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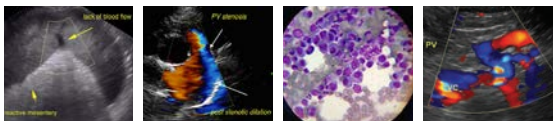

ANIMAL SOUNDS NW
MOBILE VETERINARY ULTRASOUND
IMAGING PERFORMED BY
AnimalSoundsNW.com 541-357-9423

PATIENT
DeDe Knuth

SPECIES
Canine

BREED
Miniature Pinscher

SEX
Spayed Female

AGE
10 Years

WEIGHT
14.6 Pounds

INTERPRETED BY
Beth Johnson, DVM DACVIM

IMAGING PERFORMED BY
Jenna Walsh, CVT

HOSPITAL NAME
Albany AH

REFERRING VET
Dr. Flanagan

INVOICE
45183

DATE
2/15/23

PRESENTING CLINICAL SIGNS

Intermittent vomiting Current Medications, previously cerenia Primary Question/Differential to Be Answered in This Exam R/o liver disease vs IBD, check on renal structures

Abnormal PE/Chem/CBC/UA Results: Alt 179, Ast 60, AlkP 317, GGt 33, Sdma 26, Bun 39, creat 1.2

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder is moderately 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 normal in size and contour. A relatively uniform hyperechogenicity is observed with mildly decreased corticomedullary distinction. There is no pyelectasia noted and no mineral is observed. No overt masses/nodules are observed. The left kidney measured 4.52 cm. The right kidney measured 4.84 cm.

Adrenal Glands

Adrenal glands are plump/swollen in size. Normal shape and contour are maintained without evidence of capsular invasion. Some parenchymal heterogenicity is present without concerning capsular distortion. Dystrophic mineralization is present in both adrenal glands. Visible surrounding vasculature appears normal. The right adrenal gland measures 2.32 cm long x 0.57 cm at the cranial pole and 0.91 cm at the caudal pole. The left adrenal gland measures 2.28 cm long x 0.72 cm at the cranial pole and 0.70 cm at the caudal pole.

Spleen

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. Multifocal mineral foci are noted. 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 mildly overdistended with a moderate amount of non-dependent, mildly aggregated/inspissated sludge. Hypo to anechoic cystic areas are noted between the gallbladder sludge and luminal wall. The wall is otherwise smooth without visible thickening. There is no evidence of cystic or CBD dilation. There is no evidence of effusion.

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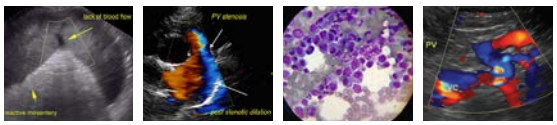

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Gastrointestinal

The stomach wall is normal in thickness (canine < 0.5 cm and feline < 0.4 cm) 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 (canine duodenum < 0.5 cm and feline duodenum < 0.4 cm; other < 0.3 cm). 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 pancreatic parenchyma is appropriately isoechoic to surrounding tissue. Visible capsule is smooth and normal in contour. There is no visible pancreatic duct dilation. There is no evidence of active peripancreatic inflammation.

Free Abdomen

There is no evidence of free peritoneal effusion noted in these images.

There is no apparent lymphadenopathy noted in these images.

ULTRASONOGRAPHIC FINDINGS

- **Emerging mucocele –** 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. The non-dependent nature of this sludge combined with the cystic areas are suggestive, however, of possible emerging cystic mucosal hyperplasia or early gallbladder mucocele.
- **Bilateral adrenomegaly –** consistent with adrenal hyperplasia secondary to pituitary dependent hyperadrenocorticism vs stress or normal variant. Interpret in combination with clinical signs of hyperadrenocorticism.
- **Nephritis –** This appearance can be consistent with chronic interstitial nephritis or glomerulonephritis. Toxic insult and/or infectious disease (pyelonephritis, Leptospirosis, etc.) cannot be ruled out. This finding should be interpreted in combination with suspicion for renal disease and/or supporting laboratory or urinalysis changes.
- **Spleen mineralization –** This is a benign change but can be associated with endocrinopathies, especially hyperadrenocorticism.
- **Hyperechoic hepatomegaly -** This appearance is non-specific and most consistent with a benign steroid (endocrine) or vacuolar hepatopathy or reactive or idiopathic hepatopathy. Inflammatory and/or infiltrative disease (such as round cell neoplasia) are also possible, but considered less likely.

