PATIENT

Kizzie Lockhart

SPECIES

Canine

BREED

Schnauzer

SEX

Spayed Female

AGE

8 years

WEIGHT

15 lbs.

INTERPRETED BY

R. McKenzie Daniel, DVM, DABVP (Canine and Feline)

IMAGING PERFORMED BY

Sarah Pender, CVT

HOSPITAL NAME

SVS Imaging QC

REFERRING VET

Dr. Doerscher

INVOICE

12358

DATE

10/13/21

PRESENTING CLINICAL SIGNS

New L-sided Grade III heart murmur DX 4/2021. HX of urolithiasis, mild CRD and elevated LE..
Presented 10/6/21 for "NFW" - some V and inappetence as well as "episodes" of seemingly staring off into space/being unaware. BW on 10/6 showed worsening CRD & LE. BP showed hypertension, UP/C elevated. Started phos binder (320 mg daily) & benazepril (2.5 mg BID) & Cerenia and P improved some this week in how she's feeling in general.
Abnormal PE/Chem/CBC/UA Results: SDMA 19 on 10/6 Creat 2.7 on 10/6 and 3.4 today BUN 57 on 10/6 and 80 today Phos 15.1 Ca 12.9 ALT 139 on 10/6 and 200 today ALP 1948 on 10/6 and 1748 today cPL was abnormal on 10/6 UP/C 7.4 BP average was 174-176

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder, trigone, cystourethral junction, and visible pelvic urethra to a depth of 3.0 cm exhibited normal thickness and tone. Anechoic urine was present in the lumen with no uroliths or sediment. The ureteral papillae were normal. The ureters were not visible which is normal. No evidence of inflammatory or neoplastic changes was noted.

The area of the aortic trifurcation was free of pathology.

Both kidneys exhibited asymmetrical margination and mild nonuniform increased cortex echogenicity. Moderate loss of corticomedullary border demarcation was present with a mild bilateral medullary rim sign. Both kidneys exhibited mild pyelectasia without evidence of concurrent left or right ureter dilation. The left kidney measured 5.1 cm in length. The right kidney measured 5.4 cm in length.

Adrenal Glands

The left adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The left adrenal gland measured 2.0 cm length x 0.53 cm width at the caudal pole. The right adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The right adrenal gland measured 2.3 cm length x 0.48 cm width at the caudal pole.

Spleen

The spleen exhibited a finely textured and homogenous parenchyma which was hyperechoic to the liver and renal cortical parenchyma. The capsule was smooth and regular without apparent expansion. The splenic vasculature at the hilus was normal in volume with no evidence of congestion or thrombosis. Acute to chronic inflammatory, neoplastic, or benign parenchyma changes were not noted.

Liver/ Gallbladder

The liver presented enlarged in size. The parenchyma of the liver was subjectively normal in echogenicity compared to the spleen and renal cortices. The liver parenchyma was uniform with a mildly coarse echotexture. The capsule of the liver was symmetrically rounded to mildly swollen in margination. The hepatic and portal vasculature were normal in appearance without signs of congestion. The gallbladder was non-distended in size with moderate, nondependent, subjectively mobile, nonorganized, echogenic gallbladder debris. The cystic and common bile ducts were normal.

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Gastrointestinal

The stomach presented mild to moderate wall thickening owing to mild to moderate prominent gastric mucosa and rugal fold. Intact wall layering was maintained and distinct. The gastric body wall measured 0.43 cm width. Mild gastric distension was present. Minor retained anechoic fluid was present in the gastric lumen.

The small intestine presented intact wall layering with 1:3 muscularis/mucosa ratio. The lumen of the small intestine was empty with no signs of ileus, obstruction, or foreign material. The jejunum wall width measured 0.35 cm.

Normal visible colon wall layers were present with apparent formed feces in lumen.

Pancreas

The parenchyma of the left limb, body, and right limb of the pancreas presented isoechoic to the adjacent omental fat. A normal curvilinear capsule contour of the pancreas was present. The visible pancreatic duct was normal. No signs of active inflammation or neoplastic disease were evident.

Free Abdomen

No overt lymphadenopathy or peritoneal effusion was present.

ULTRASONOGRAPHIC FINDINGS

Primary Findings

- Bilateral chronic nephropathy with moderate loss corticomedullary border demarcation, nonspecific medullary rim sign, and mild pyelectasia
- Hepatopathy / subjectively benign
- Moderate gallbladder debris (non-mucocele)
- Mild to moderate gastritis, potential uremic gastritis

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The bilateral kidneys are consistent with chronic glomerulonephritis or other glomerulopathy, given the elevated UPC level. ACE inhibitor +/- Amlodipine and Plavix, given the possibility of secondary thromboembolic disease owing to renal protein loss, is recommended. Continued monitoring of systemic blood pressure is indicated.

