

PATIENT

Brandy Lee

SPECIES

Canine

BREED

Yorkshire Terrier

SEX

Spayed Female

AGE

12 years 2 months

WEIGHT

12.2 lbs

INTERPRETED BY

Eric Lindquist, DMV,
DABVP(CFM), Cert.
IVUSS

**IMAGING
PERFORMED BY**

Eric Lindquist, DMV,
DABVP(CFM), Cert.
IVUSS

HOSPITAL NAME

Fidelis Animal Hospital

REFERRING VET

Dr. Kathrine Roe-
Jarisch

INVOICE

11037

DATE

1/7/2026

PRESENTING CLINICAL SIGNS

Elevated total protein and globulins. ALT 194, ALP 212, GGT 13, Cholesterol 545, Triglycerides 670, PSL 659. USG 1.023 w/3+ protein.

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder** was structurally normal. The pelvic urethra was imaged 3.0 cm beyond the cystourethral junction. Small calculus was noted measuring 2.0 mm, likely passing periodically.

The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some age-related loss of curvilinear patterns regarding the capsule and C/M junction. The cortices presented largely uniform texture with some increased echogenicity expected for his age patient. Medullary structure differed distinctly from that of the cortex and no evidence of pelvic dilation was present. Dystrophic mineralization was noted and non-obstructive at this time. Left kidney measures 4.5 cm. Right kidney contained multiple infarcts and calculi with the largest calculus measuring 0.4 cm, and the right kidney measures 4.37 cm.

Adrenal Glands

The **adrenal glands** appeared slightly enlarged and swollen. No evidence of focal capsular expansion or invasion into the phrenic veins was noted. No overt suspicion of neoplasia was noted. This is considered likely a hyperplastic change associated with stress or adrenal endocrinopathy (PDH). If isosthenuria is persistently present and the patient morphologically suggests Cushing's disease, then ACTH testing would be indicated. Left adrenal measures 1.99 cm x 0.82 cm at the caudal pole and 0.8 cm at the cranial pole. Right adrenal measures 2.23 cm x 0.6 cm at the caudal pole and 0.97 cm at the cranial pole.

Spleen

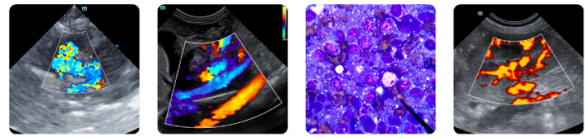
The **spleen** presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes were noted.

Liver

The **liver** was uniformly swollen and presented coarse architecture with mildly increased portal markings and subtle, mixed echogenic changes. This is consistent with vacuolar hepatopathy and some level of remodeling and history of inflammatory component. There was no overt suspicion of neoplasia. Likely reactive hepatopathy owing to the pancreatic presentation.

The gallbladder was mildly over distended with suspended and dependent debris, yet not to the level of emerging mucocele, yet sludge appears to be mildly excessive. No adjunctive inflammation was noted.

Gastrointestinal



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Examination of the **gastrointestinal tract** revealed minor upper GI thickening without loss of mural detail and a 1.5 cm slight shadowing structure likely dissolving medication. Colonic wall was mildly thickened at the proximal descending colon.

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Pancreas

The **pancreas** revealed extensive mixed echogenic hypoechoic edematous parenchyma with hyperechoic surrounding fat and minor pockets of free fluid most consistent with subacute on chronic pancreatitis. However, the free fluid is concerning. Pancreatic pathology was hyper vascular on power doppler assessment.

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Other

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Slight comet tail lung pattern noted through the lung fields.

ULTRASONOGRAPHIC FINDINGS

AGE

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- Small bladder calculus, non-obstructive. Likely small enough to pass without difficulty.
- Renal calculi, moderate degenerative changes and infarcts on the right, mild to moderate on the left.
- Subacute on chronic pancreatitis, extensive.
- Upper GI thickening with colonic thickening.
- Benign hepatopathy with minor excessive coalesced bile. Likely reactive hepatopathy as cause of liver enzyme elevation.
- Emerging PDH suspected in this patient/Cushing's. No evidence of tumors.
- Comet tail lung pattern.

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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Recommend diet change in this patient to hydrolyzed diet with Purina HA or Royal Canin HP, or similar. Blood pressure measurements indicated. Broad spectrum antibiotics, ursodiol therapy indicated. Note that positive Murphy sign was present throughout the imaging of the pancreas. Re-check sonogram in 10 – 14 days. Ultrasound guided FNA of the pancreas may be necessary in this patient depending on progress or response to therapy. If any weight loss or progressive anorexia is an issue, then FNA indicated. There's a potential for pancreatic carcinoma. Recommend a workup for Cushing's after the pancreatic pathology has been resolved.