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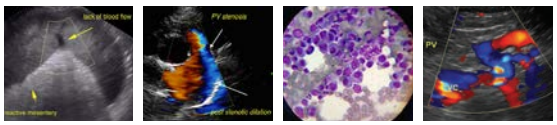

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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Given this patient's mild azotemia, if not recently evaluated, and to help better determine pre- versus renal azotemia, a urinalysis and, if indicated based on urinalysis results, urine culture are recommended. If protein is present in an otherwise quiet sediment, protein quantification with a urine protein to creatinine ratio is recommended.

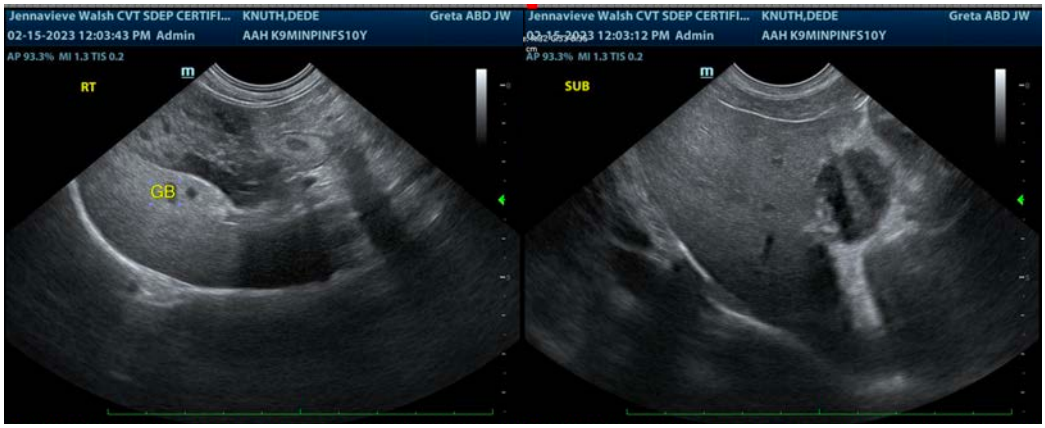
Especially if this patient is isosthenuric, testing for Leptospirosis is recommended.

The additional pathology described above including adrenal glands, liver, gallbladder, spleen, etc. are all suggestive of hyperadrenocorticism, likely pituitary dependent hyperadrenocorticism, which may also be causing the reported mildly increased liver enzymes. However, hyperadrenocorticism doesn't typically result in vomiting, and should not be pursued in the face of concurrent illness due to the likelihood of a false positive result.

Given this patient's vomiting, further diagnostic recommendations prior to working up hyperadrenocorticism include a gastrointestinal malabsorption panel (including cobalamin, folate, TLI and PLI) to Texas A&M GI Laboratory.

Additionally, if there is any evidence of cranial abdominal pain or clinical suggestion that the gallbladder could be playing a role in the nausea, further management of the emerging mucocele would be recommended. Depending on clinical signs, this could be a conservative approach, beginning with Ursodiol, broad-spectrum antibiotics, and supportive care of the GI signs while being monitored for improvement. However, if clinical signs persist and/or liver enzymes progress, etc., surgical intervention including cholecystectomy may ultimately be warranted.

Once this patient's current illness is resolved, and when and if clinical signs of hyperadrenocorticism develop down the road, testing in the form of a low-dose Dexamethasone suppression test would be recommended at that time.



