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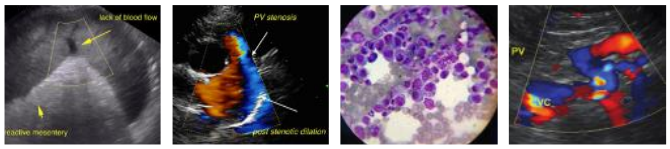

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1-800-838-4268 info@sonopath.com

PATIENT

Superman Mcleod

SPECIES

Canine

BREED

Border Collie Mix

SEX

Neutered Male

AGE

6 Years

WEIGHT

16.5 kg

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Patti Mayfield, DVM

HOSPITAL NAME

Bend AESC

REFERRING VET

Dr. Cait Lacey

INVOICE

12711

DATE

11/28/21

PRESENTING CLINICAL SIGNS

History: Primary complaint and history for this visit:*** Pt here for D+++ two days, suspected pale gums at home, sudden ataxia when walking down steps this evening. O has no suspicions of eating anything undigestible. Asked about rat bait; she was recently replacing rat bait in traps today but pt's symptoms started yesterday. Housing/Environment:*** access to 2 acres, Sunriver; moved here from Georgia 4yrs ago & Colorado/Wyoming a few months ago Normal activity level, including ball playing w/other dogs. This morning, he acutely slowed down.

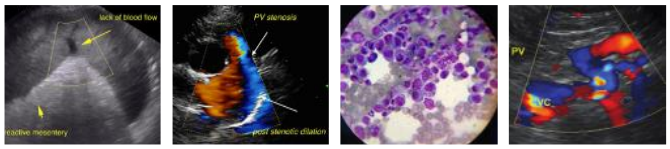
Abnormal PE/Chem/CBC/UA Results: Physical exam: Temp: 105.3 F Mildly tachypneic w/mild dyspnea & mild abdominal component to respiration, no overt wheezes or crackles ausculted x4 quadrants Abdomen:Moderately tense w/mild discomfort on abdominal palpation, palpable fluid wave; no internal masses or organomegaly readily appreciated Light pink mucous membranes, tacky. AFAST: Significant ascites. Abdominocentesis: transudate (clear liquid) AFAST (intake): Score 4/4 w/no overt splenic masses appreciated, urinary bladder intact w/no abnormalities appreciated, gall bladder unremarkable CBC: Overall unremarkable CHEM17: Mild hypoglycemia 68, **hypoproteinemia 4.1 (5.2-8.2) w/associated severe hypoalbuminemia 1.6 (globulins low normal 2.5)**, hypocalcemia 7.2, hypocholesterolemia 102 LYTES: Hypokalemia 3.0, hyponatremia 143 PCV/TS (peripheral): 61/4.0 PCV/TS (peritoneal effusion): 0/0 4Dx: Negative x4 RADS (2-view thoracic): Cardiac silhouette objectively appears mildly enlarged, scant pleural effusion noted along caudal left region, diaphragm intact, included skeletal structures are unremarkable

ULTRASONOGRAPHIC EXAMINATION OF THE HEART & ABDOMEN

CANINE CARDIAC PARAMETERS	MR VMAX (m/s)	TR VMAX (m/s)	LA/AO (Boon method)	LA/AO (Heart Base; Swe)	FS (%)	EF (%)	EPSS (cm)
NORMAL PARAMETER	4.5-5.5	<2.7	1.3	<1.6	28-40	40-100	<0.6
PATIENT	--	--	NM	1.16	26	54	0.57
CANINE CARDIAC PARAMETERS	**HR** (BPM)	**AV VMAX** (m/s)	**PV MAX** (m/s)	**BODY WEIGHT** (kg)	**LA** 2D short axis Base view (cm)	**LVIDd** Avg; 2D and m-mode short axis (cm)	**LVIDs** Avg; 2D and m-mode short axis (cm)
NORMAL PARAMETER	50-100	0.7-1.7	0.7-1.6	BELOW	BELOW	BELOW	BELOW
PATIENT	--	--	--	--	3.13	2.68	--

