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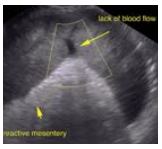

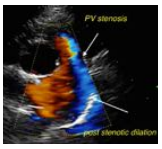

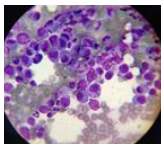
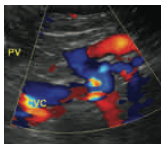

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PATIENT

Bayley Bruckner

SPECIES

Canine

BREED

Puggle Mix

SEX

Neutered male

AGE

9 years

WEIGHT

6.28 kg

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Roppolo

HOSPITAL NAME

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PRESENTING CLINICAL SIGNS

Patient initially presented in Feb 2022 with Hyphema of right eye. Blood pressure at that time was ~230 mmHg. Referred to PennVet for ER Optho consultation. Systemic hypertension confirmed at Penn. Client returned to Pennsauken AH for full hypertension workup of BW, UA, Radiographs.
Abnormal PE/Chem/CBC/UA Results: CBC/Chem WNL TXR revealed NSF UA WNL NIBP: consistently ~200 mmHg

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. The pelvic urethra was imaged 3.0 cm beyond the cystourethral junction. 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 was noted. Ureteral papillae were normal.

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 4.0 cm. The right kidney measures 4.0 cm.

Adrenal Glands

The left **adrenal gland** was uniform and measured 0.4 cm. The right adrenal gland was mildly heterogenous and measured 0.84 cm at the cranial pole and 0.61 cm at the caudal pole. There was no evidence of capsular expansion, capsular escape or vascular invasion. The vena cava was free of evident pathology. The cranial pole was heterogenous. The phrenic vein was not occupied.

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 was noted.

Liver

The **liver** images submitted revealed subjectively normal liver size, contour, and structure. Parenchymal echogenicity was naturally coarse and hypoechoic to the spleen. Vascular and biliary tracts were of normal volume with no evidence of congestion. The gallbladder presented acceptably thin walls with primarily anechoic content. The cystic and common bile ducts were normal. No pathological hepatic lymphadenopathy was evident. No overt structural evidence of inflammatory, infiltrative or regenerative pathology was evident.

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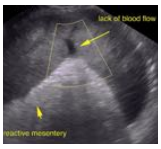
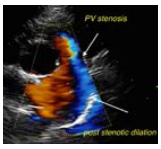
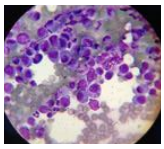
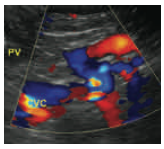

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Gastrointestinal

Shadowing material was noted in the pyloric outflow. This may represent ingesta or medications or small foreign matter and measures up to 2.0 cm. Chyme was noted elsewhere in the stomach.

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

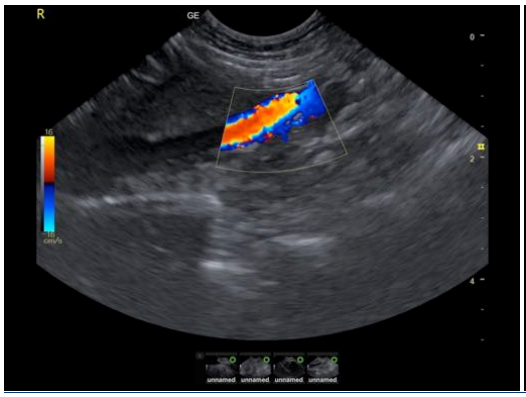
Mildly heterogenous right adrenal gland, yet no obvious masses.

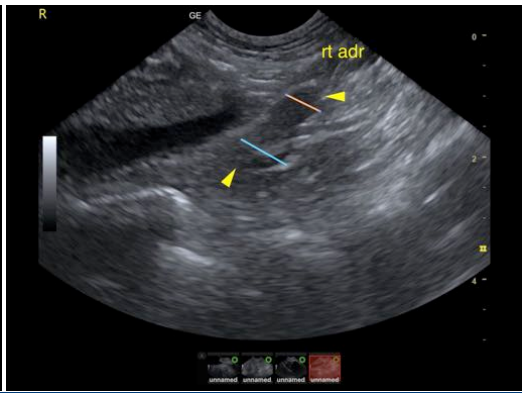
Otherwise, unremarkable abdomen.