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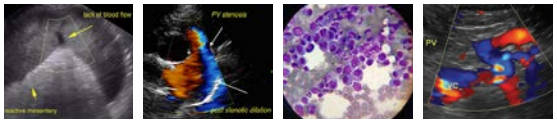

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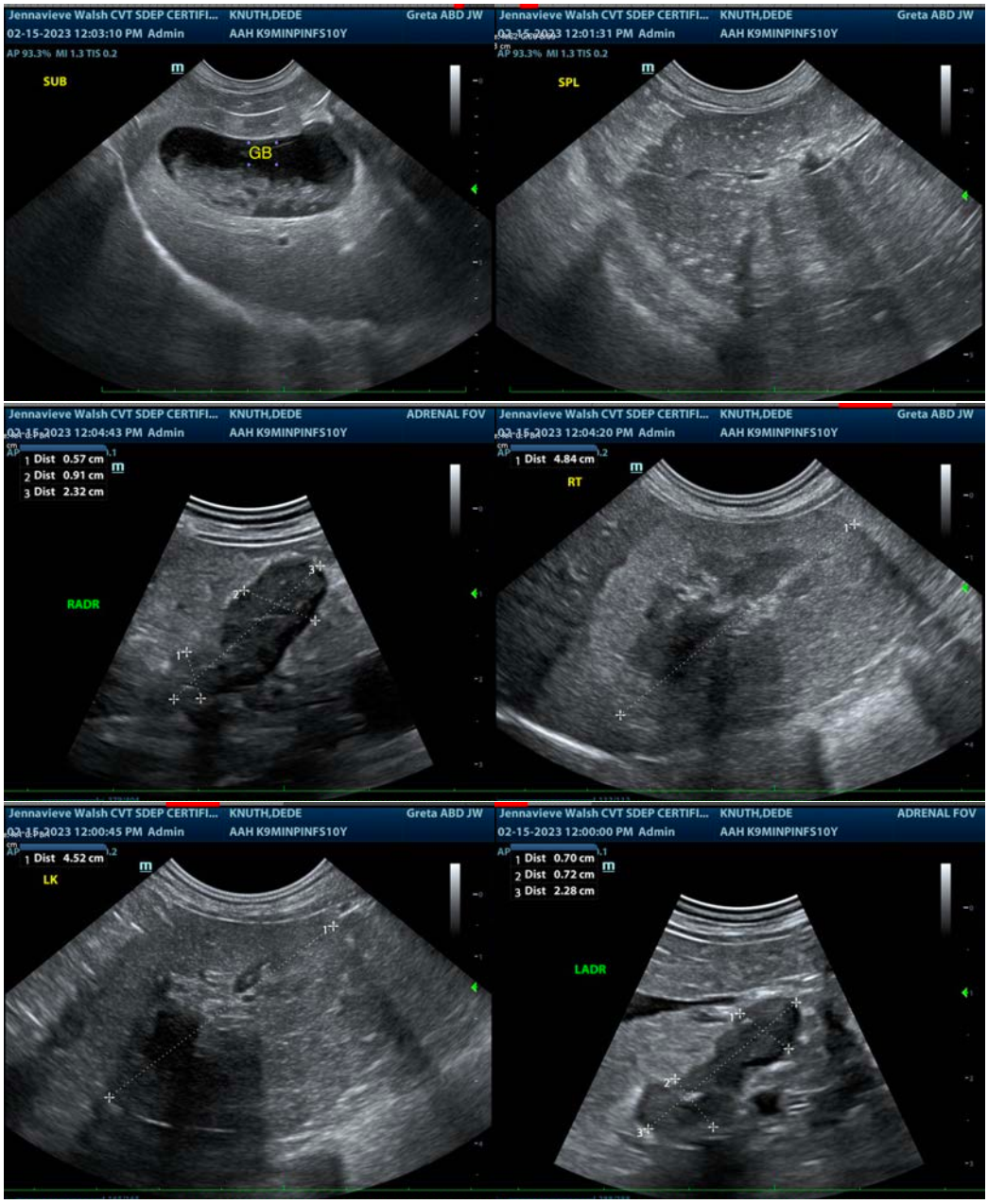

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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
Beth.Johnson@sonopath.com