Gastroprotectant protocol is suggested.

The overall liver is suggestive of benign hepatopathy with considerations Including vacuolar/reactive hepatopathy or Inflammatory hepatic or hepatobiliary process, given the ALT elevation and presence of gallbladder debris. Hepatosupportive medications including Ursodiol and monitoring for evidence of increasing cholestasis are suggested.

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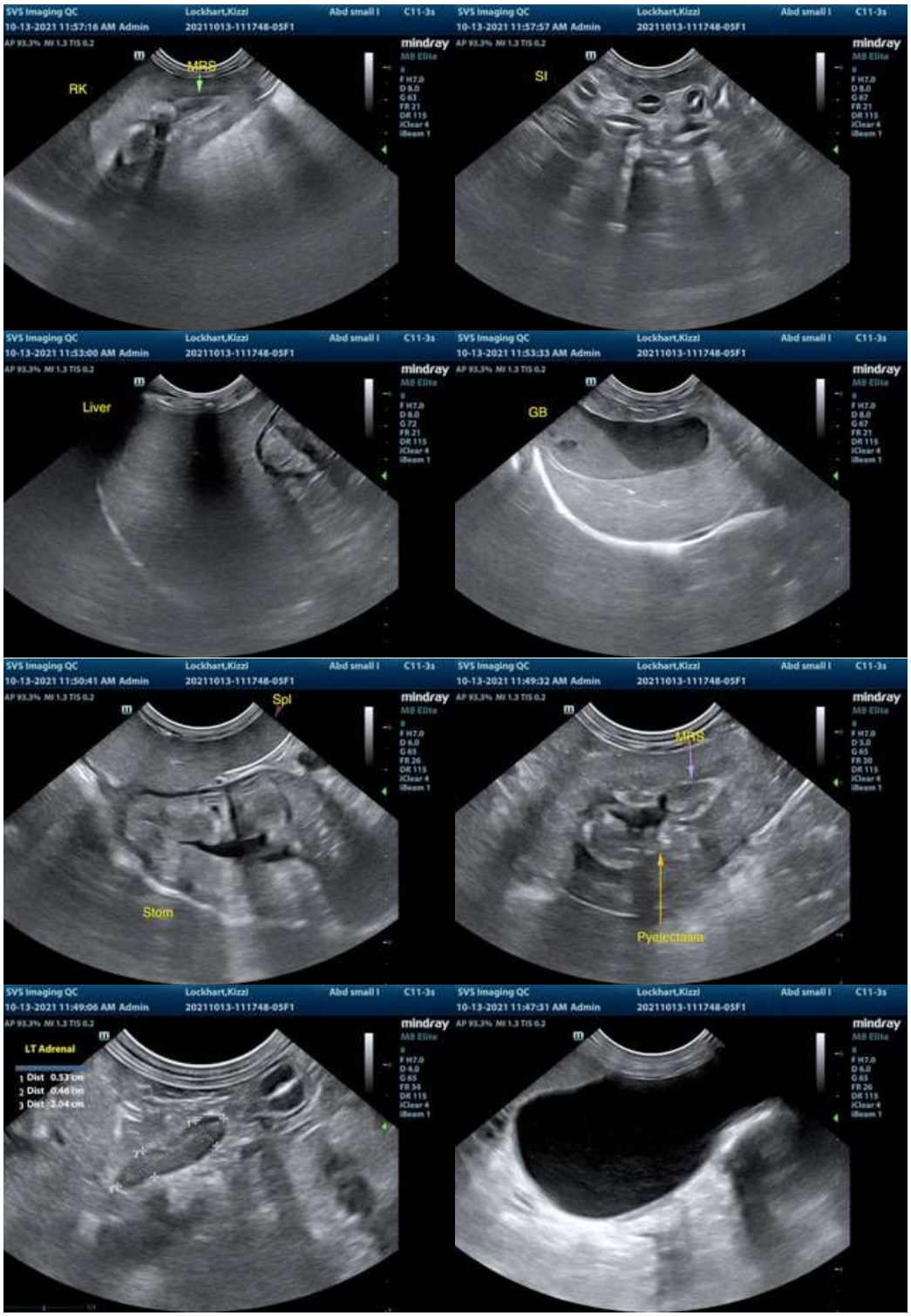

The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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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.