Minor 2.0 cm shadowing gastric material. This may be kibble, medications or possible foreign matter.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The cause of hypertension is not evident in the abdomen.





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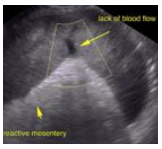
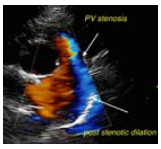
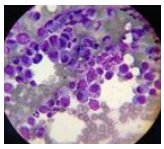
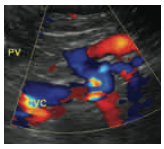

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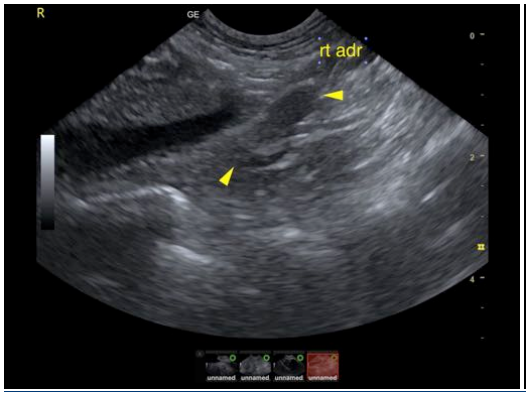

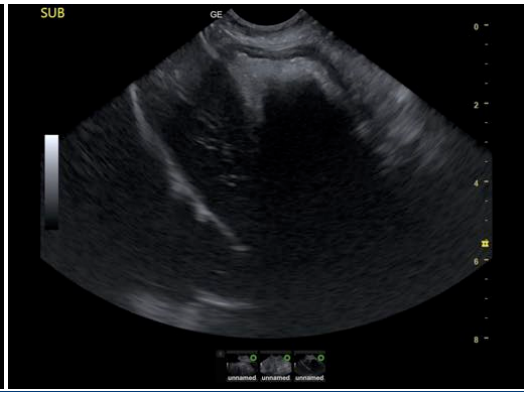

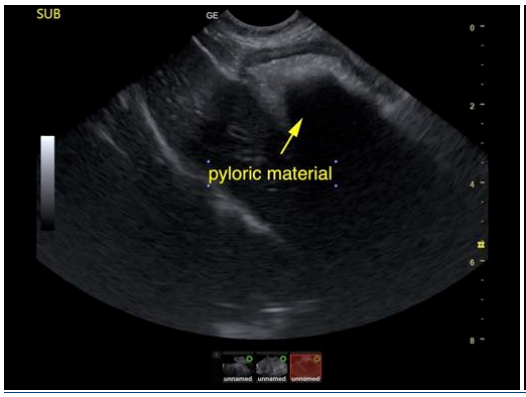

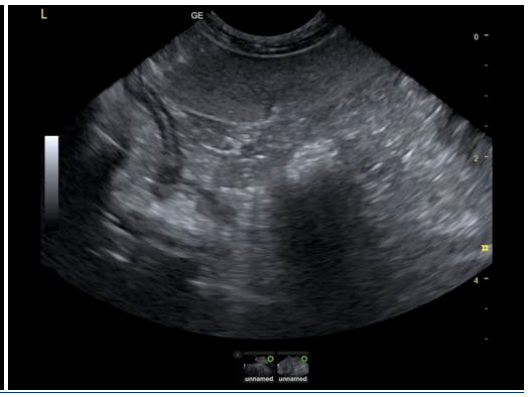

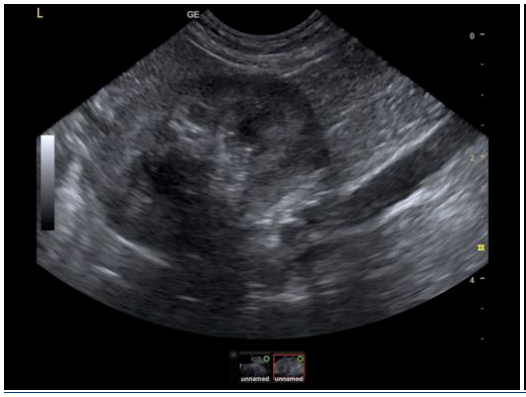

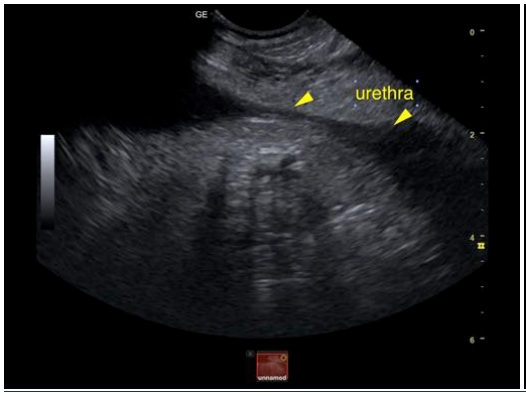

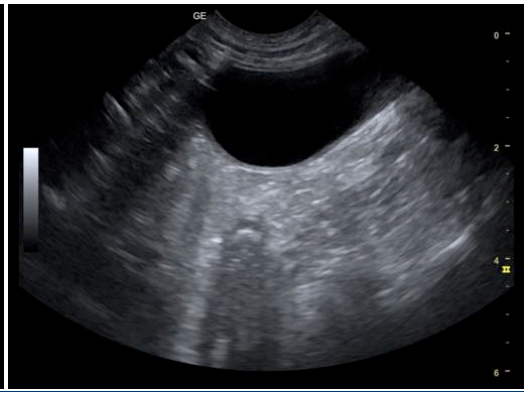

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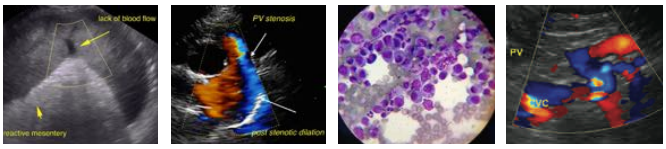

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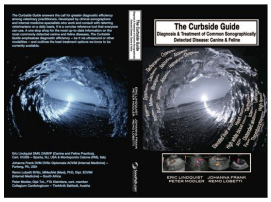
The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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



The following is an applicable excerpt from the *Curbside Guide to Diagnosis & Treatment of Sonographic Disease* offered by SonoPath.com Lindquist, Frank, and Modler.

An essential quick guide for every general practitioner and sonographer.

https://sonopath.com/products/curbside-guide-editing-due-release-12012015

Systemic Hypertension

