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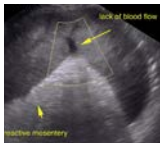
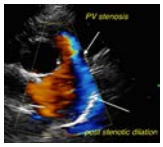
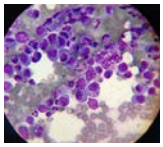
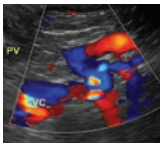

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1-800-838-4268 info@sonopath.com

PATIENT
Stanley Arasa

SPECIES
Canine

BREED
Chihuahua X

SEX
Neutered Male

AGE
11 Years

WEIGHT
11.4

INTERPRETED BY
Beth Johnson, DVM DACVIM

IMAGING PERFORMED BY
Dr. A.

HOSPITAL NAME
Surfside Pet Hospital

REFERRING VET
Dr. Americo Abadia

INVOICE
44717

DATE
8/16/23

PRESENTING CLINICAL SIGNS

History of chronic liver enzyme elevation since 2018 when pet had toxic episode with Blue and green algae. Owner informs that pet is drinking more water and having accidents in the house more often.

Abnormal PE/Chem/CBC/UA Results: Bw perfomed on july showed ALT- 727, AST- 64, ALP- 3,974, GGT- 34. U/A- shows Specific gravity of 1.020 , no blood, no increase in wbc's. Protein 1+, ph- 7, ascorbic acid- +3, Microalbuminuria - >=2.5, protein/creatinine ratio- >0.5 to <2.5

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder is moderately distended with anechoic contents. No masses, inflammatory changes, echogenic sediment or cystoliths are observed. The urinary bladder, trigone and visible pelvic urethra are normal in thickness with a smooth mucosal surface.

Prostate is normal in size, echotexture and echogenicity for a neutered male.

Kidneys are overall normal in size and shape with smooth peripheral margination. A normal 1:3 cortex to medulla ratio is maintained. The medulla and cortices are uniform in texture with some mild increased cortical echogenicity and mild loss of corticomedullary distinction, expected in this age patient. There is no evidence of pyelectasia, mineral or infarcts observed. The right kidney measures 4.77 cm. The left kidney measures 5.24 cm. Multiple small cortical cysts are noted bilaterally.

Adrenal Glands

The right adrenal gland is normal in size (0.51 cm at the cranial pole and 0.49 cm at the caudal pole), shape and contour. Corticomedullary structure is unremarkable. Visible surrounding vasculature appears normal.

The left adrenal gland is normal in size (0.55 cm at the cranial pole and 0.60 cm at the caudal pole), shape and contour. Corticomedullary structure is unremarkable. Visible surrounding vasculature appears normal.

Spleen

The spleen is subjectively normal in size with a normal smooth capsular contour. Parenchyma is appropriately finely textured and homogenous with normal echogenicity relative to surrounding tissue (hyperechoic to liver). A 0.20 cm x 0.40 cm hypo- to anechoic non-capsule disrupting nodule is noted in the mid body. Splenic vasculature appears normal.

Liver

Liver is subjectively enlarged with mildly irregular margins. Diffusely, the parenchyma is heterogenous characterized by multiple poorly defined hypoechoic nodules within otherwise hyperechoic liver parenchyma. Visible vasculature and biliary tree appear normal without distension or congestion. In the mid to caudal liver, there is a 2.4 cm x 4.2 cm homogeneous, hypoechoic, rounded liver lobe/emerging mass.

Gallbladder is moderately distended with anechoic bile as well as suspended and gravity dependent echogenic debris. The wall is smooth without visible thickening. There is no evidence of cystic or CBD dilation. There is no evidence of effusion or inflammation.

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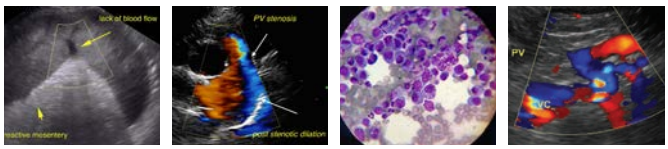

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Gastrointestinal

The stomach wall is normal in thickness (canine < 0.5 cm and feline < 0.4 cm) and layering. The lumen of the stomach is empty with no evidence of obstruction, foreign material or infiltrative disease. Pyloric outflow tract appears patent.

The visible small intestines are normal in wall thickness and layering (canine duodenum < 0.5 cm and feline duodenum < 0.4 cm; other < 0.3 cm). Small intestinal motility appears adequate (1-3 contractions per min). The lumen of the small intestine is empty with no evidence of obstruction, foreign material or infiltrative disease.

The visible colon is normal in wall thickness (< 0.2 cm) and layering. Contents are consistent with normal formed feces and gas.

