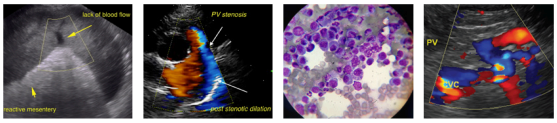

PATIENT

Max Lauder

SPECIES

Canine

BREED

Bulldog

SEX

Neutered Male

AGE

8/16/17

WEIGHT

70 lbs

INTERPRETED BY

Andrea Nicastro, DVM, Diplomate ACVIM (*Small Animal Internal Medicine*)

IMAGING PERFORMED BY

Andrea Nicastro, DVM, Diplomate ACVIM (*Small Animal Internal Medicine*)

HOSPITAL NAME

Flowertown AH

REFERRING VET

Dr. Matthew Nathan

INVOICE

36781

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PRESENTING CLINICAL SIGNS

Polydipsic, lethargic, hypoalbuminemia. Chronic skin disease. Decreased appetite.

TP: 4.8, Alb: 1.5, recheck today 2.1, Mag 1.3, Chol: 607, Amy: 1202, Plt: 639, Crea 1.6 (high normal), BUN normal.
UA: 2+ protein, Blood Trace, USG 1.036.

Bravecto q 90 days Heartgard q 30 d Cyclosporine (unknown mg) q24h eye drops (unknown brand) OU q24h Ketoconazole (unknown mg) EOD Weekly allergy shots

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder is mildly to moderately distended. The wall in the region of the apex is slightly thickened up to 0.65 cm with a slightly irregular mucosal surface. The wall tapers to a normal thickness as it extends towards the urinary bladder neck. Luminal contents are mostly anechoic. No cystic calculi are observed. The region of the trigone and the proximal urethra, visible to a depth of 1.0 cm, are normal.

The prostate is normal in size (1.72 cm in width) and shape. Parenchyma is homogenous. The prostatic urethra appears normal without evidence of dilation or obstruction.

The left kidney presented normal size (7.62 cm in length); normal shape and architecture with smooth peripheral margins. The cortex is diffusely thickened and hyperechoic and there is mild to moderate loss of corticomedullary distinction. An ill-defined, hyperechoic medullary band is observed adjacent to the corticomedullary junction. Mild pyelectasia is present at 0.39 cm in the transverse plane. There is no evidence of nephroliths, infarcts or hydroureter. Renal vasculature is normal.

The right kidney presented normal size (7.65 cm in length); normal shape and architecture with smooth peripheral margins. The cortex is diffusely thickened and hyperechoic and there is mild to moderate loss of corticomedullary distinction. An ill-defined, hyperechoic medullary band is observed adjacent to the corticomedullary junction. There is no evidence of pyelectasia, nephroliths, infarcts or hydroureter. Renal vasculature is normal.

Adrenal Glands

The left adrenal gland is normal size (0.46 cm at cranial pole) (0.62 cm at caudal pole) (2.69 cm in length); normal shape; homogenous parenchyma. The glandular echogenicity and detail are unremarkable. Capsule, cortex, and medullary definition are normal. The phrenicoabdominal vein and surrounding vasculature are normal.

The right adrenal gland is normal size (0.99 cm at cranial pole) (0.59 cm at caudal pole) (2.62 cm in length); normal shape; homogenous parenchyma. The glandular echogenicity and detail are unremarkable. Capsule, cortex, and medullary definition are normal. The phrenicoabdominal vein and surrounding vasculature are normal.

Spleen

The spleen is normal in size with a normal capsular contour. There is appropriate echogenicity and echotexture. A 1.22 cm, ill-defined, hypoechoic to slightly heterogeneous nodule is observed at the medial aspect near the hilus. Splenic vasculature is normal.