Cardiac Presentation

The echocardiogram in this patient demonstrated normal **left atrial** size based on 3 separate methods of LA evaluation. Mitral insufficiency noted, not clinically significant, no volume overload. Mitral insufficiency was centralized and compensated. The **left ventricle** presented thicknesses with linear contour and was not dilated nor restricted. The **myocardium** presented normal echogenicity without subjective evidence of significant fibrotic or ischemic disease. **Contractility** of the ventricular



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walls was adequate and in normal range for this patient evidenced by the fractional shortening measurement and subjective evaluation of the different regions of the myocardium. The **left ventricular outflow** tract demonstrated normal laminar flow and subjective structural integrity. The **right atrium** and auricle revealed normal size, structure and content. No evidence of masses was noted. **Tricuspid** valvular assessment demonstrated adequate linear morphology and kinesis. The **right ventricle** was of normal size (1/3 diameter of LV), chordae structure, myocardial echogenicity and thickness. **Pulmonary outflow** tract assessment revealed normal valve structure, laminar flow, and diameter (approx.1:1 pa/ao ratio). No visible **pericardial** or free pleura fluid was noted. The cranial **mediastinum and pericardial and extra-cardiac regions** were free of masses in the visible window.

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 left kidney measured 5.5 cm. The right kidney measured 5.5 cm.

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 right adrenal gland measured 0.65 cm at the cranial pole and 0.48 cm at the caudal pole. The left adrenal gland measured 0.44 cm at the cranial pole and 0.52 cm at the caudal pole.

Spleen

The **spleen** was mildly enlarged with slight scalloping contour. No overt masses noted. No evidence of thrombosis.

Liver

The **liver** was subnormal in size with increased portal markings. Coarse architecture was noted throughout the liver consistent with sequelae from chronic inflammatory hepatopathy. No evidence of passive congestion. The gallbladder and common bile duct were unremarkable.

Gastrointestinal

The **gastric** wall was mildly thickened in this patient with empty lumen. The gastrointestinal tract revealed diffuse, hyperechoic fogging or overlay throughout the small intestine as well as areas of mucosal striations and speckling. This striation + fogging effect appeared to exclusively affect the mucosal layer with the submucosa, muscularis and serosa left in-tact. Reactive mesentery was present associated with the serosa indicative of active inflammation. This is most consistent with protein losing enteropathy/lymphangectasia. Full thickness biopsies or endoscopy guided biopsies would be ideal to confirm. No obstructive disease or obvious suspicion of neoplasia.

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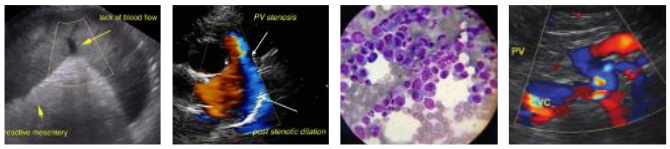

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Pancreas

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

- Stage B-1 valvular disease- Not a clinical issue in this case. No evidence of right auricular pathology present.
- Microhepatica with chronic inflammatory hepatopathy pattern
- Splenomegaly, likely owing to portal hypertension
- Gastric wall mildly thickened. Mucosal fogging.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

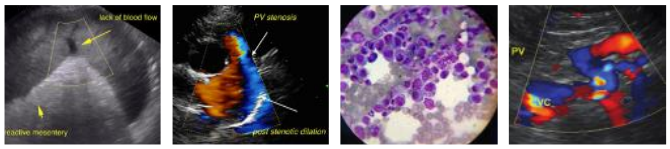
Bile acid profile warranted. I'm presuming that it will be elevated owing to the diffuse hepatic disease. The ascites is likely owing to a combination of issues given the hypoalbuminemia and hypocalcemia, this would fit protein losing enteropathy, especially with the mucosal fogging noted in the small intestinal tract. The low cholesterol can occur in PLE, however, it would fit end-stage hepatic disease. Given that the albumin was low, yet not under 1.5, complicating hydrostatic pressure owing to portal hypertension is likely combining with low albumin levels and poor oncotic pressure to create the ascites. The splenic enlargement is likely owing to portal hypertension with splenic vein congestion. Underlying neoplasia cannot be completely ruled out in this patient, especially if any cortisone has been in this patients' empirical therapy history, however, protein losing enteropathy and chronic inflammatory hepatopathy with secondary portal hypertension is likely the case.