R. McKenzie Daniel, DVM, DABVP (Canine / Feline Practice)
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Protein-Losing Nephropathy (PLN)

http://www.sonopath.com/PLN

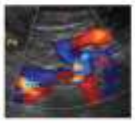
Description: Protein-losing nephropathy (PLN) is a common form of renal disease that typically affects dogs in middle age; it occurs less commonly in cats. Glomerular causes of renal protein loss encompass two broad categories: glomerulonephritis (GN) and amyloidosis. (The causes of GN in human medicine are more specifically differentiated based on a combination of histopathology, immunofluorescence, and electron microscopy findings.) Membranoproliferative glomerulonephritis is the most common cause of GN in dogs and is associated with infectious disease with secondary immune complex deposition as well as Lyme disease. Membranous nephropathy is the second most common cause of GN in dogs and the most common cause in cats. It occurs due to primary immune complex deposition on the urinary side of the basement membrane of the glomerulus, resulting in the leakage of albumin. Amyloidosis is caused by the deposition of amyloid A proteins in a β-pleated sheet configuration in the glomeruli. It is a familial disease in the Shar Pei, but occurs as a reactive disease in other canine breeds. It is also inheritable in the Abyssinian cat, but the amyloidosis occurs in the medulla and is therefore not a protein-losing condition in this breed.

Glomerular lesions can be associated with:

- Infectious diseases:
  - Protozoan: *Babesia*, *Hepatozoon*, and *Leishmania*.
  - Bacterial: *Borrelia*, *Bartonella*, *Brucella*, *Ehrlichia*, *Mycoplasma,* pyometra, pyoderma, endocarditis, and pyelonephritis.
  - Viral: FeLV, FIV, and FIP.
  - Fungal
  - Helminthic: *Dirofilaria*.
- Non-infectious inflammatory diseases: pancreatitis, chronic dermatitis, inflammatory bowel disease, periodontal disease, polyarthritis, and systemic lupus erythematosus (SLE).
- Neoplasia: lymphoma, leukemia, and mast cell disease.
- Familial conditions in the soft-coated Wheaten Terrier, Shar Pei, Beagle, Cocker Spaniel, and Bernese mountain dog.
- Idiopathic conditions.

Post-glomerular causes, such as hemorrhage and inflammation, also contribute to urine protein quantification.





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Proteinuria Classifications: Patients can be divided into three tiers, depending on their clinical characteristics:

Tier 1A: persistent subclinical proteinuria
Tier 1B: persistent proteinuria with hypertension

Tier 2A: proteinuria and hypoalbuminemia
Tier 2B: proteinuria, hypoalbuminemia, and hypertension

Tier 3A: proteinuria and azotemia
Tier 3B: proteinuria, azotemia, and hypertension
Tier 3C: proteinuria, azotemia, hypertension, and hypoalbuminemia

Diagnostics: Traditionally, urine protein loss has been detected either through a qualitative test, such as a urine dipstick, or with a semi-quantitative test, such as a urine protein-creatinine (UPC) ratio. When the latter is greater than 0.5, it is considered abnormal. False positive results can occur due to contamination of urine with red blood cells, white blood cells, and bacterial protein. Thus, one must use a urine sample with inactive sediment and a negative culture for measurement purposes. A 24-hour urine protein quantification is more accurate but technically more difficult to obtain, as it requires hospitalization and 24-hour urinary catheterization with a closed collection system. Pooling urine samples can be considered in cases where urine protein loss is stable. One must obtain three different urine samples, combine 1 ml from each sample to submit for a UPC test, and ensure that inactive sediments are present in all the samples. There should be a high degree of correlation between the UPC on the pooled sample and the mean of the three samples measured independently. Research has not yet demonstrated the accuracy of pooled samples for urine samples with high protein loss (i.e., in cases where the UPC is > 8).