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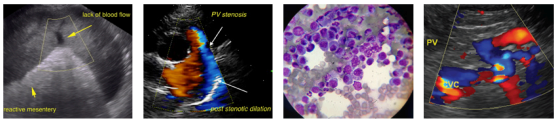

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Liver

The liver is subjectively normal in size with normal contours and structure. There is appropriate echogenicity and echotexture. No overt structural evidence of inflammatory, infiltrative or regenerative pathology is evident. Vascular and biliary tracts are of normal volume with no evidence of congestion. No pathological hepatic lymphadenopathy observed. The portal vein/caudal vena cava ratio is approximately 1:1.

The gall bladder lumen is moderately distended. The wall is thin and smooth. Luminal contents are anechoic. The cystic and common bile ducts are normal.

Gastrointestinal

The stomach and intestine are free of stasis and exhibit normal peristaltic activity. The gastric lumen is not distended. The gastric wall and pylorus are normal in thickness with a normal layering pattern. The small intestinal lumen is not dilated. The small intestinal wall thickness is normal with a normal layering pattern and appropriate mural detail. Discreet masses are not identified. The wall of the descending colon is mildly to moderately thickened up to 0.68 cm with retention of the normal layering pattern. There is no evidence of an obstructive pattern.

Pancreas

The region of the pancreas is isoechoic relative to surrounding omental fat. No obvious parenchymal abnormalities are observed. There is no evidence of regional inflammation or effusion.

Free Abdomen

The peritoneal cavity is normal. There is no evidence of inflammation or effusion. The abdominal lymph nodes are normal/not visible.

Other

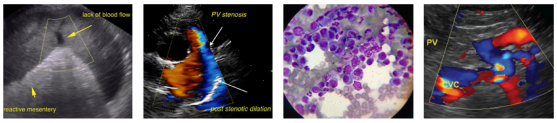
Brief echocardiogram reveals no obvious evidence of pericardial effusion.

PRIMARY FINDINGS

- Bilateral chronic nephropathy. This finding in conjunction with the patient's lab work abnormalities is concerning for a protein-losing nephropathy. Most cases of protein-losing nephropathy are idiopathic. However, they can be secondary to infectious, inflammatory, or neoplastic disease.

SECONDARY FINDINGS

- Mild urinary bladder wall thickening in the region of the apex. This may be artifactual due to lack of full repletion, or may represent low-grade cystitis. Correlation with clinical findings is recommended.
- The ill-defined splenic nodule could be consistent with a focus of lymphoid hyperplasia or extramedullary hematopoiesis. Alternatively, an emerging neoplastic process is possible.
- The mild colonic wall thickening may be secondary to inflammation, edema, or less likely emerging neoplasia.



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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Given the renal changes, the following diagnostics can be considered:

1. UPC
2. Urine culture and sensitivity
3. Baseline blood pressure measurement
4. Infectious disease testing (i.e., tick borne, Leptospirosis, heartworm).
5. 3-view thoracic radiographs to assess for occult neoplasia in the chest.

To assess for concurrent causes of hypoalbuminemia, consider the following:

1) Pre- and post-prandial serum bile acids.
2) Resting cortisol level.

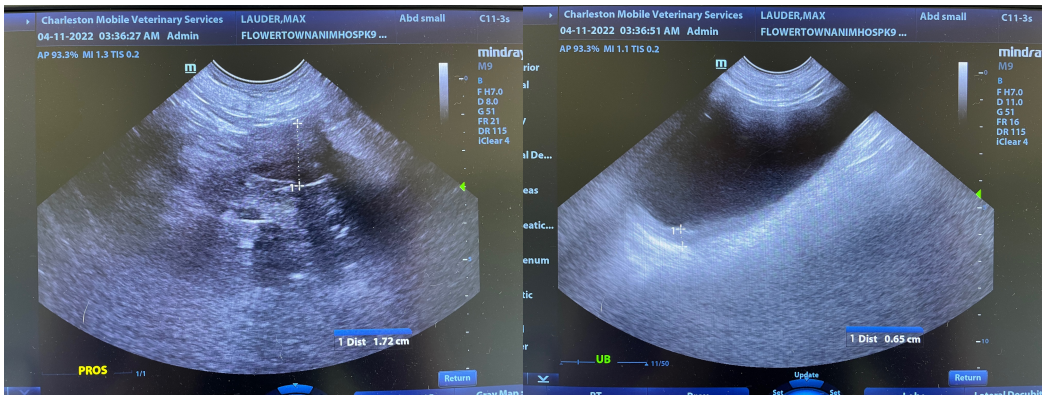
Depending on the above diagnostics, further treatment/follow-up for protein-losing nephropathy (PLN) may be warranted (see below).

