



**PATIENT**

Heidi Fernandez

**SPECIES**

Canine

**BREED**

Bichon Frise

**SEX**

Spayed Female

**AGE**

13 Years 3 Months

**WEIGHT**

10 Pounds

**INTERPRETED BY**

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

**IMAGING PERFORMED BY**

Vincent Ravancho, CVT

**HOSPITAL NAME**

Jersey City AH

**REFERRING VET**

Dr. Jiminez

**INVOICE**

35341

**DATE**

1/9/26

**PRESENTING CLINICAL SIGNS**

History: Pre-ax labs reveal ALP = 3155 Clinical findings: Hepatomegaly, mammary mass, mildly distended abdomen.

Abnormal PE/Chem/CBC/UA Results: ALP 3155, TP 8.2, Glob 5.1, A:G Ratio 0.8, Chol 375, Trig 769

**ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN**

**Urinary System**

The **urinary bladder** and visible pelvic urethra were unremarkable for the level of repletion presented. The urine, however, did present some mildly echogenic debris consistent with mucous, exfoliated cells from renal or bladder origin, and/or blood clots as these echogenic changes can all present similarly. This is often related to urinary tract infection but may represent simple evidence of exfoliated debris or sterile inflammation. Cystocentesis, urinalysis, +/- culture would be recommended to rule out and define any UTI. This is a mild change. The pelvic urethra was imaged 1.0 cm beyond the cystourethral junction.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex. The capsules were acceptably uniform without significant irregularities. The right kidney measured 5.2 cm. The left kidney measured 5.0 cm. Slight pyelectasia was noted in the left kidney. Slight mineralizations were noted bilaterally.

**Adrenal Glands**

Both **adrenal glands** were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The left adrenal gland measured 1.8 cm x 0.49 cm at the caudal pole and 0.39 cm at the cranial pole. The right adrenal gland measured 2.11 cm x 0.82 cm at the cranial pole and 0.67 cm at the caudal pole. A hyperechoic 1.0 cm nodule was noted at the cranial pole of the right adrenal gland. The vena cava was free of evident pathology despite the right adrenal nodular change.

**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** revealed hypoechoic macronodular changes (3.1 cm) in the left liver without significant disruption of architecture. Other hypoechoic nodular changes were noted in the liver. This is most consistent with nodular hyperplasia, however, cannot rule out an emerging neoplastic event. The gallbladder and common bile duct were unremarkable.



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**Gastrointestinal**

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. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

**ULTRASONOGRAPHIC FINDINGS**

- Right adrenal nodule – likely adenoma or hyperplasia, invasive
- Nodular hepatic changes in the midst of subjectively benign hepatopathy
- Moderate degenerative renal changes with pyelectasia
- Urinary bladder debris

**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**

FNA of the liver nodules would be ideal yet subjectively benign. Assessment for UTI is indicated. Full urinary work up is warranted if not already performed. If urine specific gravity is <1.020, then work up for Cushing's is indicated.

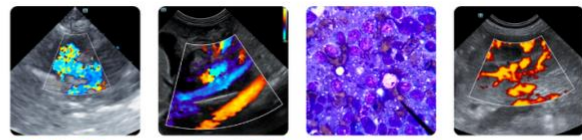
Note that 30% of Addisonian dogs are atypical and have normal sodium potassium ratios. Screening can be performed with a urine cortisol to creatinine ratio (UCCR) of less than 2.0 ug/dl is indicated as a screening for Addison's. This has near a 100% negative predictive value. UCCR less than 1.4 ug/dl is 100% sensitive and 97 % specific for Addison's. If the UCCR is greater than 2.0 ug/dl and Addisonian signs are present, then disease induced adrenal burnout may be the case. UCCR measures a 12-hour cortisol whereas baseline cortisol is a moment in time and fluctuates. Therefore, a UCCR is more sensitive and specific than baseline cortisol. Otherwise, baseline cortisol could be utilized if > 2.0 then this is negative also for Addison's, yet less sensitive and specific. Therefore, baseline UCCR is considered the best screening test. Therefore, if UCCR is less than 2.0 then full ACTH stimulation would be recommended for the diagnosis of Addison's. This is based on Del Baldo, et.al JVIM 2022

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.