Prognosis is extremely guarded to poor long-term depending upon response to therapy. Liver biopsy would be ideal, however, may be somewhat risky given the ascites and hemorrhage would be able to be ascertained upon sampling from ultrasound guided perspective. The cause of the fever is unclear. Leptospirosis should be ruled out as an underlying player.

PLE Therapy

Part or all of this protocol may be considered based on your clinical impression of the patient:

OBJECTIVE: keep albumin levels > 2 g/dl, avoid thromboembolism and cavitary effusions, monitor concurrent PLN (Wheaton Terrier PLE/PLN) and liver disease:
Plasma 10 mL / kilogram IV over 4 hours
Or **Human albumin** 2 ml/kg/h over 10 hours. Total daily volume 20.l/kg/day
And Colloids/Hetastarch
10 to 20 mL per kilogram per day and dogs
10 to 15 mL per kilogram per day cats
(Can bolus first 1/3 of dose over 15 minutes)
& maintain on LRS maintenance otherwise.
Metronidazole (10-20 mg/kg po bid)
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



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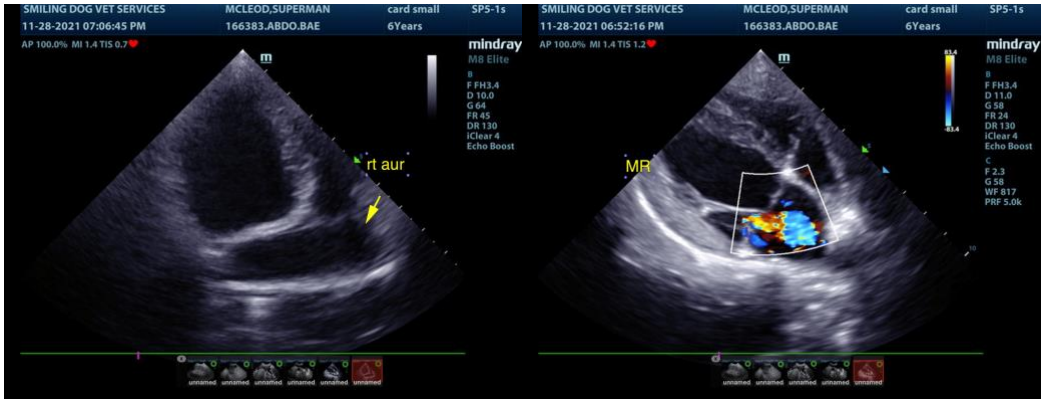
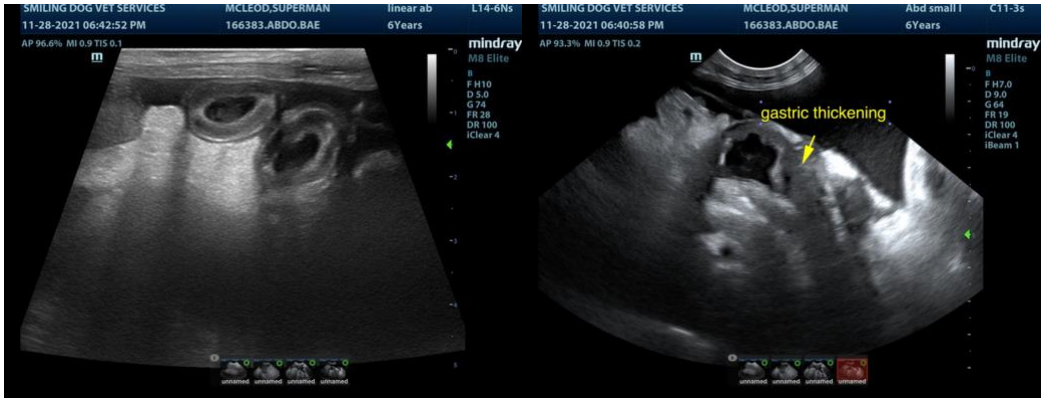
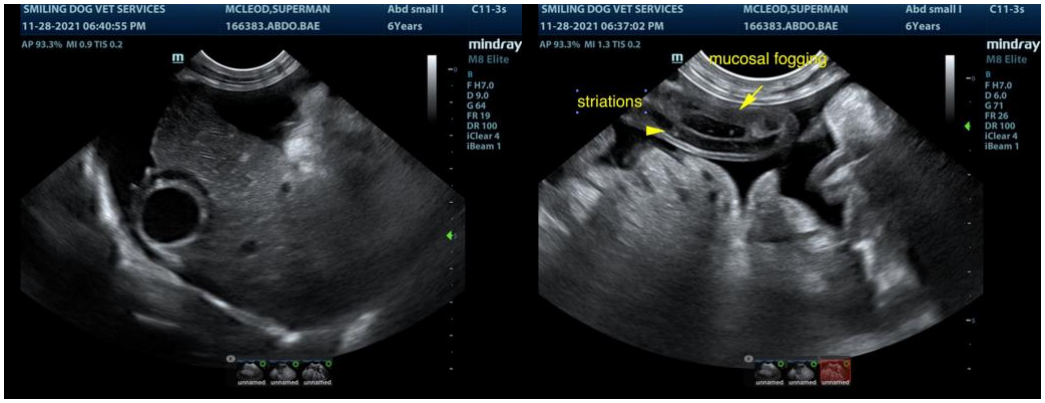
Diet: Highly digestible high quality protein, low fiber, low fat diet (< 15% of dry matter). Hydrolyzed protein or novel protein. Purina HA or Royal Canine HP or similar.
Prednisone or prednisolone 2 mg/kg bid x 3-5 days then 2 mg/kg sid. **Chlorambucil** in refractive severe IBD/alimentary lymphoma cases (monitor cbc for rare bone marrow suppression) 4 mg/m^2 Q 24-48 hours.
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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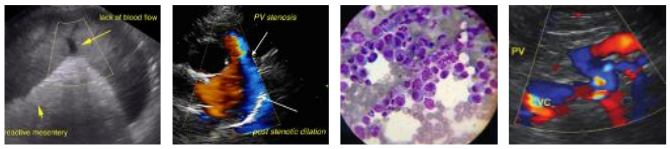