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Efficient & Accurate Cushing's Work up-Lindquist

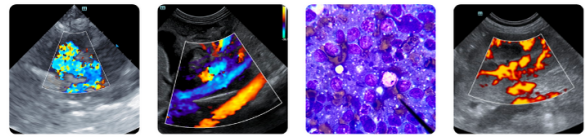
Notes regarding Cushing's Clinical Presentations:

Nearly all Cushing's dogs have SAP elevations and true PU/PD (USG < 1.025) and most are polyphagic. Cushing's dogs are > 6 years and usually > 9 years old, usually have poor skin coats, body scores > 3/5, and are usually sedentary animals.

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Its important to remember that Cushing's dogs usually look and play the part and other diseases cause false + stress related cortisol spikes. On rare occasion a Cushing's dog will not follow the rules but this is truly an exception.



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Potential Cushing's patient workups can be costly and frustrating if not definitive and, in my experience, the non-definitive patient usually has something else going on that may be contributing to some of the clinical signs a Cushing's dog will have, especially SAP elevations or PU/PD. Based on this prelude of information I came up with the following algorithm in the spirit of diagnostic efficiency.

The following suggested protocol is based on current available literature on Cushing's disease and extensive clinical-sonographic experience evaluation + Cushing's and False + LDDST & ACTH stim. cases in order to maximize the efficiency of a Cushing's workup in practice.

Screen first, workup second

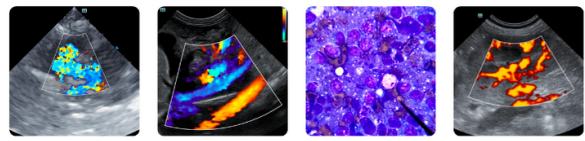
1) **UA:** Repeatable (2-3 urine samples) Urine specific gravity & urine cortisol/creatinine ratio (UCCR): If **repeatable USG < 10.20 and + UCCR** move to next step 2.

Note: UA is inexpensive and easy to obtain and if UA criteria is not met for Cushing's then resources can be spent into other more pertinent diagnostics or left on hold until the UA criteria is met in emerging Cushing's cases.

2) **Sonogram:** Does the patient **have concurrent disease** clinically or sonographically as non-Cushing's illness will influence the potential false + LDDST or even ACTH stim. The sonogram gives a global perspective of the internal health of the patient to be considered in the Cushing's workup as an assessment of concurrent disease. Is there a concurrent neoplastic process, UTI pancreatitis, mucocele....? Are the adrenals enlarged (Cushing's-PDH, stress, age related or breed variant), or atrophied (iatrogenic Cushing's or adrenal burnout), have asymmetric enlargement (Adrenal tumor, hyperplasia, adenoma, age related variant), or is there vascular invasion (Invasive pheo with false + UA criteria or adenocarcinoma or phrenic thrombosis)? The sonogram answers these questions proactively.

Address & treat concurrent disease first before performing Cushing's testing or testing will be artificially altered increasing false negatives and positives.

3) **LDDST** (0.01 D-Sodium phosphate mg/kg IV **with precise dosing******) (Better screening test but plagued with false + but considered more specific than ACTH stim) Use if there is potential early Cushing's or if adrenal asymmetry present on sonogram suspecting tumor. Use LDDST in cats at a higher dose (0.1 mg/kg IV). **Interpretation LDDST:** Look at 8-hour post first: If > 1.4 = Cushing's. Then look at 4-hour: if > 1.4 or > 50% baseline = Cushing's. 4-hour do then 8-hour spike most consistent with PDH. Flat line high constant curve without dip more consistent with tumor but can be PDH. See attached graph.



