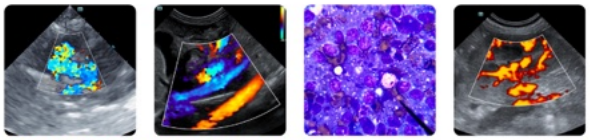

PATIENT

Tucker Johnson

SPECIES

Canine

BREED

King Charles Cavalier

SEX

Neutered male

AGE

7 years

WEIGHT

21 lbs

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

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PRESENTING CLINICAL SIGNS

History: Symptoms started a week ago. V/D and hypoalbuminemia, low normal globulins. Low TP. No protein in urine.
Recheck: persistent loose stools with blood-tinged stool noted this morning; GI signs ongoing since initial presentation. No vomiting since initial visit. Eating and drinking well, attitude still lethargic.
History of cervical/neck pain: o reports pt improving; jumping on couch again, moving normally. On carprofen, d/c today.
Abnormal PE/Chem/CBC/UA Results: Persistent hypoalbuminemia with hypoproteinemia, normal globulin, on both dates: -- Jun 17: TP 4.6 (5.2-8.2), albumin 1.8 (2.3-4.0), globulin 2.8, A:G 0.6 -- Jun 14: TP low (same range), albumin low, globulin 2.9, A:G 0.6 Low-normal BUN (9 on Jun 14) and low-normal cholesterol (114). Liver enzymes are unremarkable (ALT 37-48, ALP 27, GGT 0, total bili 0.2) -- No azotemia (creat 0.8-0.9, BUN 9-18), USG 1.034 -- UA (free catch): dipstick protein TR only, blood 1+, LE 2+ (leukocyte esterase , pH 6.5, USG 1.034 -- Sediment: WBC 4/HPF, RBC 3/HPF, cocci "suspect presence," squamous <1, non-squamous epi 1-2/HPF

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 was noted. Ureteral papillae were normal.

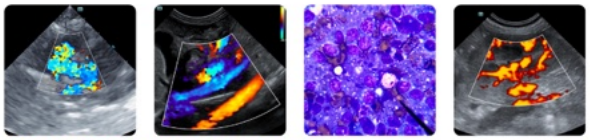
The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The right kidney measured 4.75 cm. The left kidney measured 4.02 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 left adrenal gland measured 1.84 x 0.34 cm. The right adrenal gland measured 1.52 x 0.33 cm.

Spleen

The **spleen** in this patient was mildly enlarged with parenchymal changes and was folded upon itself cranially. This is a positional variant and is not pathological. There was no evidence of significant disease.



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Liver

The **liver** images from right and left intercostal as well as subcostal views revealed subjectively normal liver size, contour, and structure. Some age-related parenchymal remodeling was noted but likely not clinically significant at this time. Vascular and biliary tracts were of normal volume and no evidence of congestion was noted. Coalesced bile was noted with areas of mineralization in the gallbladder.

Gastrointestinal

The **gastrointestinal** presentation revealed mild uniform prominence of the gastric mucosa as well as areas of "ropey" small intestinal wall with slight disruption of the normal 1:3 muscularis/mucosal ratio. Increased submucosal layering was noted. This is indicative of chronicity. The wall measured up to 0.3 cm. Mucosal fogging and striations were noted in portions of the small intestine. The cecum was mildly thickened. Reactive mesenteric lymph nodes measuring up to 1.2 x 0.56 cm.

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

Chronic inflammatory bowel. Lymphangectasia pattern.

Age related abdominal changes noted otherwise.

Reactive mesenteric lymph nodes.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Full thickness GI biopsies would be ideal in this patient for long term management. There was no evidence of neoplasia. Screening for occult Addison's is also indicated even though the adrenal glands appear normal, yet this can be a cause of protein loss.