http://www.sonopath.com/Hypertension

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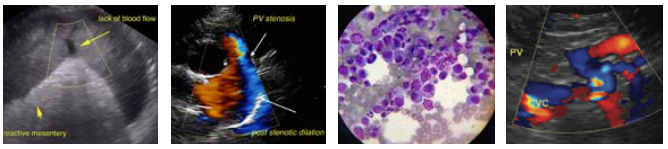

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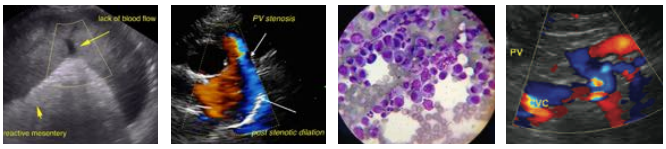
Long axis of the left kidney in a hypertensive dog with nephritis and protein losing nephropathy. The Power Doppler shows a high perfusion intensity within the interlobar and arcuate vessels. Also note the abnormal presence of excessive renal cortical perfusion. Acute PLN cases often have unremarkable structural renal chang sonographically.

Description: Systemic hypertension refers to the persistent elevation of systemic blood pressure (BP) and is classified as either primary (essential or idiopathic) or secondary. Persistent damage caused by hypertension is typically noted in effector organs such as the eyes, central nervous system (CNS), heart, and kidneys. In cats, the most common diseases associated with systemic hypertension are chronic kidney disease and hyperthyroidism, whereas the same condition in dogs more typically results in chronic kidney disease (especially proteinuric renal disease), hyperadrenocorticism, diabetes mellitus, and pheochromocytoma. Less typical causes of systemic hypertension in both species include hyperaldosteronism, acromegaly, and the use of hypertensive medications (e.g. phenylpropanolamine and excessive thyroxine supplementation).