PLN protocol:

1. Angiotensin II receptor blocker (e.g., telmisartan)
2. Antithrombotic (e.g., clopidogrel at 2.5 mg/kg PO q 24 hours)
3. Omega-3 fatty acids (65 mg/kg of DHA and EPA combined daily)
4. Prescription renal diet
5. Baseline blood pressure measurement with serial monitoring thereafter
6. Routine monitoring of UPC and bloodwork (CBC, chemistry panel) to assess for progressive disease

If the hypoalbuminemia appears to be of GI origin (if hepatic and renal origins are ruled out), gastrointestinal biopsies would be warranted.

Regarding the splenic nodule, consider a repeat ultrasound in 3-4 weeks to assess for progression. Alternatively, an ultrasound guided fine needle aspirate can be considered if clotting status is appropriate.



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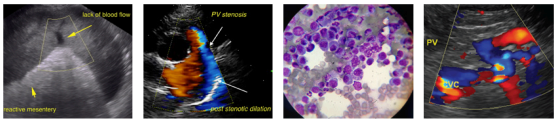

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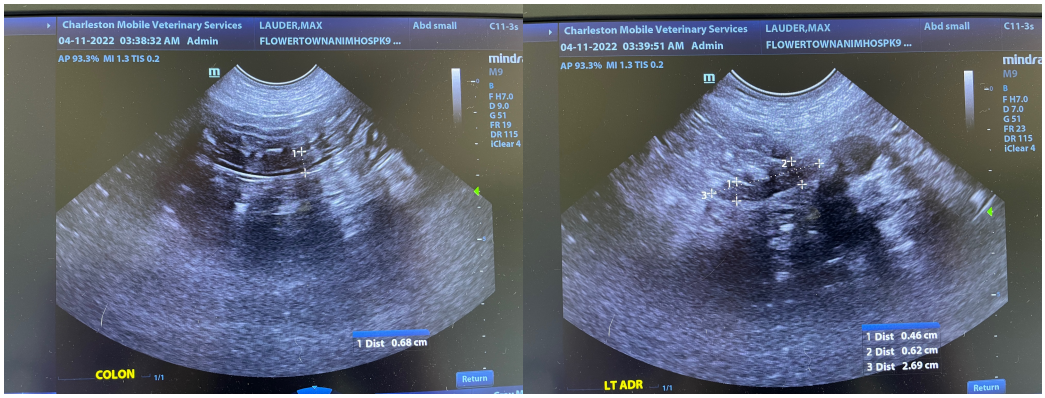