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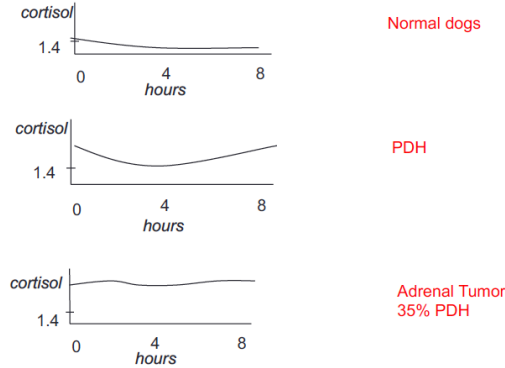
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LDDS



Courtesy: Rebecca Berg DACVIM, DECVIM

4) **ACTH stim.** (Better confirming test but can have false +) Use if the patient “looks” Cushingoid or if bilateral adrenal enlargement is present, or high normal width on sonogram, or if iatrogenic Cushing’s suspected (Cortisone Tx in past). ACTH stim is better for diagnosis of Addison’s, iatrogenic Cushing’s, and Cushing’s therapy monitoring but problematic with initial Cushing’s diagnosis. First dx LDDST is suggested.

5) If **diabetic** then run both LDDST & ACTH stim but stabilize as much as possible first.

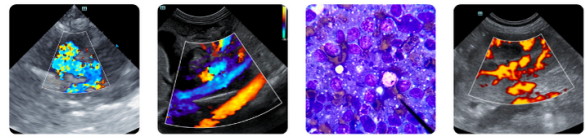
5) Run a **serial blood pressure** in a BP friendly non “white coat effect” atmosphere. Run at least 3 at different times over a few hours or when eating as the patient tends to be calm when eating or give Torbutrol when entering the facility. Cushing’s hypertension is usually 150-180 systolic range while pheochromocytoma range is more often > 180 systolic.

6) **Perform CT** of the pituitary to identify macro adenoma expansion if any lethargy or dullness or other central clinical CNS signs are minimally present. CT for adrenal may be more thorough for adrenalectomy surgical planning if ultrasound views of the CVC were problematic.

7) **Adrenalectomy** for adrenal mass is prescribed then it is essential to stabilize the patient first regarding secondary disease such as organ dysfunction, hypertension, diabetes mellitus, hypernatremia, thromboembolic risk urinary and other infection in order to minimize potential for operative and postoperative complications as they are common in adrenalectomy. Trilostane stabilization therapy for Cushing’s would be the first approach then address surgery and hypertension should be managed ideally < 160 systolic with ace inhibitors, phenoxybenzamine, or amlodipine.

Suggested reading:

Behrend EN, Kooistra HS, Nelson R, et al. Diagnosis of Spontaneous Canine Hyperadrenocorticism: 2012 ACVIM Consensus Statement (Small Animal). J Vet Intern Med 2013;27:1292-1304 .