Clinical Signs: Although hypertension is often clinically silent, ophthalmologic changes are usually the most obvious signs. Ophthalmologic changes secondary to hypertension are better described in cats than in dogs, and include intraocular hemorrhage, hypertensive retinopathy, hypertensive choroidopathy, and hypertensive optic neuropathy. CNS signs can include seizures, vascular accidents, and changes in mentation. Congestive heart failure secondary to hypertensive cardiac changes is rare, but affected animals may exhibit increased sensitivity to fluid administration.

Diagnostics: Blood pressure values obtained during diagnostic evaluation must be assessed in conjunction with other clinical findings, such as retinal detachment as well as a history of polyuria and polydipsia, as these associated findings may point to the cause of the hypertension or indicate hypertension-associated organ damage. A single high blood pressure value should never be used to diagnose systemic hypertension in the absence of other clinical data, as patient distress or stress may artificially elevate blood pressure. Current recommendations suggest that a systolic blood pressure greater than 160 mm Hg is indicative of hypertension; however, the use of antihypertensive medications may not be warranted if blood pressure elevations are mild and underlying etiologies addressed. The minimum



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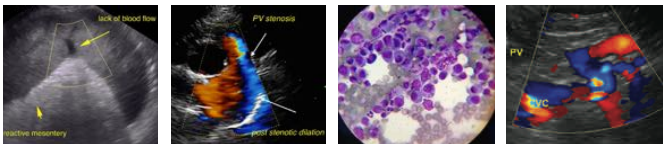
database should include physical examination, funduscopic examination, CBC, serum chemistry profile, urinalysis with urine protein quantification, and serum T4 assay in cats. Measuring the blood pressure in separate moments throughout the day in a quiet, non “white coat effect” environment, in a home environment, or potentially with light opioid sedation (Buprenorphine 0.01 mg/kg) can also be considered when looking for hypertension. Empirically, in our experience, this opioid approach tends to be an effective approach when assessing for hypertension in excited patients. However, a complete comparative study is warranted.

Treatment: First, the use of any hypertensive medications should be discontinued immediately. Drug groups that can be used to control hypertension include angiotensin-converting enzyme (ACE) inhibitors (enalapril or benazepril 0.5 mg/kg PO BID), calcium channel blockers (amlodipine 0.625 mg PO Q24hr for cats and 0.1 mg/kg PO Q24hr for dogs), and beta blockers (atenolol 6.25-12.5 mg PO BID for cats and 0.25-1 mg/kg PO BID for dogs). Medications from different groups may be additionally administered if the original medication is not sufficiently controlling the condition.

Long axis of the right adrenalglan and caudal vena cava in a dog witl pheochromocytoma and hypertension. The enlarged heterogenous adrenal gland is seer on the image right (large arrow). There is tumoral invasion (small arrow) into the caudal vena cava v the phrenicoabdominal vein. The echogenic tumor material occupie the caval lumen entirely. Note the significant centrifugal mass effect the intravascular tumor on the ves wall as indicated by focal widenin of the caval diameter. Part of the right liver lobes and descending duodenum are seen on the image l and in the near field.



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Long axis of a left-sided adrenal mass a imaged using a right lateral sonographic approach ina hypertensive dog. The enlarged heterogenous left-sided adrena tumor is seen deep to the descending duodenum and caudal vena cava. Note t presence of multifocal mineralizations (MIN) within the tumor and the left phrenicoabdominal artery passing atop the mass lesion.

References:

Brown S, et al. Guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats. *J Vet Intern Med* 2007;21:542-58.

Michell AR, Bodey AR, Caulfield M. Evolution, essential hypertension and the high arterial pressures in certain athletic breeds of dogs. *Vet J* 2011; 88:125-27.

Jepson RE, Elliott J, Brodbelt D, Syme HM. Effect of control of systolic blood pressure on survival in cats with systemic hypertension. *J Vet Intern Med* 2007;21:402-09.

Steele JL, Henik RA, Stepien RL. Effects of angiotensin-converting enzyme inhibition on plasma aldosterone concentration, plasma renin activity and blood pressure in spontaneously hypertensive cats with chronic renal disease. *Vet Ther* 2002;3:157-66.