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)

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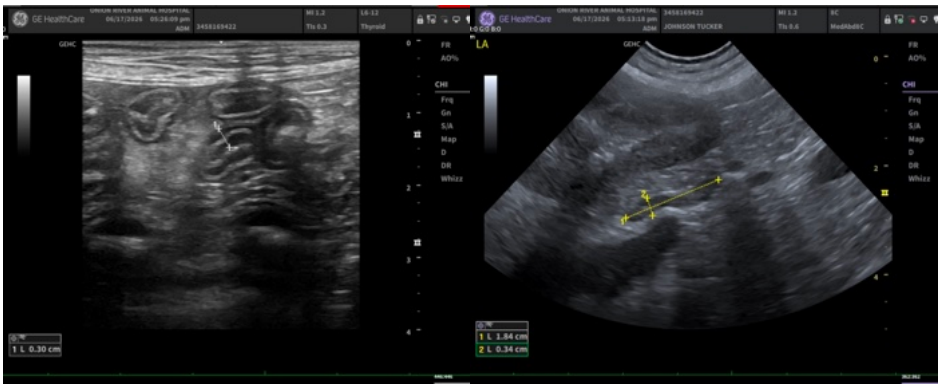
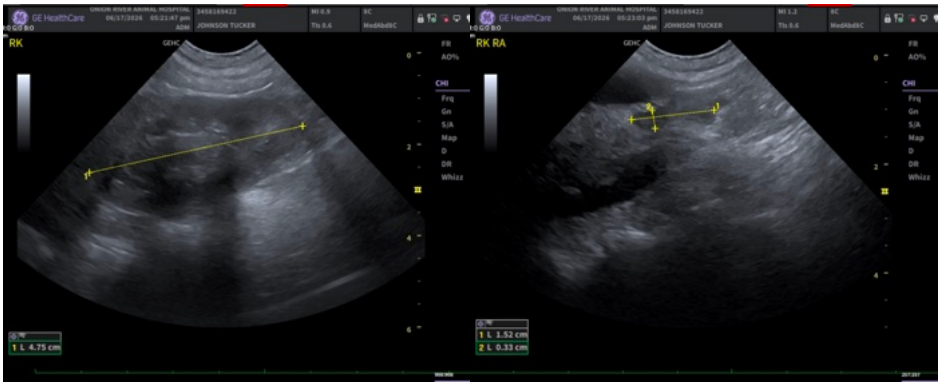
& maintain on LRS maintenance otherwise.
Metronidazole (10-20 mg/kg po bid)
Famotidine 1 mg/kg Iv Im po dc Sid /bid
Sucralfate 0.5-1 g po tid dogs, 0.5 g bid cats in slurry **Or Misoprostol** 1-5 ug/kg po tid
Diet: Highly digestible high quality protein, low fiber, low fat diet (< 15% of dry matter). 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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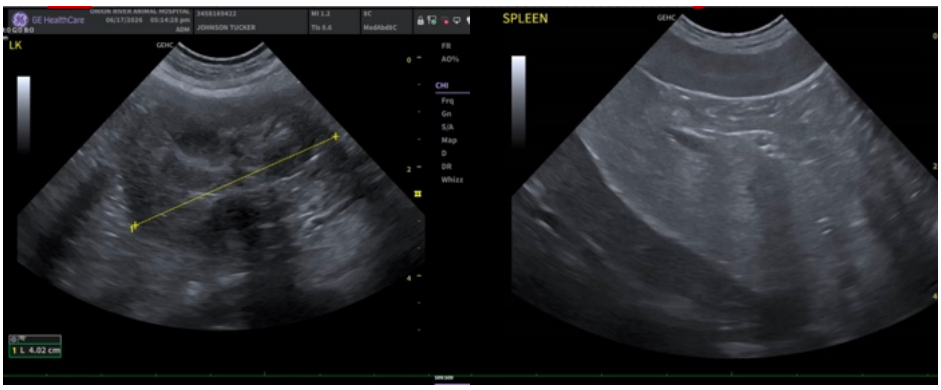