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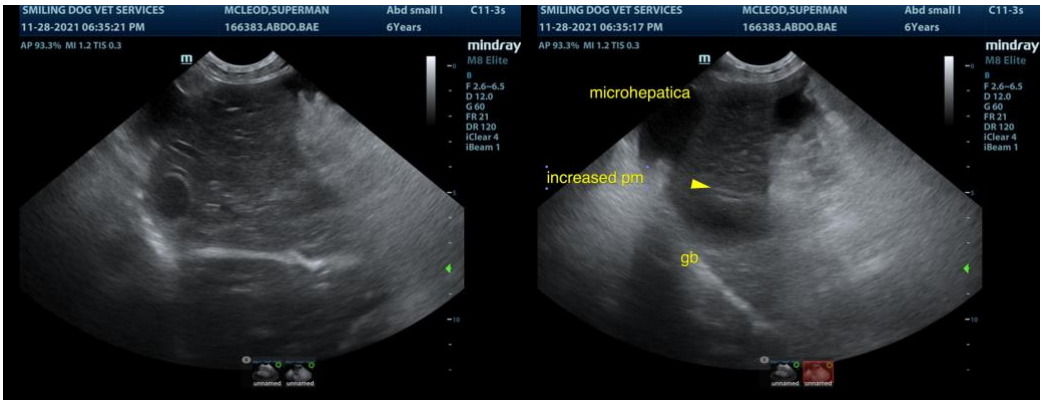

