



**PATIENT**

Tucker Percharsky

**SPECIES**

Canine

**BREED**

Mix

**SEX**

Neutered male

**AGE**

11 years

**WEIGHT**

32 lbs

**PRESENTING CLINICAL SIGNS**

History: PU/PD, elevated liver enzymes, elevated prot/creat ratio  
 Abnormal PE/Chem/CBC/UA Results: Reticulocyte 23.8, ALT 300, AST 62, ALP 528, GGT 19, Chol 475, Urine protein 3.1, 3+ Protein, SG: 1.011

**ULTRASONOGRAPHIC EXAMINATION OF THE HEART**

The echocardiogram in this patient demonstrated normal **left atrial** size based on 3 different LA measurement methods. Chamber volumes and echogenicity were normal. The cranial and caudal **mitral** valve leaflets presented vegetative thickening consistent with endocardiosis. Doppler indicated measurable insufficiency. The **left ventricle** presented thicknesses with linear contour and was not dilated nor restricted. The **myocardium** presented normal echogenicity without subjective evidence of significant fibrotic or ischemic disease. **Contractility** of the ventricular walls was adequate and in normal range for this patient evidenced by the fractional shortening measurement and subjective evaluation of the different regions of the myocardium. The **left ventricular outflow** tract demonstrated normal laminar flow and subjective structural integrity. The **right atrium** and auricle revealed normal size, structure and content. No evidence of masses was noted or chamber overload. **Tricuspid** valvular assessment demonstrated adequate linear morphology. The **right ventricle** was of normal size (1/3 diameter of LV), chordae structure, myocardial echogenicity and thickness. **Pulmonic** tract assessment revealed normal valve structure, laminar flow, and diameter (approx. 1:1 pa/ao ratio). No visible **pericardial** or free pleura fluid was noted. No echographically detectable evidence of infiltrative disease was visible. The cranial **mediastinum and pericardial regions** were free of masses in the visible window.

**INTERPRETED BY**

Eric Lindquist, DMV  
 DABVP, Cert. IVUSS

**IMAGING PERFORMED BY**

Valeryia Shumskaya

**HOSPITAL NAME**

Marsh AH

**REFERRING VET**

Dr. Milwicki

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CANINE CARDIAC PARAMETERS	MR VMAX (m/s)	TR VMAX (m/s)	LA/AO (Boon method)	LA/AO (Heart Base)	FS (%)	EF (%)	EPSS (cm)
NORMAL PARAMETER	4.5-5.5	<2.7	1.3	<1.6	28-40	40-100	<0.6
PATIENT	6.0		1.1	1.7	31	61	0.1
CANINE CARDIAC PARAMETERS	HR (BPM)	AV VMAX (m/s)	PV MAX (m/s)	BODY WEIGHT	LA (2D short axis Base view) (cm)	LVIDd (Avg; 2D and m-mode short axis) (cm)	LVIDs (Avg; 2D and m-mode short axis) (cm)
NORMAL PARAMETER	50-100	0.7-1.7	0.7-1.6	BELOW	BELOW	BELOW	BELOW
PATIENT	85	2.0	1.2	32 lbs	3.0 max	2.84	



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**ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN**

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**Urinary System**

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The **bladder** in this patient was mildly thickened with slight echogenic mural changes. No calculi or masses were noted. Slight micropolypoid changes were noted. This is a frequent finding in older animals and may be linked to a history of chronic urinary tract infection or active urinary tract infection. Urinalysis would be recommended with culture if any evidence of inflammatory sediment is present. The region of the trigone and visible pelvic urethra were normal.

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The residual prostate measured 0.8 cm.

**SEX**

Neutered male

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 and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The left kidney measured 6.5 cm. Solid blood flow to the kidneys was noted on power Doppler assessment.

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**Adrenal Glands**

**WEIGHT**

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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. The right adrenal gland measured 1.88 x 0.95 cm at the cranial pole and 0.74 cm at the caudal pole. The left adrenal gland was at the upper limits of normal and measured 1.96 x 0.41 cm at the cranial pole and 0.67 cm at the caudal pole.

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**Spleen**

**IMAGING PERFORMED BY**

Valeryia Shumskaya

The **spleen** was normal size and relatively normal contour with multifocal hyperechoic areas of mineralization. This is a benign change; however, can be related to Cushing's disease or other endocrinopathies.