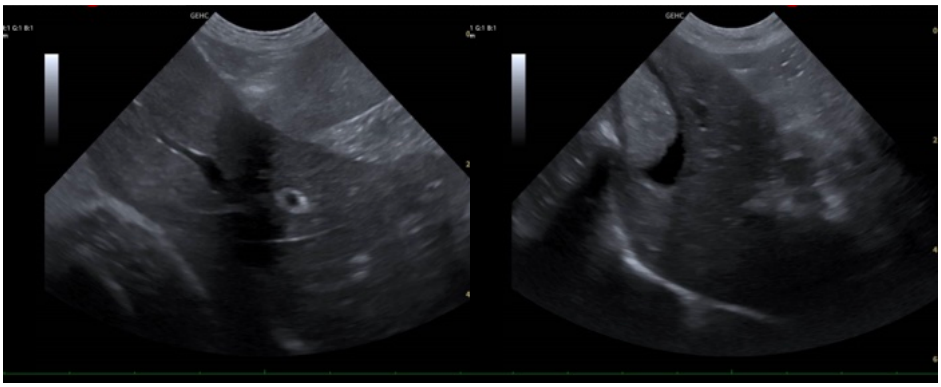

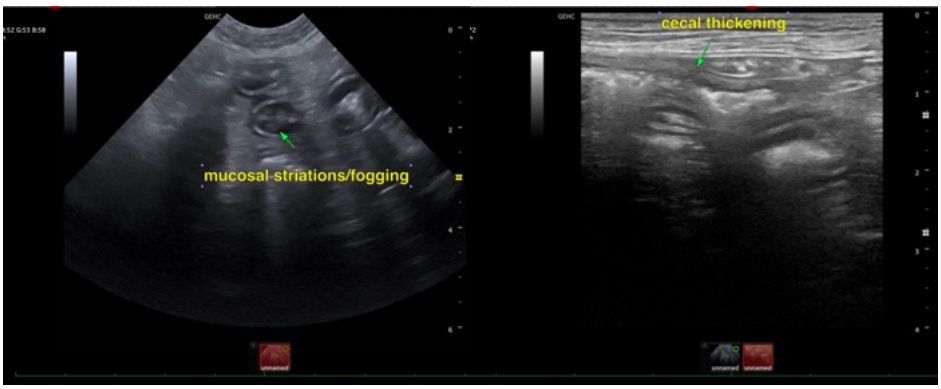

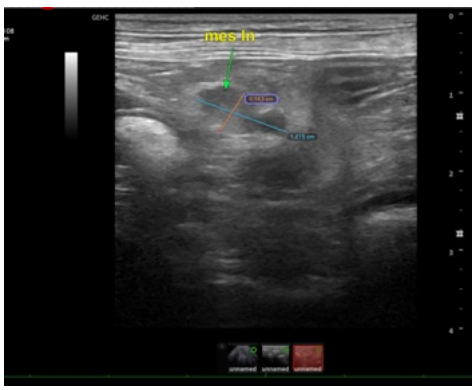

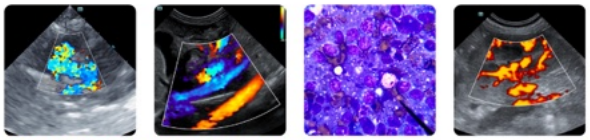

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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Eric Lindquist, DMV, DABVP (CFM), Cert. IVUSS, CEO of SonoPath.com

info@SonoPath.com

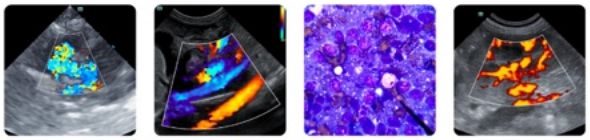
Excerpt from the Curbside Guide: https://sonopath.com/thecurbsideguide/

Protein Losing Enteropathy

DESCRIPTION Protein-losing enteropathy (PLE) is characterized by conditions or disease processes that cause protein loss through the gastrointestinal (GI) mucosa. A loss of oncotic pressure will occur when albumin levels drop below 1.5 g/dL resulting in ascites, pleural effusion, and peripheral edema. Causes of PLE may include primary lymphangiectasia, inflammatory bowel disease, dietary hypersensitivity, ulcerative disease, granulomatous disease (fungal disease), immunoproliferative enteropathy, and neoplasia (lymphoma being most common), with the latter list of conditions possibly resulting in secondary lymphangiectasia as they cause dilation of the lymphatic ducts. Intussusception and parasitic infection can result in PLE in young animals. Primary lymphangiectasia is a congenital disease typically found in young dogs, especially Basenjis and Norwegian Lundehunds. Some breeds, such as Wheaten Terriers, Rottweilers, German Shepherds, Norwegian Lundehunds, Yorkshire Terriers, and Basenjis, are more predisposed to PLE than others. Heritability has been demonstrated in Wheaten Terriers and Basenjis. Yorkshire Terriers are ten times more likely to develop IBD and nine times more likely to suffer hypocalcemia and hypomagnesemia with IBD.

CLINICAL SIGNS Canine patients are typically the most susceptible to PLE (cats are less commonly affected) and will often display anorexia, weight loss, vomiting, and diarrhea. Interestingly, some patients may present with pleural or peritoneal effusion secondary to severe hypoalbuminemia, but they may not exhibit primary signs of gastrointestinal disease, such as diarrhea or vomiting. Ascites and/or pleural effusion or subcutaneous edema can occur subsequent to hypoalbuminemia (albumin < 1.5 g/dL). Signs of thromboembolic disease, such as dyspnea due to pulmonary thromboembolism, can occur secondary to a lack of antithrombin III (AT III).