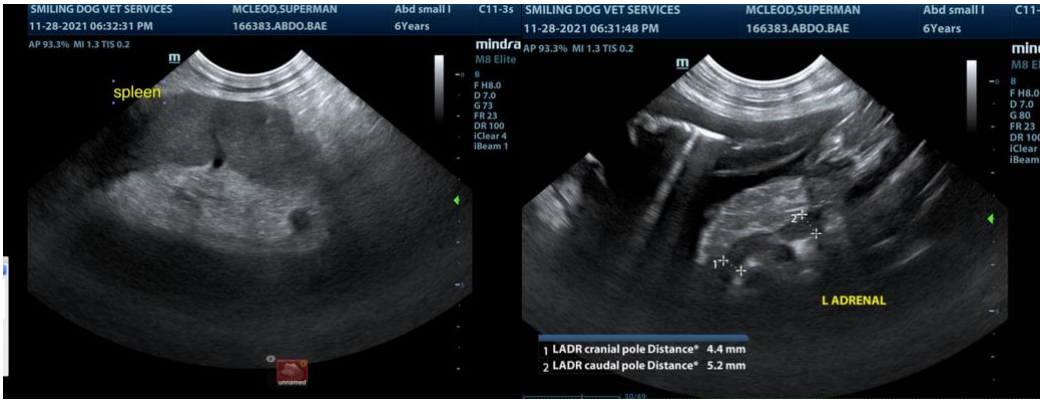

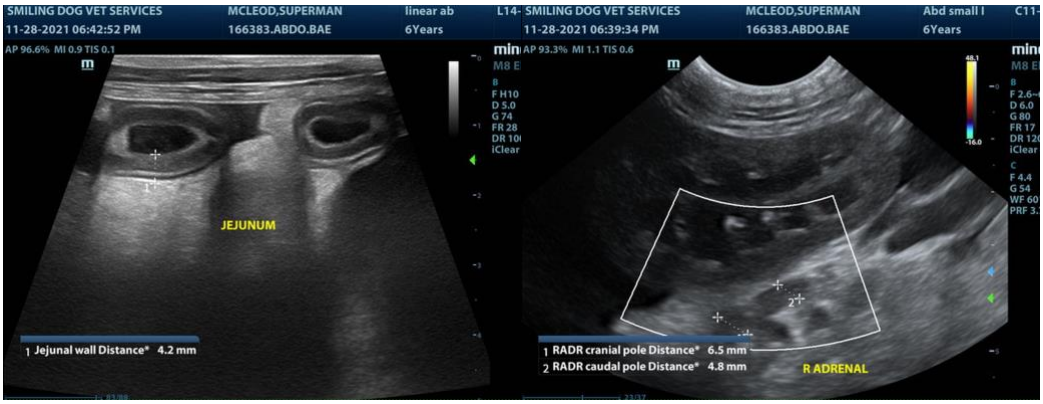

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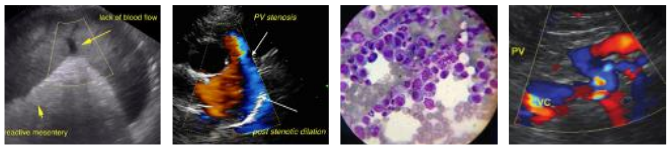

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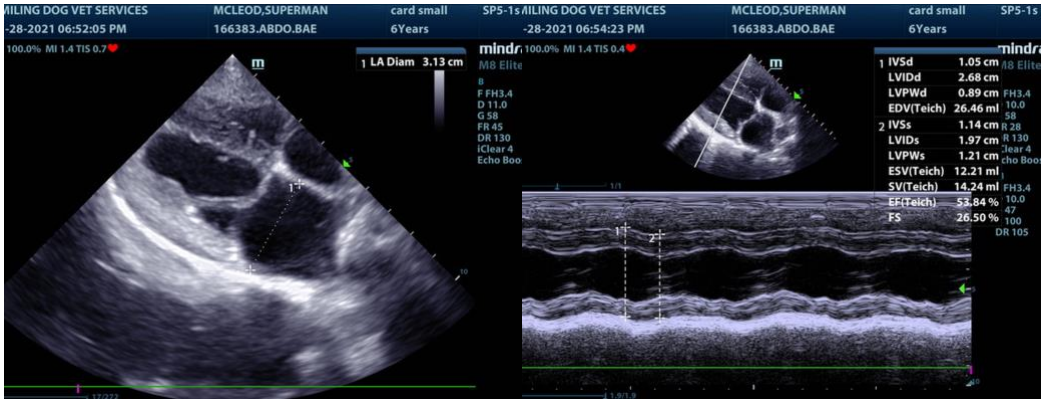

