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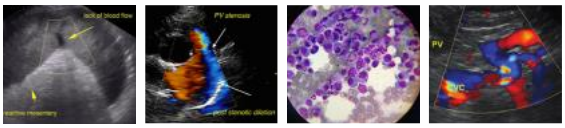

ANIMAL SOUNDS NW
MOBILE VETERINARY ULTRASOUND
IMAGING PERFORMED BY
AnimalSoundsNW.com 541-357-9423

PATIENT

J.D. Thompson

SPECIES

Canine

BREED

Beagle Mix

SEX

Spayed Female

AGE

14 Years

WEIGHT

32 Lbs.

INTERPRETED BY

Eric Lindquist, DMV, DABVP, Cert. IVUSS

HOSPITAL NAME

The Veterinary Hospital

REFERRING VET

Dr. Johnson

INVOICE NUMBER

13443

DATE

1/14/22

PRESENTING CLINICAL SIGNS

History: presented for mobility/pain management/ weakness/ ataxia - labs revealed concerns for hepatic disease

Abnormal PE/Chem/CBC/UA Results:labs done 11/21/21 (previous valuse were 9/14/21) - elevated ALT 767 (was 576) Alk Phos 454 (was 291) GGT 44 (was 30) Chol 438 Trig 250 Current Medications Tramadol, Gabapentin, chinese herbal 'Hindquarter Weakness'

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. The ureters were not visible which is normal. No uroliths or sediment were visualized and anechoic urine was present. No evidence of inflammatory or neoplastic changes were noted. Ureteral papillae were normal. The pelvic urethra was imaged 2.0 cm beyond the cystourethral junction.

The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some moderate 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. The left kidney measured 4.73 cm. The right kidney measured 6.36 cm.

Adrenal Glands

The **adrenal glands** appeared slightly enlarged and swollen. No evidence of focal capsular expansion or invasion into the phrenic veins were noted. No overt suspicion of neoplasia was noted. This is considered likely a mild 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. The right adrenal gland measured 2.42 cm x 1.04 cm at the cranial pole and 0.61 cm at the caudal pole. The left adrenal gland measured 2.41 cm x 0.93 cm at the cranial pole and 0.58 cm at the caudal pole.

Spleen

The **spleen** was largely smooth with subtle heterogeneous parenchymal changes while maintaining normal echogenic relationship to the liver and kidney. These changes are consistent with normal mild age-related alteration. The capsule was smooth without noticeable impingement from within the spleen or from pathology in the adjacent abdomen. The splenic vasculature demonstrated normal volume without signs of congestion or significant contraction. No evidence of active acute or chronic inflammatory, neoplastic, or infarctual changes were noted. Cranial folding of the spleen was noted.

Liver

The **liver** was uniformly enlarged with minor uniformly coarse architecture with occasional hyperechoic lipogranulomatous type nodules with variable swelling, consistent with vacuolar hepatopathy and remodeling/nodular hyperplasia. FNA could be considered for further definition. Neoplasia is unlikely. The gallbladder and common bile duct were unremarkable.

Gastrointestinal

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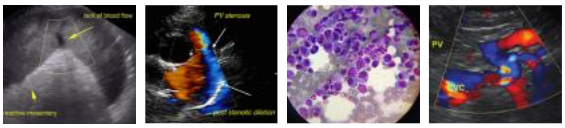

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Examination of the **gastrointestinal tract** revealed a stomach and intestine free of stasis, of normal wall thickness, acceptable curvilinear mural detail, and peristaltic activity. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively. No obstructive or overt infiltrative disease was noted. No associated abnormal lymphatic activity was noted.

Pancreas

The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Some mild parenchymal remodeling, however, with mild deviation from curvilinear normalcy was observed. Pancreatic duct and capsular irregularities were present consistent with age related changes. If pain upon imaging (+ Murphy sign) was present or if the patient is focally painful in subxyphoid palpation then low-grade smoldering chronic pancreatitis should be suspected.

ULTRASONOGRAPHIC FINDINGS

- Benign hepatopathy/nodular hyperplasia pattern
- Age-related renal changes
- Bilateral adrenal hypertrophy, consistent with pituitary dependent hyperadrenocorticism, if all cushingoid parameters are present.
- Pancreatic remodeling. A history of pancreatitis is likely in this patient. Minor low-grade inflammation possible.

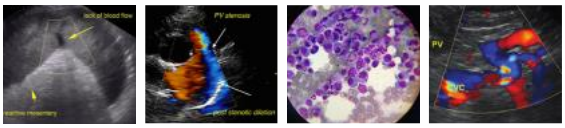
INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

No evidence of neoplasia. I do not believe that the liver is overtly responsible for the weakness and ataxia, however, bile acid profile could be considered for further definition. Assessment for orthopedic, CNS or thoracic disease would be indicated. Leptospirosis titers warranted given the ALT elevations. FNA of the liver warranted for further definition. The liver is not likely to be the primary cause of the clinical signs, however, if an acute insult has occurred given the ALT elevations, it may be an underlying issue. Bile acid profile would help differentiate the benign changes from functional changes.