Pancreas

The pancreatic parenchyma is appropriately isoechoic to surrounding tissue. Visible capsule is smooth and normal in contour. There is no visible pancreatic duct dilation. There is no evidence of active peripancreatic inflammation.

Free Abdomen

There is no evidence of free peritoneal effusion noted in these images.

There is no apparent lymphadenopathy noted in these images.

PRIMARY FINDINGS

- **Diffusely heterogenous Liver with a focal rounded liver lobe/mass –** These changes are most consistent with benign processes such as nodular hyperplasia, steroid (vacuolar) hepatopathy, extramedullary hematopoiesis or possibly chronic inflammatory disease and less commonly infiltrative round cell or metastatic neoplasia. The focal rounded liver lobe/mass likely represents a benign adenoma/hepatoma. However, infiltrative neoplasia (i.e., hepatocellular carcinoma, round cell neoplasia, other can't be ruled out without tissue sampling).

- **Moderate gallbladder debris -** Cholecystic debris is of unknown clinical significance. It can be seen with biliary stasis from fasting or illness. Cholecystic debris is not necessarily related to hepatobiliary disease. Echogenic bile is most commonly an incidental finding in dogs and should be interpreted in combination with clinical signs such as nausea, inappetence, cranial abdominal discomfort and/or laboratory changes such as increased ALP and/or increased Tbili.

SECONDARY FINDINGS

- **Hypo to anechoic splenic nodule** – likely represents a benign lesion such as a cyst, hematoma, nodular hyperplasia, extramedullary hematopoiesis, etc., however while considered less likely, infiltrative neoplasia can mimic benign lesions, and cannot be ruled out.

- Age related kidney changes

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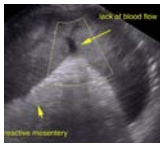
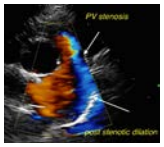
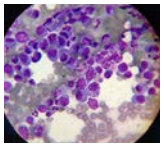
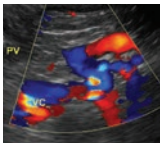

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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Differentials for PU/PD are vast and include, but are not limited to:

Primary polyuria caused by chronic kidney disease, pyelonephritis, liver disease, diabetes mellitus, hyperthyroidism, hypercalcemia, hyperadrenocorticism, hypoadrenocorticism, E.coli infections ie) pyometra in females, polycythemia, central diabetes insipidus or primary nephrogenic diabetes insipidus.

Primary polydipsia caused by psychogenic polydipsia, fever, pain, or central nervous system disease.

Most causes of PU/PD can be diagnosed with a comprehensive history and physical exam, a first AM urine specific gravity to see if urine concentration is possible (as most animals naturally consume less water overnight) followed by a comprehensive CBC, serum chemistry panel, electrolytes, and urinalysis.

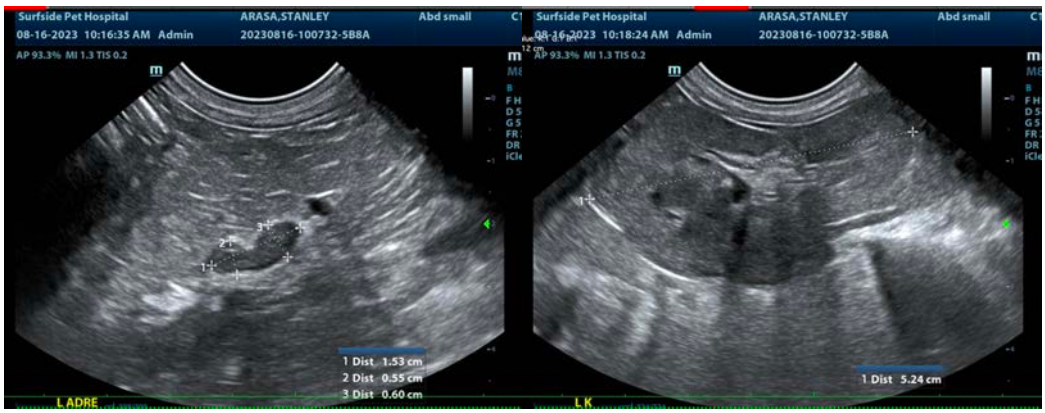
If not, next step(s) may include a urine culture, low dose dexamethasone suppression test, T4, bile acids, Leptospirosis testing and/or an empirical course of antibiotics.

If a diagnosis is still not obtained, a more advanced work-up is indicated and consultation with an internist may be warranted.

Specifically for this patient, further liver workup in part depends on if there has been an acute exacerbation in liver enzyme versus these being this patient's normal since the previous toxic insult.

