. In humans, porcelain gallbladder can be a manifestation of chronic gallbladder disease, chronic cholecystitis, intramural hemorrhage with subsequent calcification, imbalances in calcium metabolism, and even giardiasis. This finding should be interpreted in combination with any clinical signs and/or laboratory changes suggestive of biliary disease and/or calcium dysregulation, etc. Some of the debris has a mineral/sand appearance with no visible evidence of obstruction noted in these images at this time.

- 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.
- A moderate amount of echogenic urinary bladder debris.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The appearance of the hepatobiliary system trends in appearance toward benign with top differentials, based on history and presenting complaint, for the liver enzyme changes being a new diabetic hepatopathy or steroid hepatopathy. Having said that, other pathologic disease can't be ruled out and tissue sampling via fine needle aspirates of the liver could be considered if patient's coagulation status is appropriate.

Patient's clinical illness based on history, may be in part secondary to emerging acute pancreatitis. Although the changes as described above are very mild/subtle. Therefore, other contributing factors to clinical illness including potentially ketoacidosis should be ruled out. If not already evaluated, assessment for ketones in the urine could be considered beginning with 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.

If not recently evaluated, a quantitative PLI is recommended if not already evaluated.

In the meantime, medical management of pancreatitis with anti-emetics, gastroprotectants, appetite stimulants or nutritional support as needed, pain management, broad spectrum antibiotics, and fluid therapy is recommended. Monitoring of the pancreas with power doppler is recommended to identify possible necrosis as well as other potential sequelae such as abscesses, etc. Insulin therapy may be warranted in addition to management of ketones, if present, etc.

Ultimately, in the newly diabetic patient with an acute increase in liver enzymes, steroids should be tapered as quickly as patient can tolerate, if possible.



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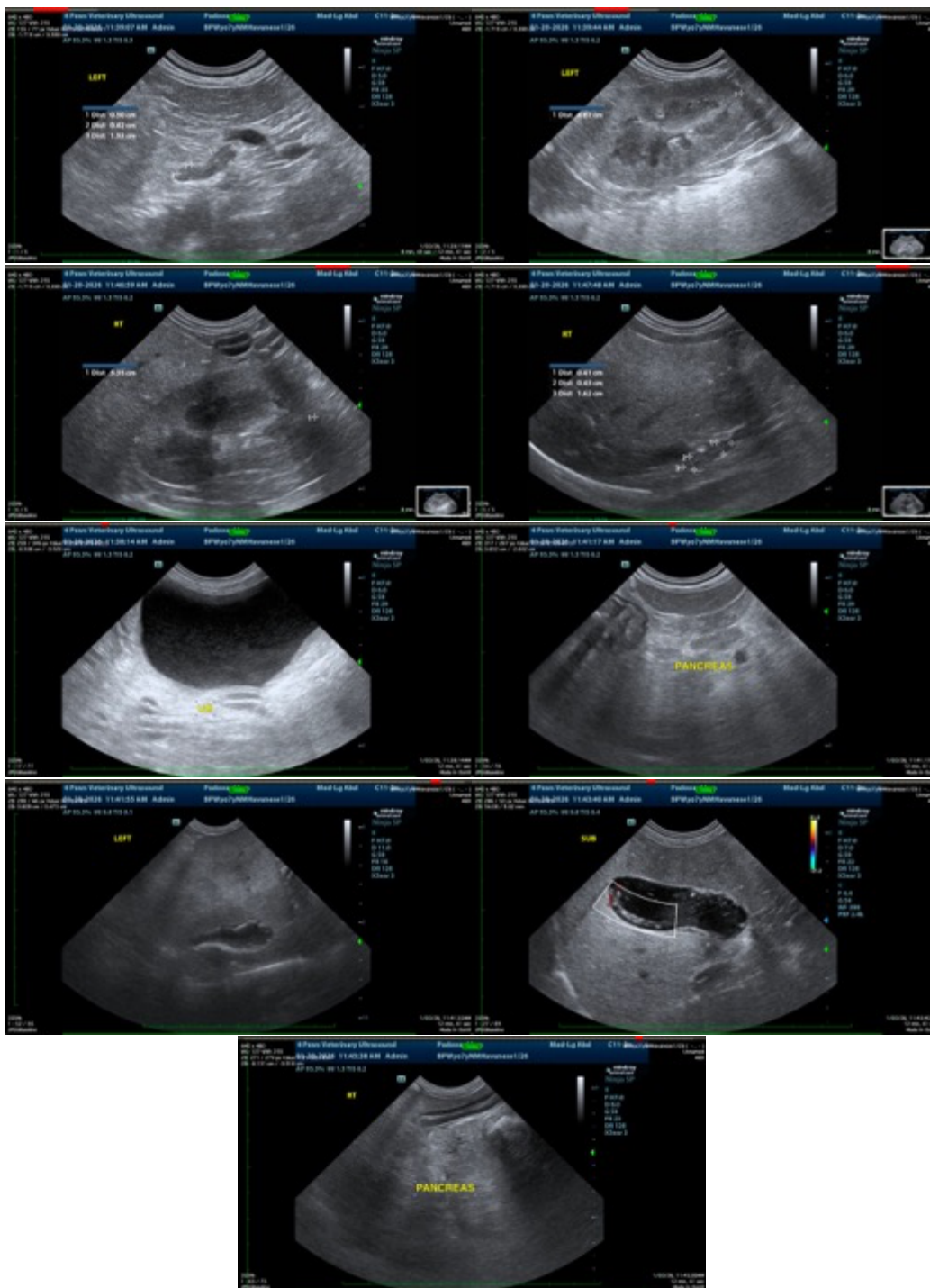
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.



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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