



PATIENT

Prince NV-Pollaehne

SPECIES

Canine

BREED

German Shepherd

SEX

Intact Male

AGE

5 Years

WEIGHT

52 Pounds

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Cara, CVT, RDMS

HOSPITAL NAME

1st Pet VC- North
Valley

REFERRING VET

Dr. Wachtel

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13395

DATE

10/1/21

PRESENTING CLINICAL SIGNS

History: History Hx of a suspected colitis with chronic intermittent loose stool. A fecal was performed at some point. They have tried a GI diet and probiotics. He'll eat dog food one day and need sandwich meat or something else. For the past 4 weeks, his eyes have seemed watery and the vet recommended Benadryl. He's been drinking more water than normal starting last night. Last night, he was laying on his bed and passing a clear liquid stool that smelled foul. He's been chewing at a wooden bench and a curtain. The owner gave ondansetron and famotidine PO 2 days ago that may have helped somewhat. Sometimes he'll pass undigested food and sometimes he'll have a "mushy" diarrhea.

Abnormal PE/Chem/CBC/UA Results: XRAYS 5 electronically transmitted radiographs dated October 1, 2021 were reviewed. These include right lateral, left lateral and ventrodorsal projections of the abdomen, and lateral and ventrodorsal projections of the thorax. The patient is thin with reduced muscle mass. The cardiac silhouette is small. The caudal vena cava is mildly enlarged. The pulmonary vessels are unremarkable. The pulmonary parenchyma is unremarkable. There is decreased abdominal serosal detail, and the lack of intra-abdominal fat. Within this limitation, the liver is within normal limits. The stomach is mildly fluid and gas distended. There are segments of small intestine which are relatively empty. There are other segments of small intestine which are mildly fluid and gas distended the colon is moderately gas distended Conclusion Segmental distention of small intestine of unknown significance. Ultrasonography may be of value in further assessing this patient. Hypovolemia. Jim Hoskinson, DVM, DACVR 10/1/2021 11:25:10am Chem 17 elect: hyponatremia, hypokalemia, hypocalcemia (ionized) CBC: mild non-regenerative anemia, monocytosis with basophilia Chem: hypocalcemia (total), hypoproteinemia (alb and globulin), elevated ALT, elevated GGT, elevated t. bili, hypocholesterolemia PCV 54%, TS 3.2

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. 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 were 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 right kidney measured 7.3 cm. The left kidney measured 7.8 cm.

The pelvis was poorly visible in this patient.

Adrenal Glands

Both **adrenal glands** were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The left adrenal gland measured 0.53 cm at the caudal pole and 0.63 cm at the cranial pole. The right adrenal gland measured 0.6 cm.

Spleen



PATIENT	The visible spleen was unremarkable and uniform with no evident pathology noted.
Prince NV-Pollaehne	Liver
SPECIES	The liver was mildly swollen with irregular contour and increased portal markings. Localized free fluid was noted. Enhanced surrounding mesentery noted. The gallbladder was collapsed and empty. No evidence of posthepatic obstruction.
Canine	Gastrointestinal
BREED	A large amount of GI artifact noted. Mucosal fogging noted in portions of the small intestine. Fluid filled colon noted.
German Shepherd	Pancreas
SEX	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.
Intaact Male	Free Abdomen
AGE	Free fluid noted throughout the mid abdomen . The mid abdomen was difficult to visualize owing to excessive GI gas.
5 Years	
WEIGHT	ULTRASONOGRAPHIC FINDINGS
52 Pounds	<ul style="list-style-type: none"> • Undefined free fluid • Swollen liver with irregular contour
INTERPRETED BY	INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS
Eric Lindquist, DMV DABVP, Cert. IVUSS	Protein enteropathy suspected given the mucosal fogging noted. Underlying leptospirosis should also be considered as a potential. FNA of the liver warranted for further definition. No obvious evidence of neoplasia, yet cannot be entirely ruled out. If albumin levels are <1.5, then this would justify the free fluid owing to poor oncotic pressure from protein losing enteropathy. Ampicillin, metronidazole and the following protocol would be recommended to treat PLE. Further definition based on leptospirosis titers and FNA of the liver.
IMAGING PERFORMED BY	PLE Therapy
Cara, CVT, RDMS	Part or all of this protocol may be considered based on your clinical impression of the patient:
HOSPITAL NAME	OBJECTIVE: keep albumin levels > 2 g/dl, avoid thromboembolism and cavitory effusions, monitor concurrent PLN (Wheaton Terrier PLE/PLN) and liver disease:
1 st Pet VC- North Valley	Plasma 10 mL / kilogram IV over 4 hours
REFERRING VET	Or Human albumin 2 ml/kg/h over 10 hours. Total daily volume 20.l/kg/day
Dr. Wachtel	And Colloids/Hetastarch
INVOICE	10 to 20 mL per kilogram per day and dogs
13395	10 to 15 mL per kilogram per day cats
DATE	(Can bolus first 1/3 of dose over 15 minutes)
10/1/21	& maintain on LRS maintenance otherwise.
	Metronidazole (10-20 mg/kg po bid)



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Famotidine 1 mg/kg Iv Im po dc Sid /bid

Sucralfate 0.5-1 g po tid dogs, 0.5 g bid cats in slurry **Or Misoprostol** 1-5 ug/kg po tid

Diet: Highly digestible high quality protein, low fiber, low fat diet (< 15% of dry matter). Hydrolyzed protein or novel protein. Purina HA or Royal Canine HP or similar.