DIAGNOSIS Typical laboratory abnormalities include hypoalbuminemia and/or hypoglobulinemia. If globulin levels are within normal limits, they are usually at the lower end of normal or may be “relatively” elevated owing to over production of globulins which result in normal range. Lymphocytes and cholesterol may be decreased, especially in cases of lymphangiectasia, due to a loss of lymphocytes and cholesterol in the lymph. A regenerative anemia can occur due to blood loss, although anemia due to iron deficiency may ensue in chronic cases. Hypocalcemia may transpire secondary to albumin loss (pseudohypocalcemia) or the calcium can be truly subnormal as a result of hypovitaminosis D due to PLE. Hypomagnesemia is common as well. Severe PLE can lead to a decline in AT III levels which can then result in a



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prothrombotic state. Thus, AT III levels should be measured in severely hypoalbuminemic patients.

Ultrasound is a non-invasive method to help determine the cause of hypoalbuminemia as it can be utilized to evaluate the GI tract, kidneys, liver, and adrenals. It will also help identify the potential sources of albumin loss (GI or renal), whether there is a lack of albumin production (liver), or if the condition is linked to hypoadrenocorticism, which also may be associated with hypoalbuminemia (presence of isoechoic flattened adrenals < 0.32 cm). These findings should also be considered in combination with a bile acid test to rule out hepatic insufficiency, a urine protein:creatinine (UPC) ratio to assess for urine protein loss, and a fecal α1-proteinase inhibitor test to assess for GI protein loss. Basal cortisol and/or an ACTH stimulation test may be indicated if hypoadrenocorticism is clinically suspected.

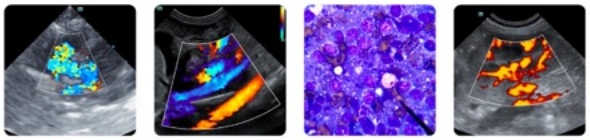
An intestinal panel assessing infectious and parasitic disease, TLI, folate, and cobalamin is recommended as part of the assessment.

Sonographic abnormalities may include thickening of the intestinal wall and mucosal striations. One study has shown that the presence of mucosal striations has a sensitivity of 75% and specificity of 96% in dogs that have PLE; however, mucosal stippling appears to be a nonspecific finding. Administration of corn oil (0.5–1 mL/kg) one hour prior to the ultrasound will enhance the visibility of mucosal striations in the small intestine during the sonogram. Solitary masses or focal intestinal thickening and lymphadenopathy can be evaluated, and sometimes fine-needle aspiration (FNA) of a mass or enlarged lymph node may yield a diagnosis, especially in cases of lymphoma. If the results are inconclusive, then surgical biopsy ideally should be guided by an intraoperative ultrasound, especially if the lesions are focal. An ultrasound-guided core biopsy would only be considered if a bowel mass was large enough to biopsy the tissue without sampling through to the lumen, which could result in the leakage of bowel contents and subsequent peritonitis.

A definitive diagnosis of PLE can only be obtained via histopathology. Preferably, this is achieved with a surgically obtained full-thickness biopsy or an endoscopic-guided biopsy performed the morning after the patient has eaten a high-fat meal so that the lacteals are dilated and lymphangiectasia can be adequately diagnosed. There may be some increased risk to obtaining full-thickness biopsies in patients with severe hypoalbuminemia due to decreased healing and increased risk of dehiscence. Thus, the cost-benefit of full-thickness biopsy versus an endoscopic biopsy should be considered on a case-by-case basis.

Endoscopy should be performed using two approaches: one via the stomach to biopsy the duodenum, and one via the colon to biopsy the ileum. These two approaches thereby maximize the information one can yield from biopsy. Yet, transmural disease, such as lymphoma affecting the muscularis and submucosa, is not typically assessed very readily via endoscopy. A sonogram to "map" the GI tract can help determine whether the pathology is luminal—and therefore available for sampling through endoscopy—or mural or serosal, therefore necessitating surgical biopsy.