Cushing Work UP

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.
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.
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.



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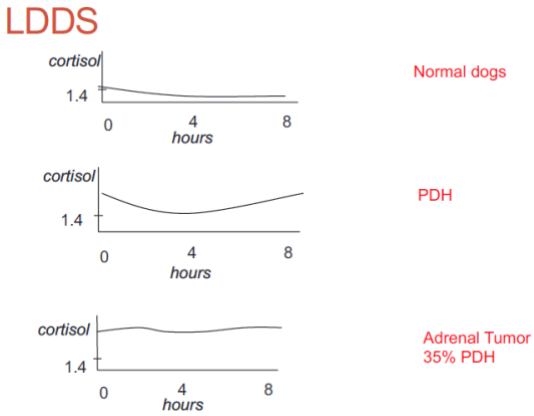
The following suggested protocol is based on current available literature on Cushing's disease and extensive clinical-sonographic experience evaluation + Cushing's and False + LDDST & ACTH stim. cases in order to maximize the efficiency of a Cushing's workup in practice.

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.

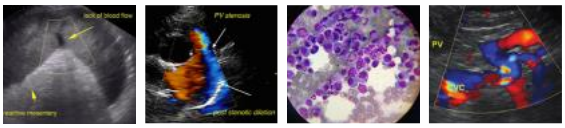
2) **Sonogram:** Does the patient **have concurrent disease** clinically or sonographically as non-Cushing's illness will influence the potential false + LDDST or even ACTH stim. The sonogram gives a global perspective of the internal health of the patient to be considered in the Cushing's workup as 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). **Interpetation 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.



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 Addisons, Iatragenic 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



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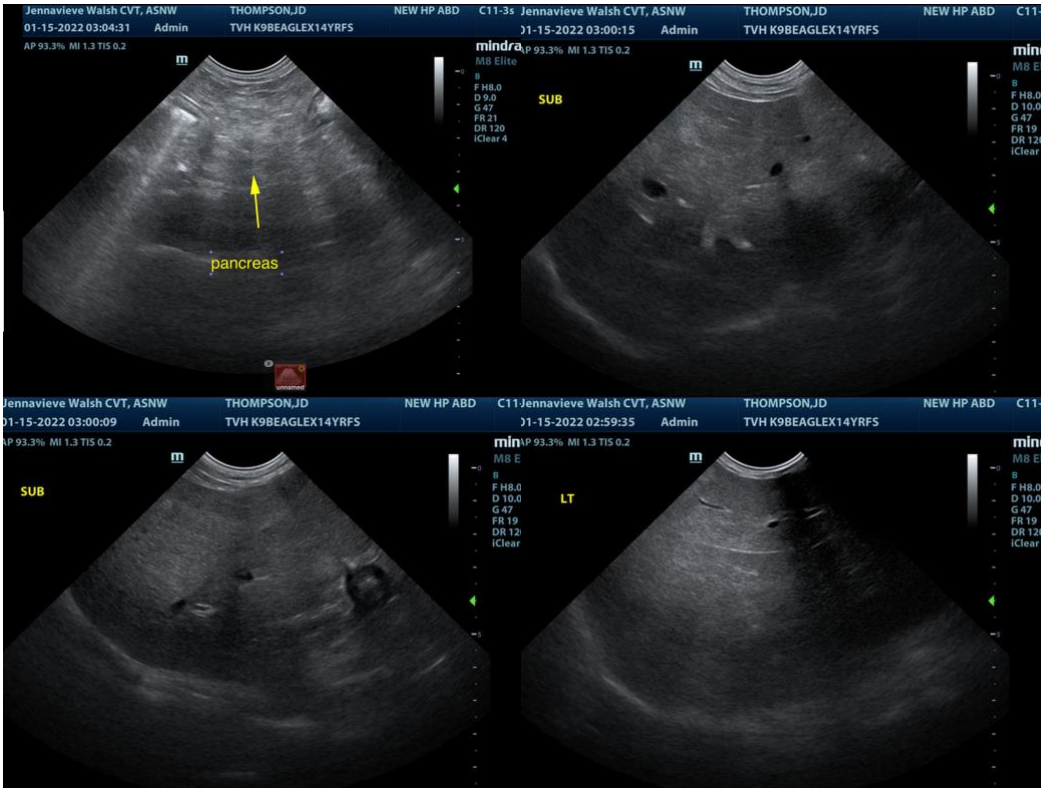
eating or give Torbutrol when entering the facility. Cushing's hypertension is usually 150-180 systolic range while pheochromocytoma range is more often > 180 systolic.
6) **Perform CT** of the pituitary to identify macro adenoma expansion if any lethargy or dullness or other central clinical CNS signs are minimally present. CT for adrenal may be more through for adrenalectomy surgical planning if ultrasound views of the CVC were problematic.
7) **Adrenectomy** 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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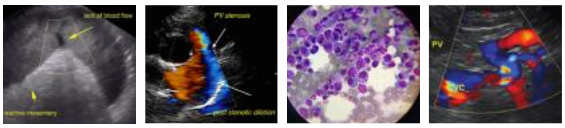