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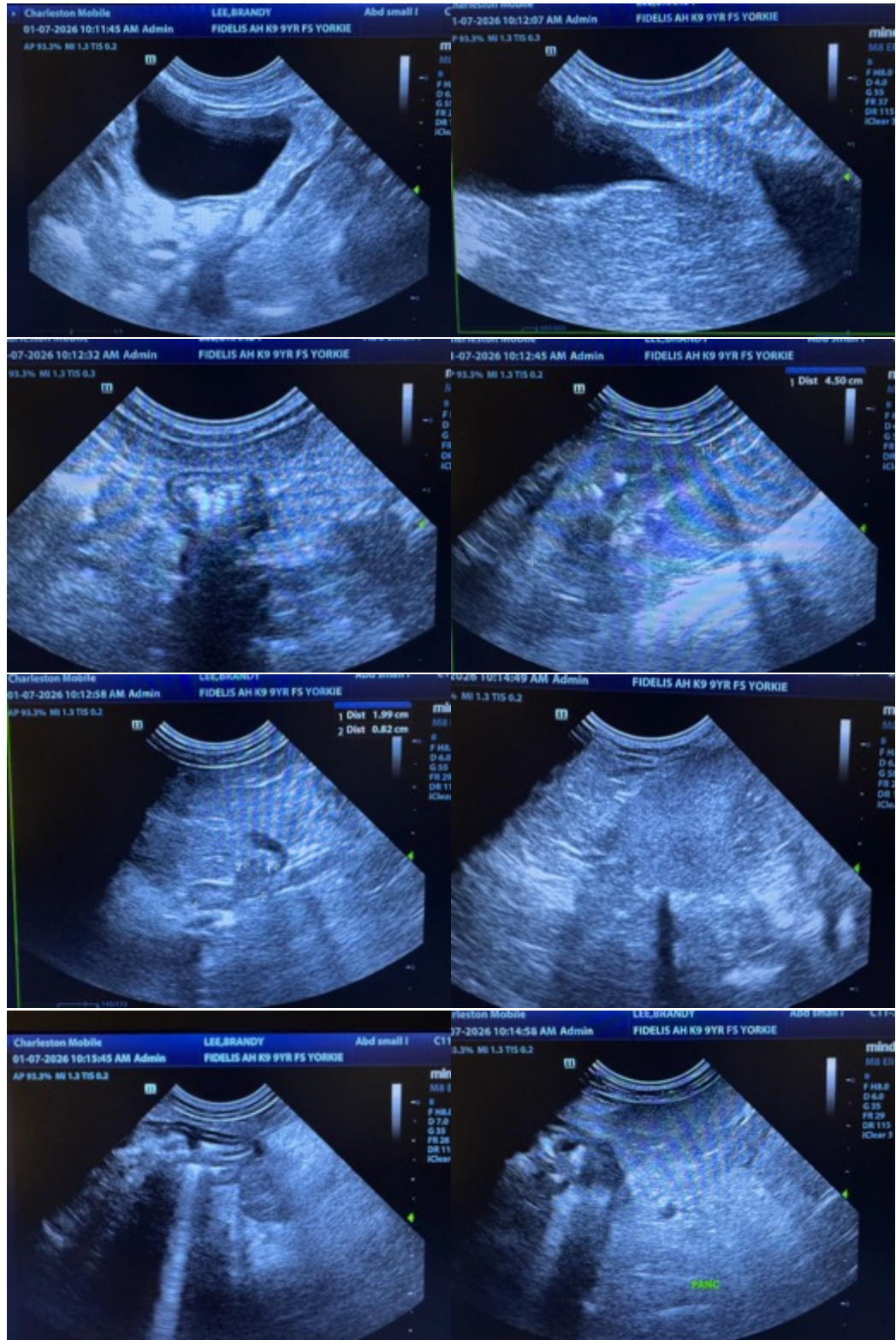
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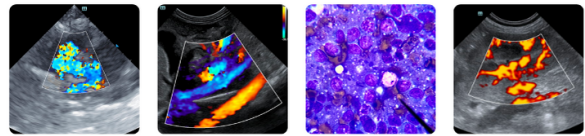
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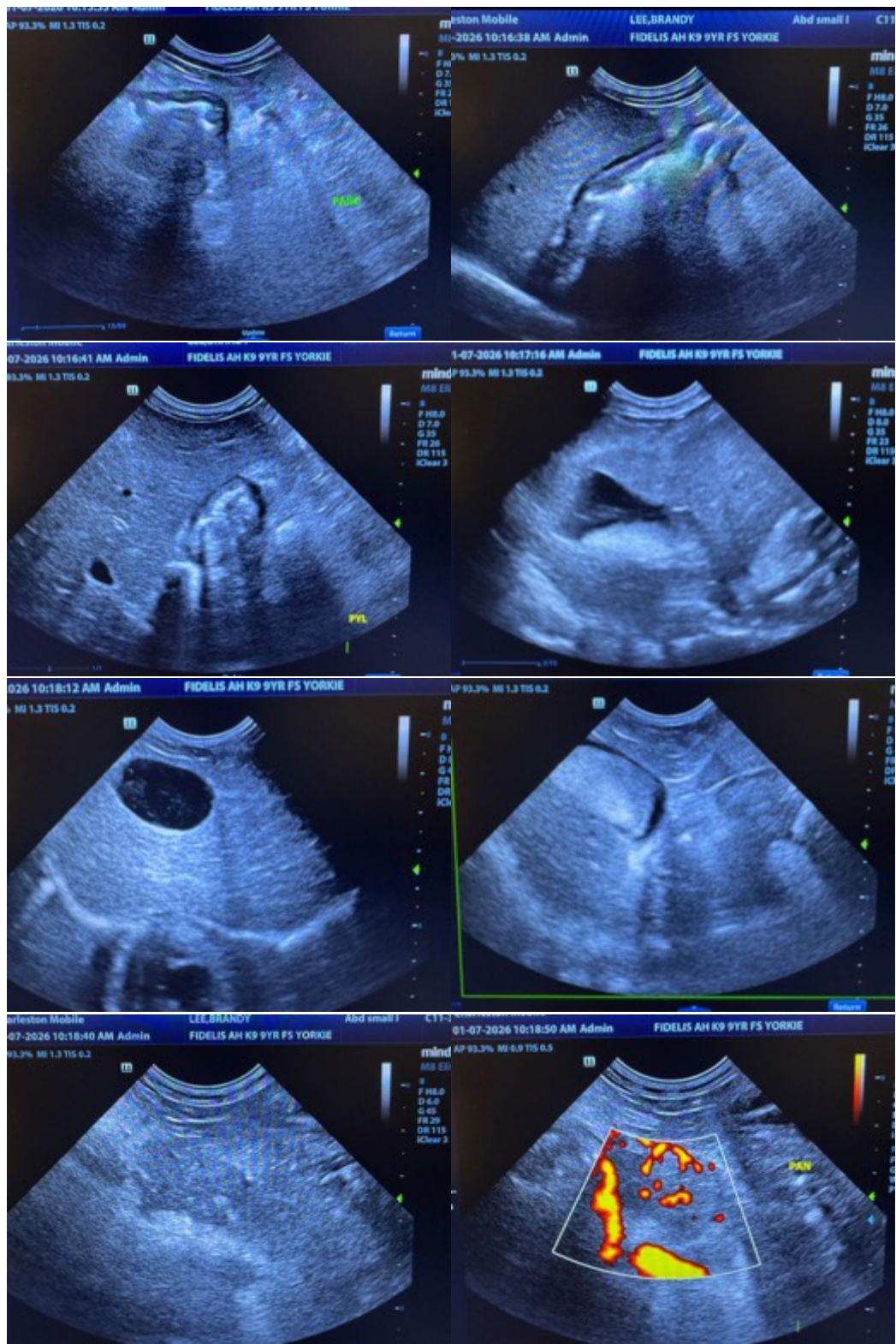