Pending that information, bile acid testing could be considered if patient's total bilirubin is normal. Additionally, testing for Leptospirosis is recommended, and as is reportedly already pending, a fine needle aspirate of the rounded liver lobe/liver mass, if patient's coagulation status is appropriate.

In the meantime, if patient's UPC warrants therapy, medical management for protein losing nephropathy could be considered, as could hepatic nutraceuticals, including Ursodiol, given the moderate amount of gallbladder debris.



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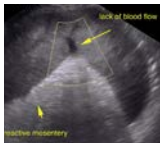
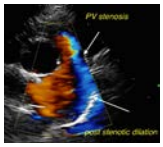
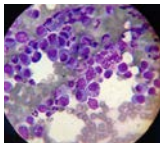
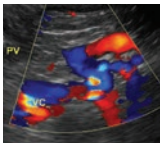

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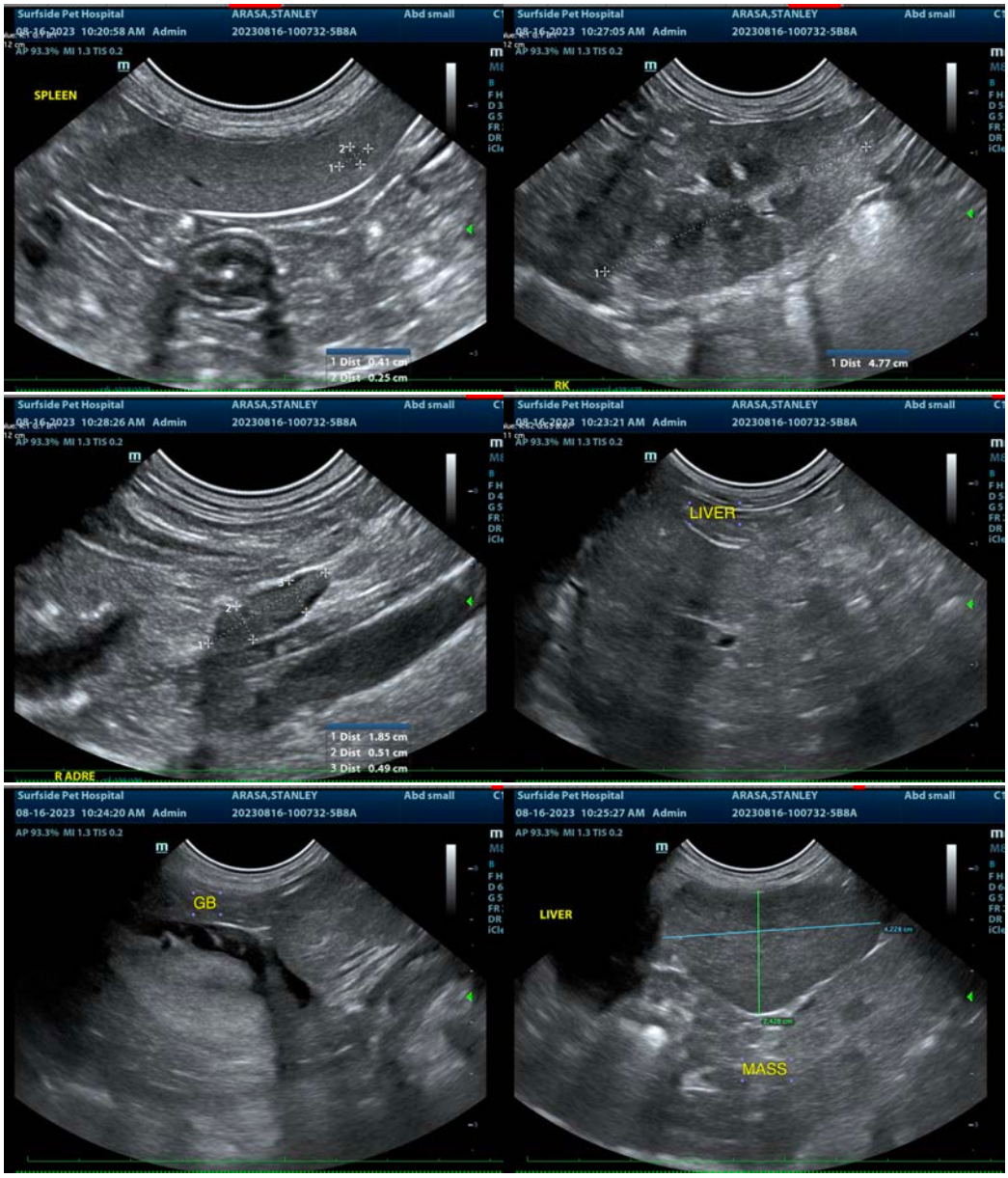

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Beth Johnson, DVM, DACVIM
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