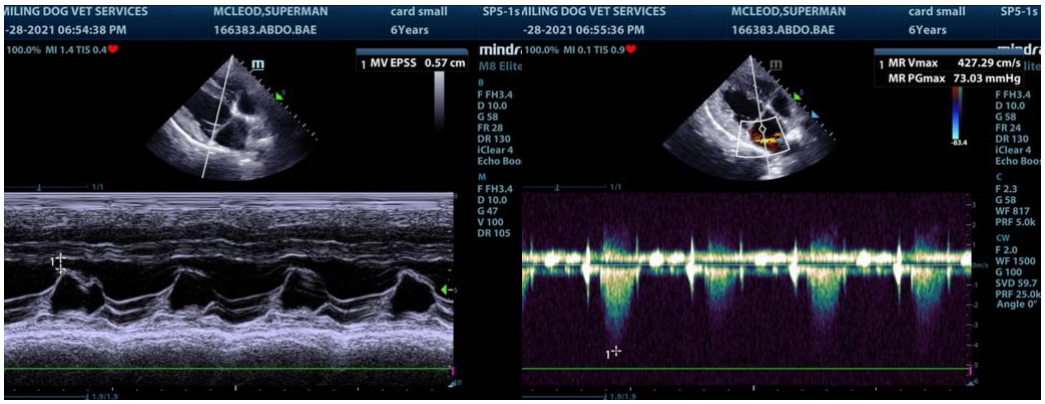

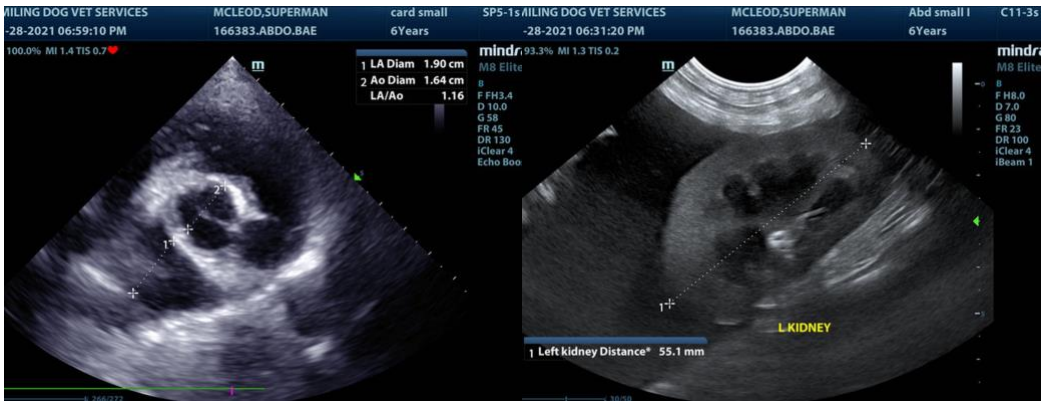

The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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)

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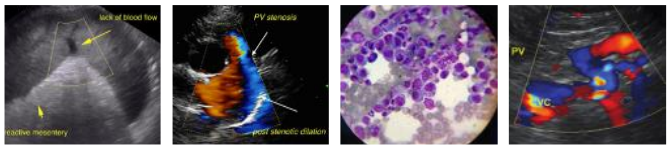

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http://www.sonopath.com/PLN

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.

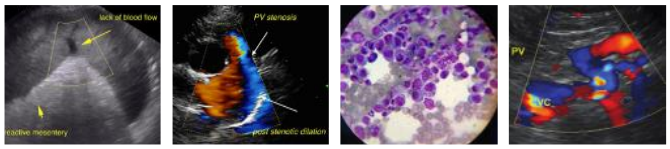
Post-glomerular causes, such as hemorrhage and inflammation, also contribute to urine protein quantification.