Further diagnostic tests will depend on the tier classification. Once proteinuria is documented repeatedly, additional tests can be considered to assess for potential underlying causes, and, further to that, possible sources of antigen stimulation. Depending on presentation, tests may include:

- CBC and biochemical profile
- Urine culture and sensitivity
- 4DX
- Blood pressure measurement
- Thoracic and abdominal radiographs
- Spinal radiographs to assess for discospondylitis
- Abdominal ultrasound to assess for evidence of underlying infection or neoplasia
- Echocardiogram to assess for vegetative endocarditis and possible effects of hypertension
- Screen for Cushing's disease, especially if hypertensive (LDDST or ACTH stimulation)
- ANA
- Expanded tick or infectious disease screen

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- Renal biopsy to differentiate among specific causes of PLN

Renal biopsy should be considered if proteinuria is severe (UPC > 3.5) and hypoalbuminemia and/or hypertension have been documented. Renal biopsy is an invasive procedure and should be considered only to determine if there is an underlying disease process that would benefit from specific therapy. If the patient is debilitated, severely azotemic, or has uncontrolled hypertension or coagulation abnormalities, then the risk of the procedure and anesthesia may be too great and should not be pursued.

Tissue samples should be submitted for a combination of light microscopy (in formalin; use with special stains), immunofluorescence (in Michel's solution or frozen), and electron microscopy (in formalin with glutaraldehyde). It is imperative to request special media before obtaining the biopsy. Samples can be obtained via ultrasound guidance, laparotomy, or laparoscopy, but cortical samples must be divided so that they can be placed in the three different media. One must ensure that the pre-surgical clotting profile and platelet count are both normal. Patients should undergo pre-biopsy and post-biopsy diuresis.

Treatment: The main goals of therapy are to i) reduce proteinuria (i.e., UPC < 1.0); ii) prevent a thrombotic event; iii) manage hypertension; and iv) replace fluid deficits. Fluid therapy should be approached cautiously, especially in patients with nephrotic syndrome. Standard therapy for PLN includes a low-protein diet, which in itself will reduce proteinuria, and the administration of an angiotensin-converting enzyme (ACE) inhibitor, such as enalapril (0.5 mg/kg PO BID) or benazepril (0.5 mg/kg PO Q24hr). Newer proposed therapeutic protocols include increasing the ACE inhibitor dose slowly while monitoring BUN and creatinine carefully. The dose can be raised to 1 mg/kg PO BID if needed, provided creatinine has not increased more than 30% from the baseline level.

Another class of drugs currently being used is angiotensin receptor blockers, such as Losartan (the dose in azotemic dogs is 0.125-0.25 mg/kg/day PO Q12-24hr and 0.5-1.0 mg/kg/day in non-azotemic patients). This can be combined with an ACE inhibitor, but it is important to monitor BUN, creatinine, and potassium levels. Spironolactone has been used in people in combination with the other two classes of drugs to further modify the renin-angiotensin-aldosterone system (RAAS) (1-2 mg/kg PO BID); however, the effect of using all three drug classes in dogs has not yet been fully investigated. All of these medications are potassium sparing; thus, monitoring for hyperkalemia is important.

Hypertension is managed with amlodipine (0.1-0.2 mg/kg PO Q12-24hr) when an ACE inhibitor is insufficient to control blood pressure. Supplementing with an anti-thrombotic agent, such as aspirin (1 mg/kg PO Q24hr), may be considered in advanced cases, especially once the patient is hypoalbuminemic. Omega-3 fatty acids can be given (0.25-0.5 g/day), but are typically increased in standard kidney diets.

The most recent controversy in the management of glomerular diseases is the use of immunosuppressive medications. Because it is possible to arrive at a more definitive

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diagnosis in human patients, the use of immunosuppressive agents can be useful in the management of the disease, specifically when the disease is immune-mediated in its pathogenesis, such as SLE, membranous nephropathy, and minimal change disease glomerulonephritis. The procurement of a renal biopsy is being advocated in dogs so that practitioners can identify the population of patients that may benefit most from immunosuppressive therapy. Presently, there is no evidence-based medicine to suggest that immunosuppressive therapy should definitely be incorporated into a daily protocol for canine patients; however, it could be beneficial in some cases and may even result in remission. Further investigation is warranted. Trials are currently being conducted in patients with Lyme nephritis that are treated with immunosuppressive agents in addition to standard antibiotic therapy. The IRIS Treatment of Canine Glomerular Disease Study Group has suggested the trial use of immunosuppressive therapy in severe, persistent, or progressive PLN, even without a biopsy diagnosis in specific cases that are unresponsive to standard therapy (i.e., nephrotic syndrome, progressively azotemic, hypoalbuminemic patients). One can also consider administering the following drugs: pulse steroid therapy, myclophenolate, cyclosphosphamide, azathioprine, and chlorambucil. One should monitor blood work, UPC ratio, and blood pressure weekly for 2 weeks, then biweekly for 6 weeks, then monthly. If there is further deterioration, immunosuppressive therapy should be discontinued.

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