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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.*

*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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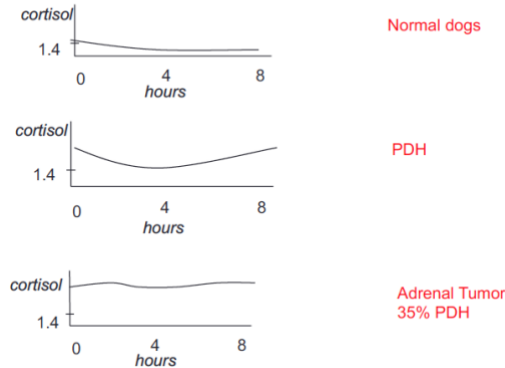
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**LDDS**



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 Addisons, 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.

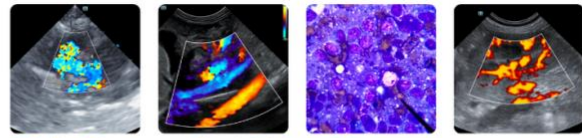
5) Run a **serial blood pressure** in a BP friendly non “white coat effect” atmosphere. Run at least 3 at different times over a few hours or when eating as the patient tends to be calm when eating or give Torbutrol when entering the facility. Cushing’s hypertension is usually 150-180 systolic range 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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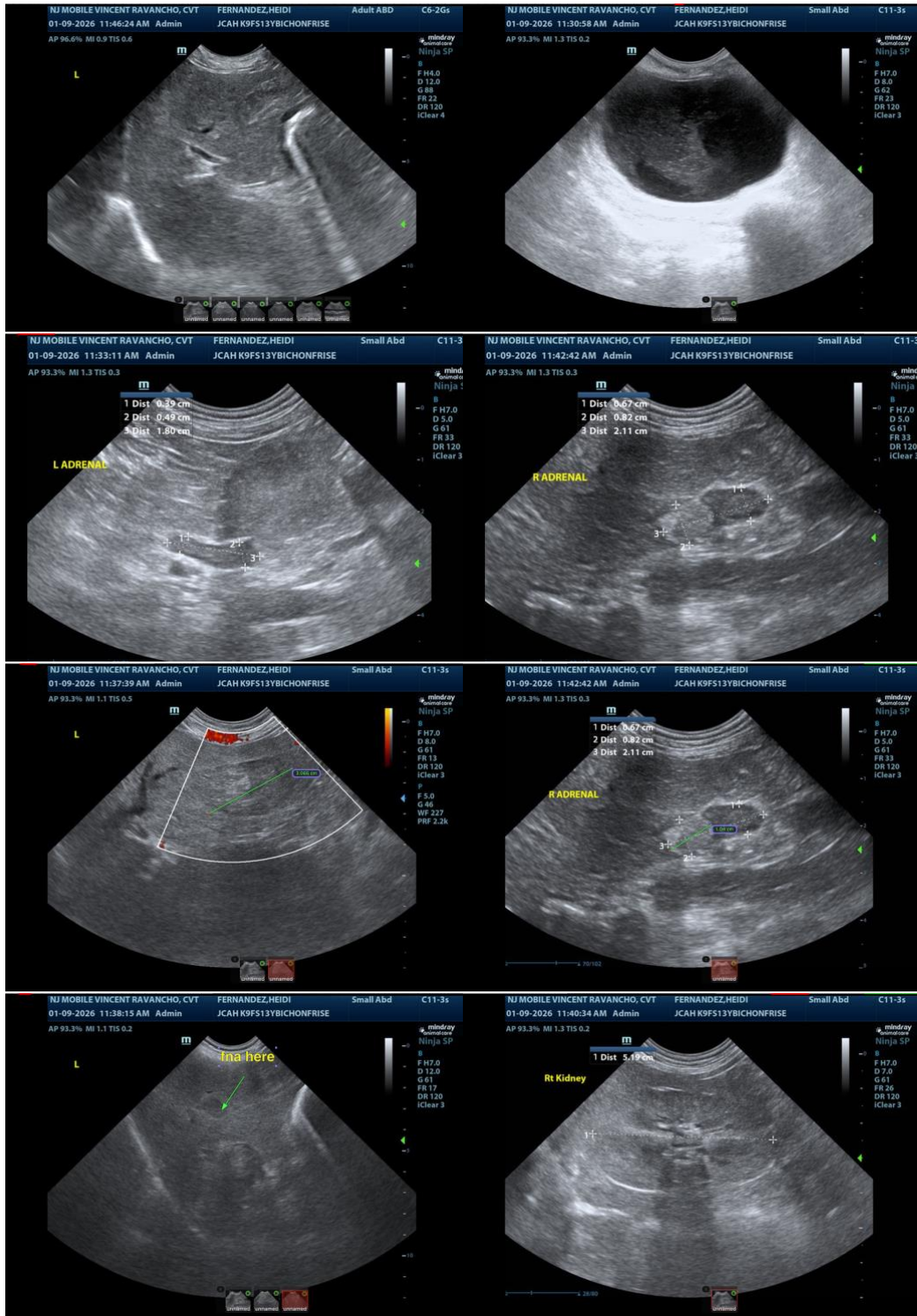
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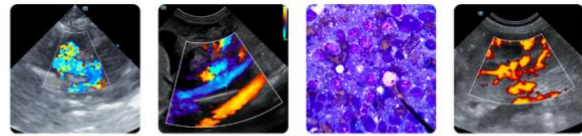
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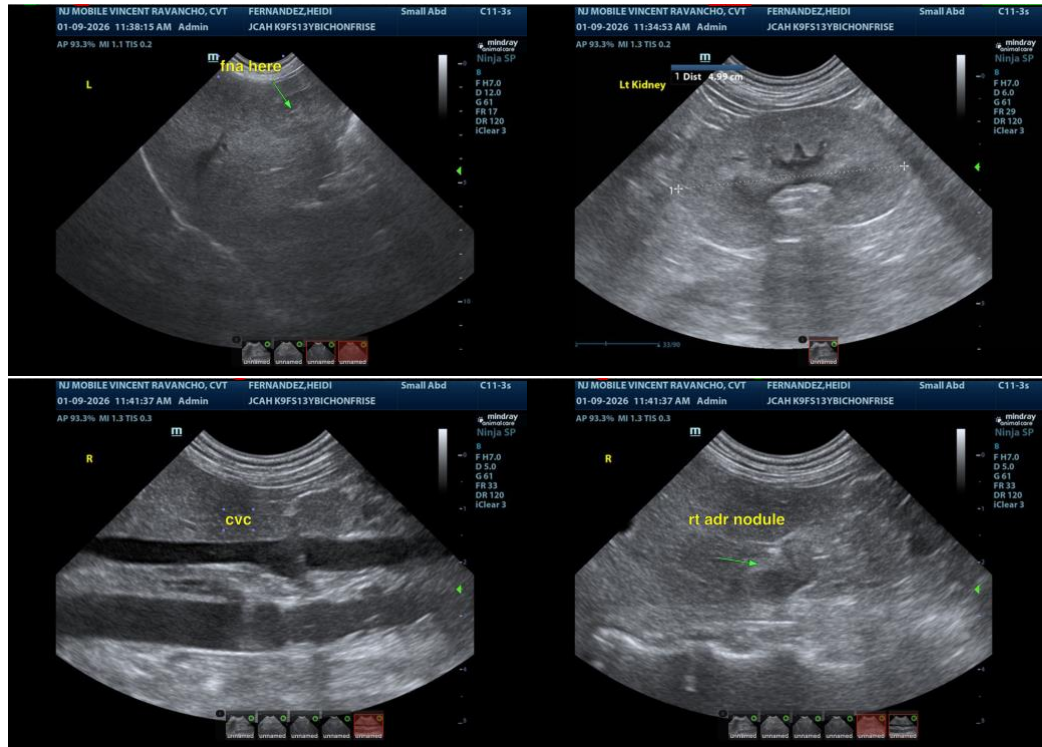
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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(CFM), Cert. IVUSS,  
CEO, Owner, Founder -- SonoPath.com  
[info@SonoPath.com](mailto:info@SonoPath.com)