TREATMENT Therapy for PLE is dependent on the underlying disease process. Given that a significant fraction of PLE cases is the result of a food allergy causing IBD, regardless of whether lymphangiectasia is concurrent, dietary trials with either hypoallergenic or a hydrolyzed protein/novel protein diet are a good choice, especially if IBD has been confirmed on biopsy. However, if severe lymphangiectasia has been diagnosed, a fat-restricted diet is preferred. In some cases, a specially formulated homemade diet may be most appropriate and should be determined in consultation with a veterinary nutritionist.



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Empirical broad-spectrum deworming should be pursued using fenbendazole at 50 mg/kg PO Q24hr for five days; repeat in two weeks.

If IBD has been confirmed, immunosuppressive therapy with prednisone should be administered at 2 mg/kg/day for a 2–4 week induction period. Subsequently, the patient should be weaned slowly to 1 mg/kg/day, and eventually dosed every other day. In large and giant breed dogs, dosing per body surface area is recommended to avoid overdosing and the precipitation of severe side effects; the recommended dose is 30–40 mg/m2 for large breed dogs. Concurrently administer azathioprine (2 mg/kg PO Q24hr for 10 days, then 1 mg/kg PO Q24hr, and eventually every other day on alternate days to the prednisone; note that alternative protocols exist at a dose of 1–2 mg/kg PO Q24hr and can be considered if the patient is nonresponsive to prednisone alone). Cyclosporine is an alternative immunosuppressant; however, it can be quite expensive, especially in large dog breeds, and should be dosed at 3–5 mg/kg PO Q12–24hr to start.

In the presence of effusions, colloid therapy may be beneficial and can include hetastarch at 10–20 mL/kg, which can be given as an initial bolus and the rest over 4–6 hours, or, alternatively, over a 24-hour period as a CRI (1–2 mL/kg/hr, not to exceed 20 mL/kg/24hr). Fresh frozen plasma is typically ineffective at raising albumin levels; however, in an emergency situation, one can give it at 10–20 mL/kg IV over 3–4 hours. Human albumin is more effective at raising serum albumin levels; it also helps provide oncotic support during diagnostic procedures, such as obtaining biopsies, for example. Repeat administration can result in anaphylactic reactions, but that outcome is rare.

Diuretics can be utilized in the face of severe ascites, but they are not particularly effective. Spironolactone is preferred (2 mg/kg PO Q12hr) and low-dose Lasix can be added if necessary (1–2 mg/kg PO Q12hr). Abdominocentesis should only be pursued if the patient is experiencing discomfort due to exaggerated abdominal distention. Excessive drainage will cause further depletion of the protein supply which runs counter to restoring balanced protein levels and can also often result in rapid fluid shifts; this leads to acute hypovolemia and hypotension. Anticoagulant therapy is suggested in the face of severe hypoalbuminemia (less than 1.5 g/dL).

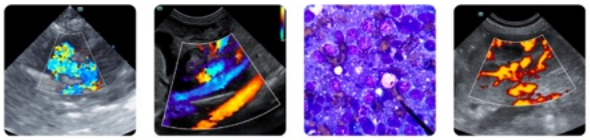
Therapeutic options include clopidogrel (2 mg/kg PO Q24hr) or aspirin (1 mg/kg PO Q24hr) in the hopes of preventing a potential thromboembolic episode which can be the source of sudden death in cases of significant hypoalbuminemia in which there has been AT III loss.

Patients should be supplemented with cobalamin at 25–50 µg/kg once per week for 4–6 weeks, then once every other week to once a month as needed.

If ionized calcium levels are decreased with corresponding clinical signs of hypocalcemia, calcium levels should be corrected with parenteral calcium gluconate (50–150 mg/kg IV over 12–24hrs). Long-term supplementation may be necessary for dogs suffering from concurrent hypovitaminosis D, secondary to IBD; this would entail administering calcitriol as well as oral calcium (calcium carbonate). In the face of hypomagnesemia, magnesium sulphate (1 mEq/kg/day IV) or magnesium oxide (milk of magnesia 10–20 mg/kg PO Q12hr) may be utilized for magnesium supplementation; however, the latter may cause diarrhea.

CONCLUSION PLE can be a challenging syndrome to treat given the multiple possible underlying etiologies and the severity of clinical sequelae characteristic

of severe hypoalbuminemia. If possible, it is important to obtain a definitive diagnosis, and addressing all potential comorbid issues is crucial to the success of its management. Dietary therapy is an important factor in long-term treatment, as is attending to the underlying cause of the disease.



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