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



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Tier 2B: proteinuria, hypoalbuminemia, and hypertension

Tier 3A: proteinuria and azotemia

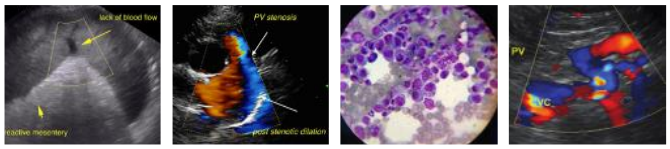
Tier 3B: proteinuria, azotemia, and hypertension

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
- Renal biopsy to differentiate among specific causes of PLN



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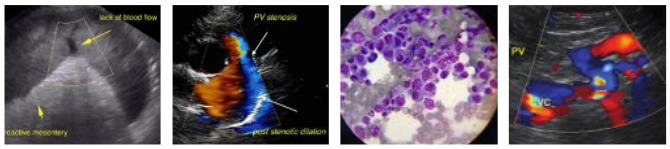
Renal biopsy should be considered if proteinuria is severe (UPC > 3.5) and hypoalbuminemia and/or hypertension have been documented. Renal biopsy is an invasive procedure and should be considered only to determine if there is an underlying disease process that would benefit from specific therapy. If 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.



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PATIENT

Superman Mcleod

SPECIES

Canine

BREED

Border Collie Mix

SEX

Neutered Male

AGE

6 Years

WEIGHT

16.5 kg

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Patti Mayfield, DVM

HOSPITAL NAME

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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 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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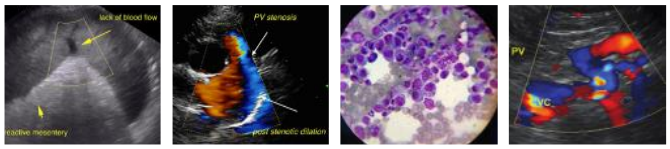

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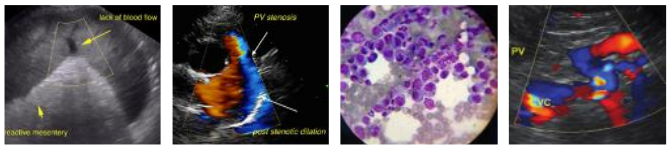
Fever of Unknown Origin

http://www.sonopath.com/FUO

Description: The definition of a fever of unknown origin (FUO) has not been clearly defined for animals. Currently, it is either understood to be a fever that does not resolve within the period one would expect for a "self-limiting infection" being treated with appropriate antimicrobial therapy, or that for which an underlying diagnosis has not been determined despite considerable diagnostic effort. The common causes of FUO were summarized concisely in a presentation at the American College of Veterinary Internal Medicine 2004 Forum. The presenters synthesized information from three veterinary papers on the subject, which suggested the following:

Final Diagnosis	Bennett (dogs & cats)	Dunn and Dunn (dogs only)	Lunn (dogs & one cat)	Total
Infection	21	16	10	47
Immune	18	22	6	46
Bone marrow disease	4	22	2	28



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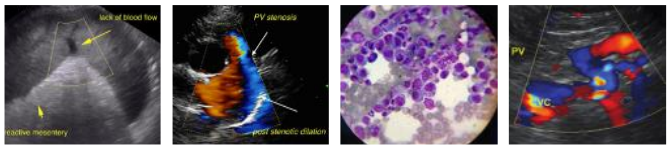
Final Diagnosis	Bennett (dogs & cats)	Dunn and Dunn (dogs only)	Lunn (dogs & one cat)	Total
Neoplasia (outside marrow)	0	10	2	12
Miscellaneous	2	12	2	16
No diagnosis	0	19	2	21
TOTALS	45	101	24	170

The types of infection diagnosed in this case series were varied, ranging from discospondylitis (8 cases), blastomycosis (6), and bacterial endocarditis (4), to leishmaniasis (1), prostatitis (1), and *Ehrlichia canis* infection (1); a multitude of other infectious causes also fell within the spectrum. Of the cases in which immune-mediated disease was found, 44% had immune-mediated polyarthritis. Bone marrow diseases included myeloproliferative disease, myelodysplasia (8), lymphocytic leukemia (8), myeloma (3), chronic granulocytic leukemia (3), lymphoblastic leukemia, and malignant histiocytosis. The types of neoplasia located outside the bone marrow included lymphoma (6), metastatic disease (2), and neoplasms of the lung, spleen, and stomach. Finally, miscellaneous diseases included hypertrophic osteodystrophy (6), meningitis (3), portosystemic shunt (3), lymphadenitis (2), panosteitis, and intervertebral disc disease. Overall, the most common causes across all cases were polyarthritis (44), lymphoid neoplasia (15), discospondylitis (8), myelodysplasia (8), hypertrophic osteodystrophy (6), and blastomycosis (6).