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Prednisone or prednisolone 2 mg/kg bid x 3-5 days then 2 mg/kg sid. **Chlorambucil** in refractive severe IBD/alimentary lymphoma cases (monitor cbc for rare bone marrow suppression) 4 mg/m² Q 24-48 hours.

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Cobalamine (B12) 250-1500 ug/dog weekly x 6 weeks.

Calcium supplementation if necessary.

Aspirin 0.5-1 mg/kg/day **or Clopidrel** (Plavix) 1-5 mg/kg/day.

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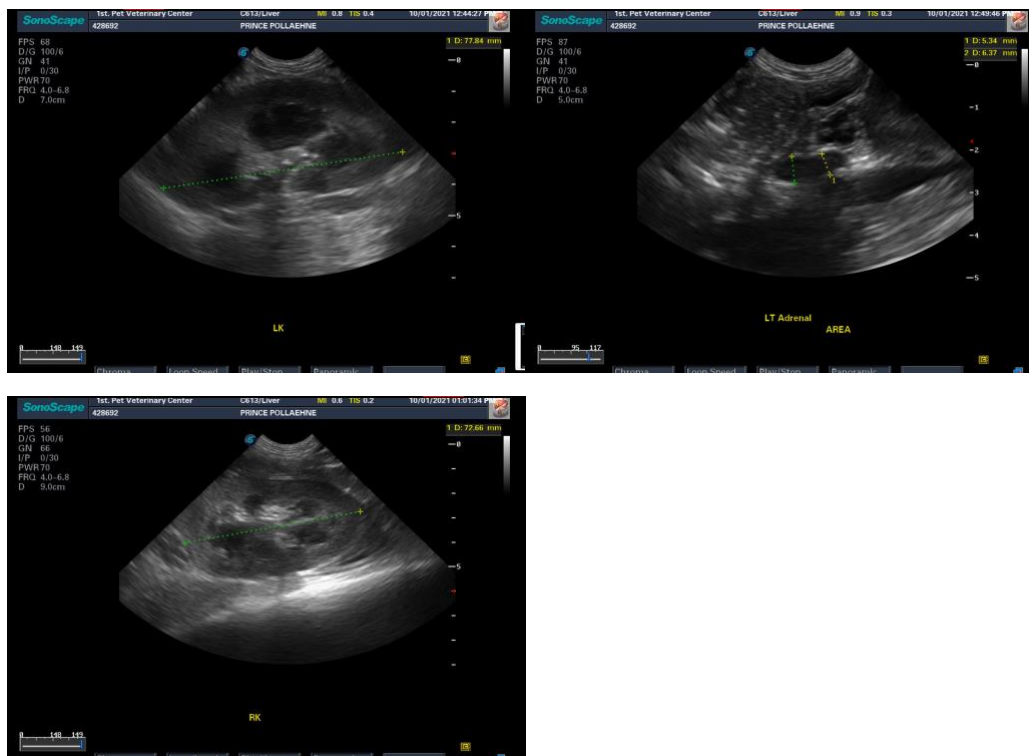
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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

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



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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.

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Glomerular lesions can be associated with:

BREED

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- 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.

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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

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Tier 2A: proteinuria and hypoalbuminemia

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Tier 2B: proteinuria, hypoalbuminemia, and hypertension

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Tier 3A: proteinuria and azotemia

Tier 3B: proteinuria, azotemia, and hypertension

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

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Diagnosics: 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



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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
- 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



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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.

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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.

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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.

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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.

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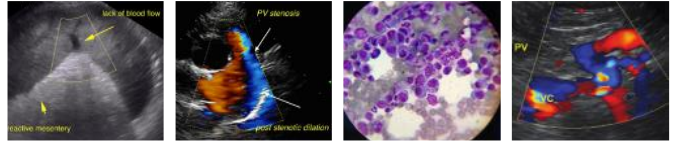
The most recent controversy in the management of glomerular diseases is the use of immunosuppressive medications. Because it is possible to arrive at a more definitive diagnosis in human patients, the use of immunosuppressive agents can be useful in the management of the disease, specifically when the 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

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unresponsive to standard therapy (i.e., nephrotic syndrome, progressively azotemic, hypoalbuminemic patients). One can also consider administering the following drugs: pulse steroid therapy, myclophenolate, cyclophosphamide, 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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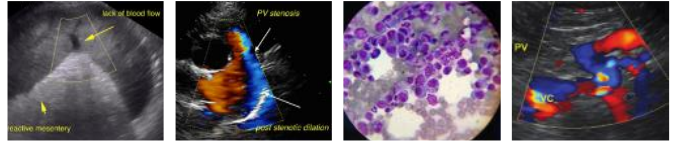
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