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**Liver**

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The **liver** was uniformly swollen with minor, excessive gallbladder debris and over distension with dependent and suspended bile without evidence of overt mucocele formation. However, excessive sludge was present. The liver 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.

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**Gastrointestinal**

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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.



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**Pancreas**

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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.

**BREED**

Mix

**ULTRASONOGRAPHIC FINDINGS**

Stage B1 valvular disease, compensated.

Bilateral adrenal hypertrophy.

**SEX**

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Splenic mineralization.

Moderate degenerative renal changes.

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**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**

The heart is stable without clinical disease. No overt contraindication for anesthesia of brief to moderate duration. I suggest Torbutrol premed, Propofol induction, Isoflo maintenance or similar protocol if anesthesia is desired. Blood pressure recommended if not already performed and target white coat negative systolic pressure of < 160 mmHg. If higher than this ACE-inhibitor is suggested to reach this level. Recheck echocardiogram is recommended in 6 months, earlier if murmur grade increases or clinical signs initiate.

**WEIGHT**

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I am concerned for pituitary dependent hyperadrenocorticism. If the patient appears Cushingoid and the urine specific gravity is persistently less than 1.020 then work-up for PDH is indicated. Concurrent protein losing nephropathy, benign hepatopathy is likely. FNA of the liver can be considered for further definition.

**IMAGING PERFORMED BY**

Valeryia Shumskaya

**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. 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.*

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**Screen first, workup second**



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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.*

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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.

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

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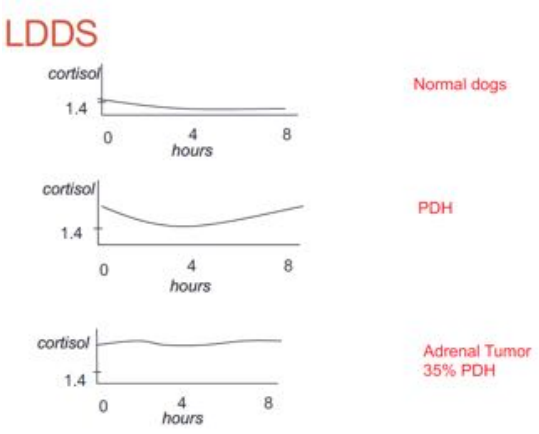
3) **LDDST** (0.01 D-Sodium phosphate mg/kg IV **with precise dosing\*\*\*\***) (Better screening test but plagued with false + but considered more specific than ACTH stim) Use if there is potential early Cushing's or if adrenal asymmetry present on sonogram suspecting tumor. Use LDDST in cats at a higher 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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Courtesy: Rebecca Berg DACVIM, DECVIM

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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.

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



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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.

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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.

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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.

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Neutered male

Suggested reading:

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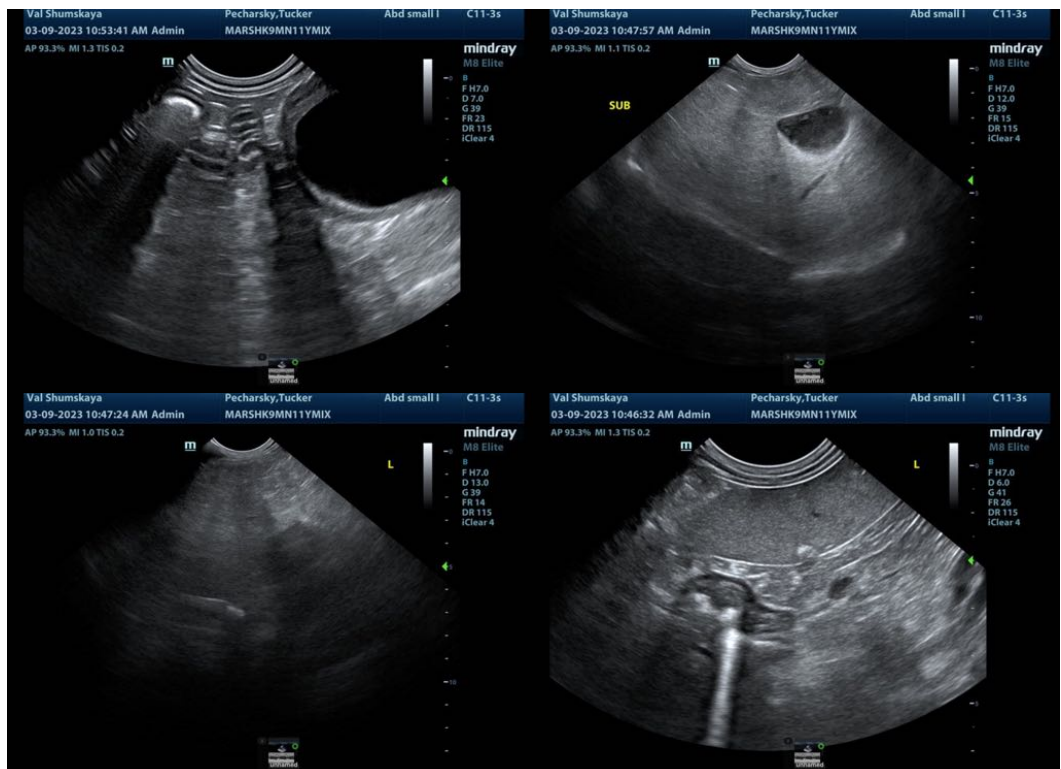
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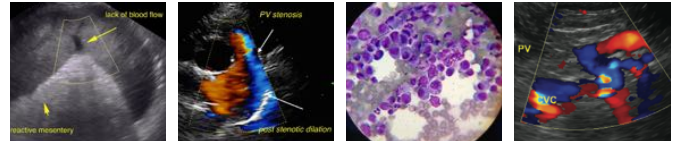
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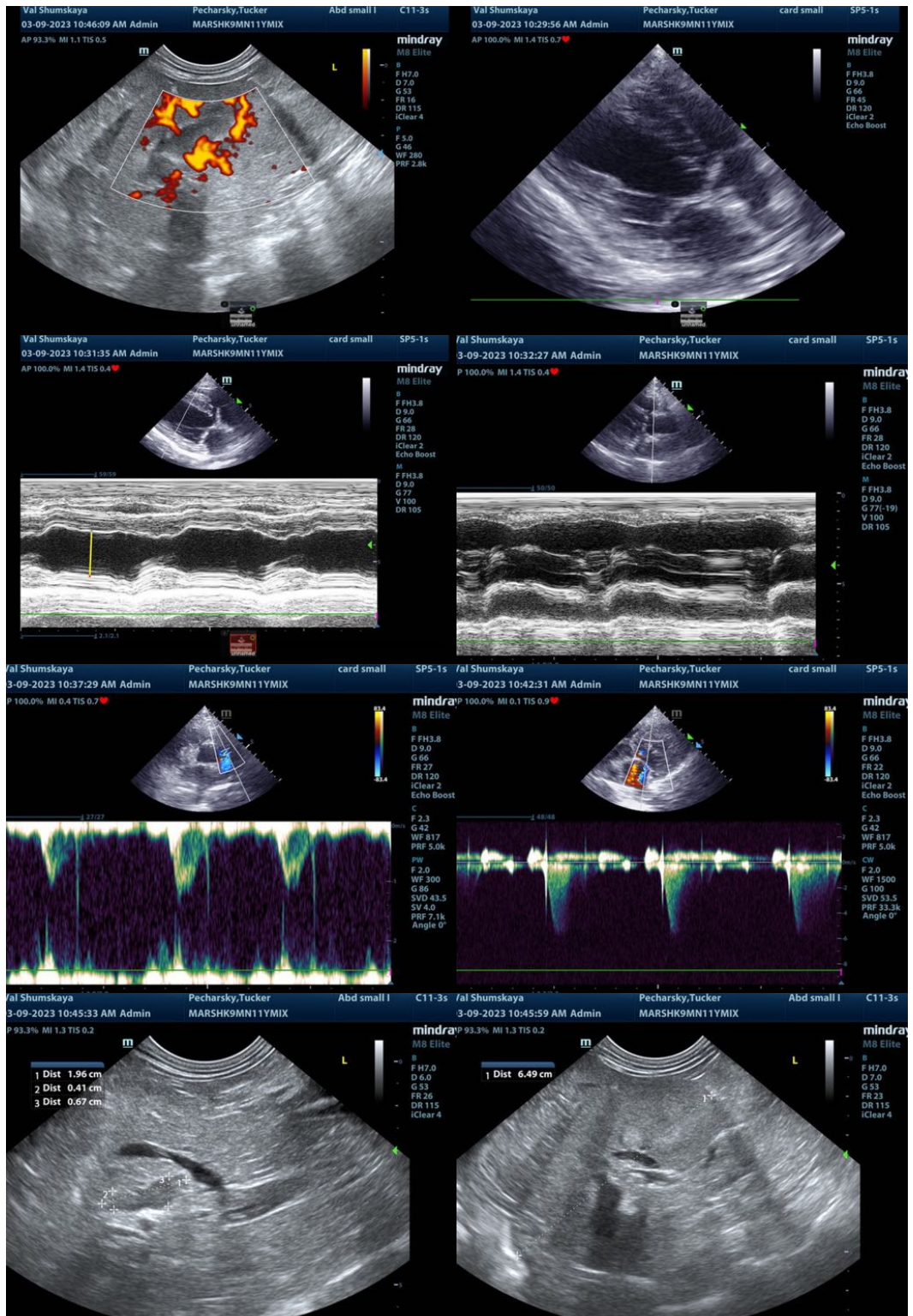