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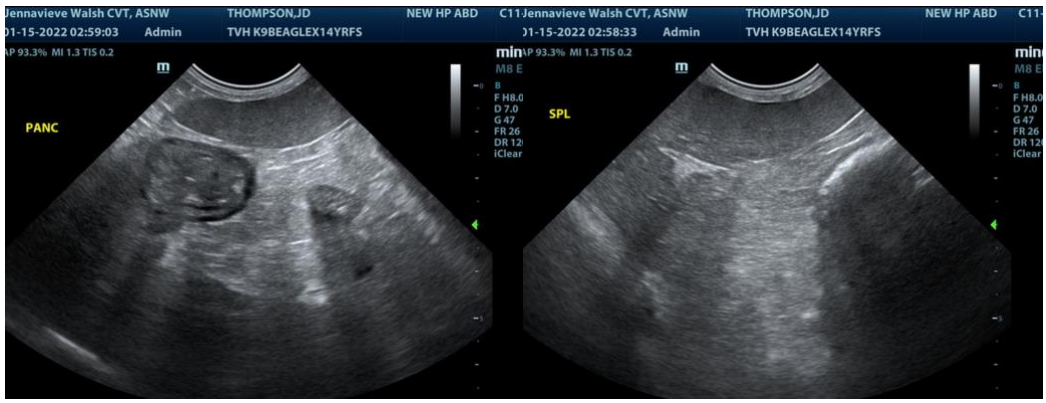

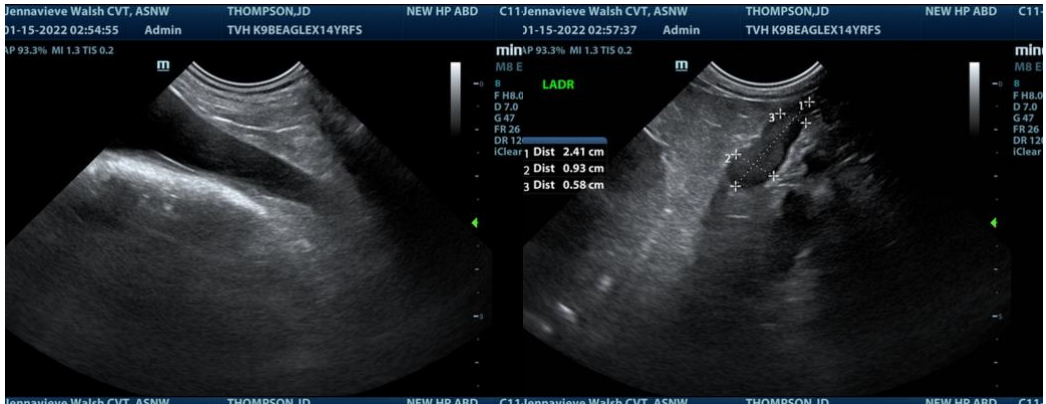

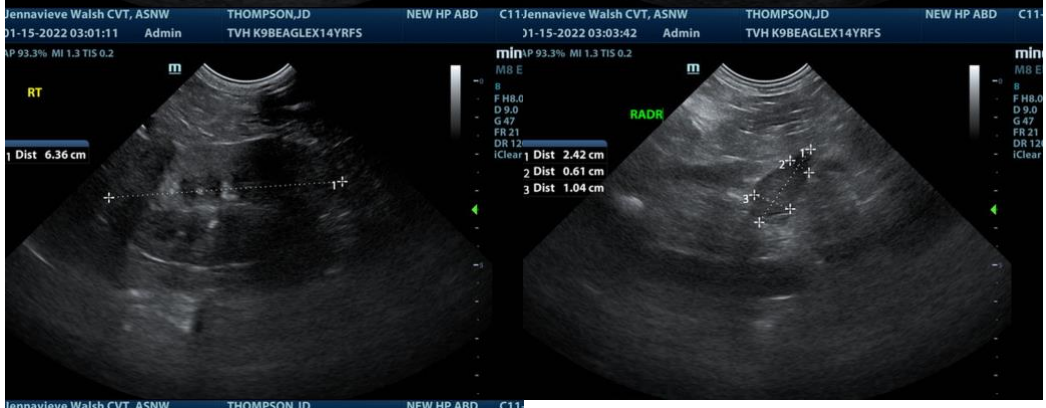

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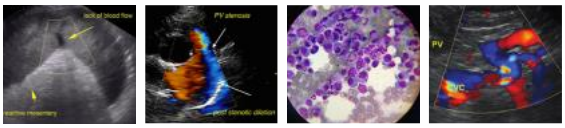

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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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
Eric.Lindquist@SonoPath.com