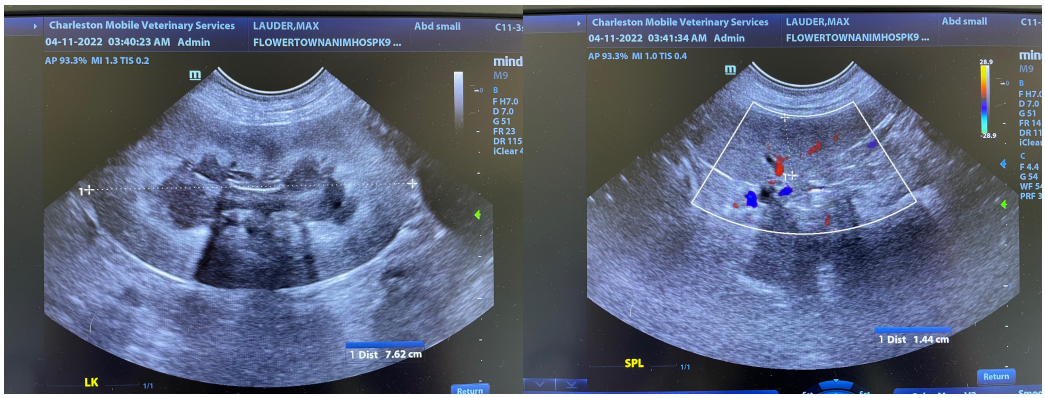

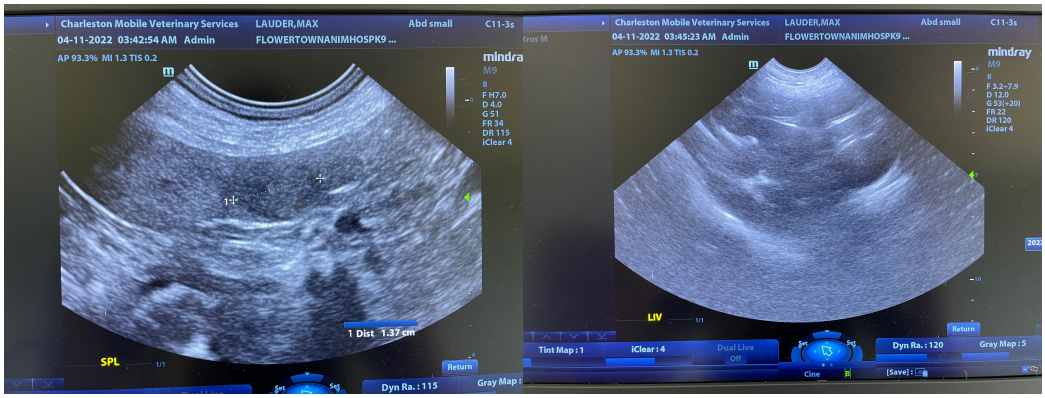

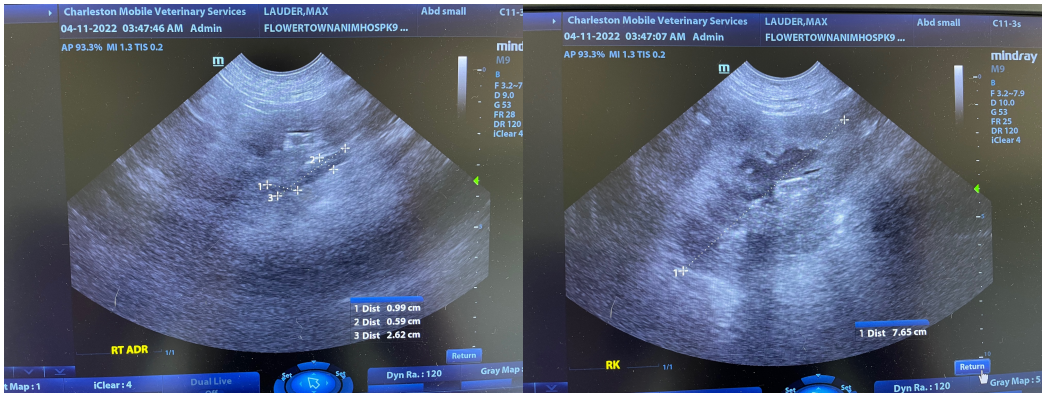

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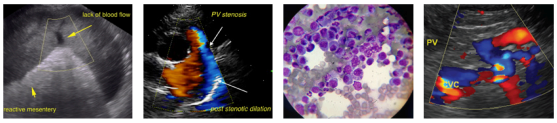

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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Andrea Nicastro, DVM, Diplomate DACVIM (Small Animal Internal Medicine)
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