Clinical Signs: Animals usually present with either persistent or waxing and waning fevers ranging from 103ºF to 106ºF. Other clinical signs depend on the underlying cause of the fever. Careful and thorough physical examination is required to assess potential causes.

Diagnostics: FUO etiologies are partly related to geography, and thus locale or travel history should factor into a practitioner's diagnostic approach. A patient's lifestyle may also provide clues regarding exposure to certain etiologic agents. Therefore, conducting a thorough history can unveil important pieces of the diagnostic puzzle. Physical examination is especially important and should include an inspection of all accessible lymph nodes, palpation and movement of the joints, a fundic examination, a neurological evaluation, spinal and limb palpation and range of motion tests, and a rectal examination.

A minimum database should include a CBC reviewed by a clinical pathologist, as well as a biochemical profile and urinalysis. Retroviral testing should also be considered in cats. In areas where tick-borne disease is prevalent, in-house testing should be performed early. Advanced laboratory work can include: urine culture, blood culture, and infectious disease panels (PCR and/or serology). In dogs, one may screen for the following infectious agents: *Ehrlichia* spp., *Borrelia burgdorferi*, Rock Mountain Spotted Fever, *Bartonella* spp. (culture and PCR), and *Leptospira* spp. in cases of hepatic or renal involvement. In cats, one should evaluate for FeLV, FIV, feline infectious



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peritonitis (FIP) virus, toxoplasmosis, *Hemoplasma* spp. (*Mycoplasma*), and *Bartonella* spp. (culture and PCR). Testing for *Ehrlichia* spp., *Rickettsia* spp., and *Anaplasma phagocytophilum* can also be considered. A fungal assay is indicated if the patient lives in or has had exposure to a region with a higher incidence of fungal disease. Other infectious disease tests may be performed depending on the geographical location of the pet. Screening for *Brucella* should be done in breeding dogs. Immune-mediated disease screening can include a Coomb's test, a slide agglutination test (if the patient is anemic), and an antinuclear antibody (ANA) test. Immune disease is often a diagnosis of exclusion.

Imaging should include thoracic radiographs, abdominal ultrasound, and/or abdominal radiographs. Ultrasound can be very useful for assessing evidence of cholangiohepatitis, pyelonephritis, chronic urinary tract infection, abscess formation, peritonitis, and neoplasia; it also permits an examination of the intra-abdominal lymph nodes. An echocardiogram can offer assessment for vegetative endocarditis, whereas spinal radiographs offer assessment for discospondylitis. In cases where all other testing has proven negative and the patient has not responded to broad-spectrum antibiotics and supportive care, arthrocentesis should be considered to evaluate for septic joint disease, immune-mediated polyarthritis, and infectious disease. Finally, one can consider assessing the cerebrospinal fluid for meningoencephalitis, GME, and meningitis/arteritis. A bone marrow exam should be performed if blood dyscrasias are noted on the CBC.

Treatment: Treatment of the fever depends entirely on the underlying cause. Ideally, a thorough diagnostic plan will yield a diagnosis that will guide the appropriate therapeutic course. However, if an exhaustive approach has not produced a definitive diagnosis and there is no response to broad-spectrum antibiotics, trial therapy with immunosuppressive agents such as prednisolone can be considered to treat presumed immune-mediated diseases. Given the potential for negative sequelae should an underlying infection be present, one must be certain that the investigation is thorough and monitor the patient's response carefully.

Conclusion: If a documented fever has not responded to antibiotics, antipyretics, or general nursing care, it is important to obtain a diagnosis to guide more specific treatment. A systematic physical examination and thorough history-taking will help inform further diagnostics in addition to what is revealed by the minimum database.

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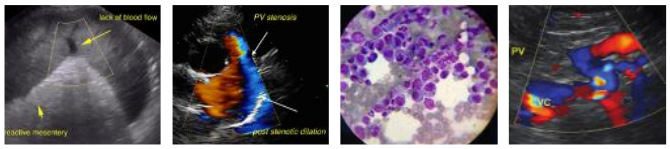

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