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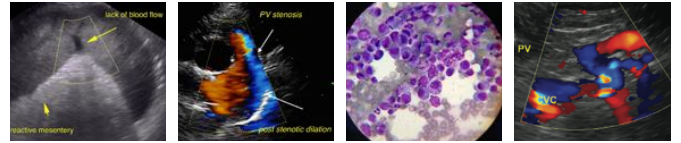
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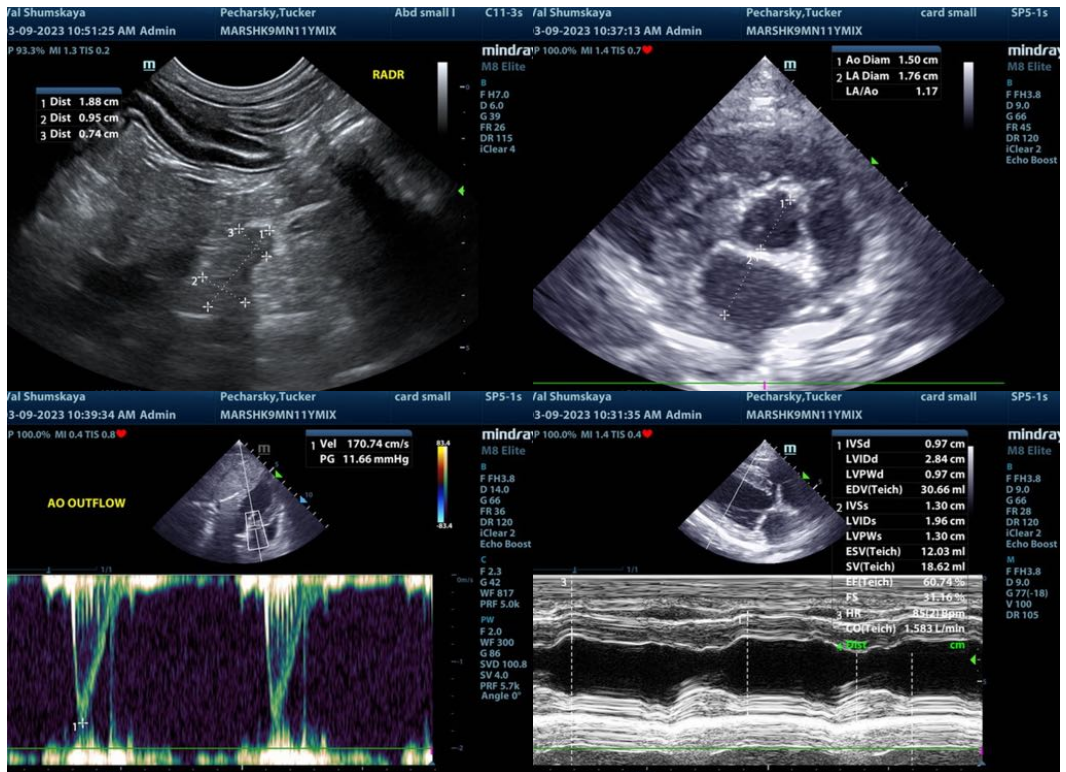
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

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