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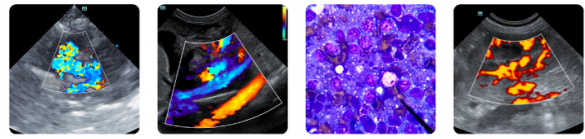
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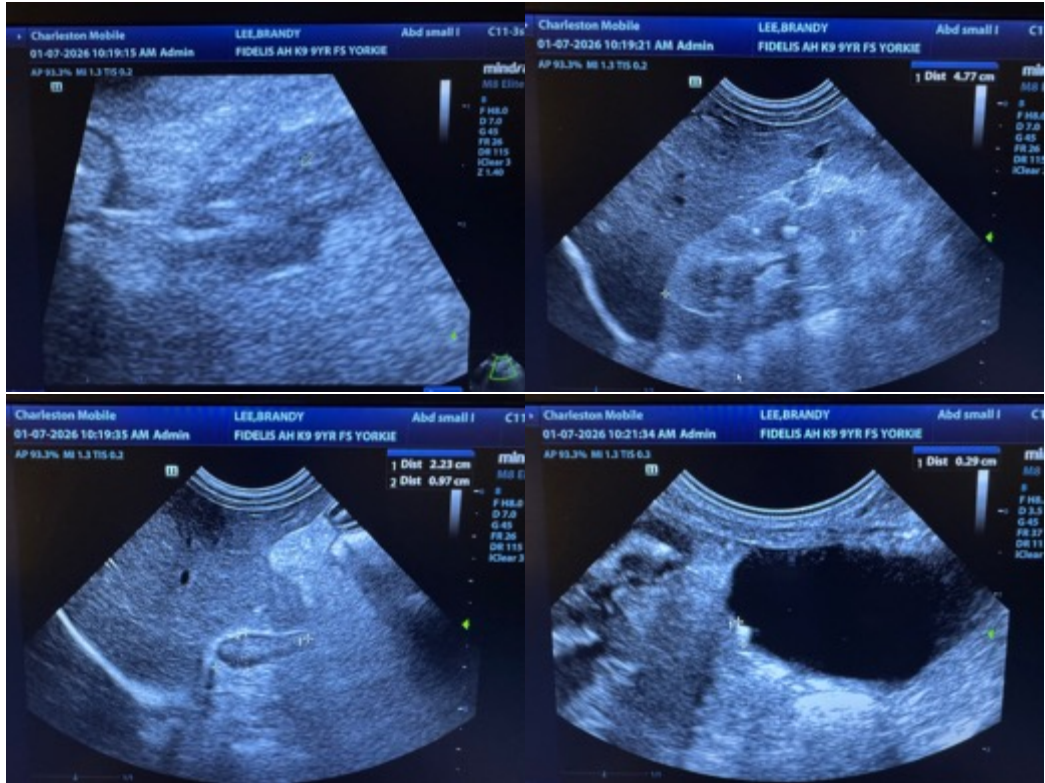
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Eric Lindquist, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com

info@SonoPath.com