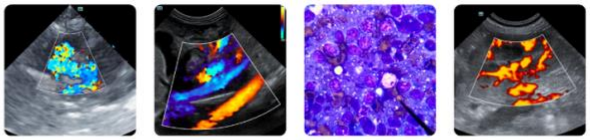

PATIENT

Flex Gonzalez

SPECIES

Canine

BREED

Toy Fox Smooth Terrier

SEX

Neutered Male

AGE

13 Years 1 Month

WEIGHT

11 pounds

INTERPRETED BY

R. McKenzie Daniel, DVM, DABVP

IMAGING PERFORMED BY

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PRESENTING CLINICAL SIGNS

FLEX presented today for possible episode of pancreatitis. O stated that P had an episode like this back in April. Stated that P would tense up and freeze in a hunch position that looked like a seizure, but it was rolled out that it was pancreatitis. Since then, P has been on RCVD GI LF diet and O stated that the P will go from having normal to soft stools. The start softer stool started yesterday with the painful episodes. O says there are no blood or mucus in the stool and that its lighter in color than normal. Recently hospitalized for pancreatitis and hematochezia. Did well overnight. NG tube place and began receiving Recovery diet slurry 1/4 RER/day. No vomiting noted. Hematochezia last noted at 9PM.

Abnormal PE/Chem/CBC/UA Results: Grade II/VI heart murmur PCV/TS: 43%/4.8g/dL iSTAT Chem 8+: Glu 92, BUN 17, Creat 0.8, Na 150, K 4.0, Cl 114, iCa 1.19 Chem 10: TP 3.2 (L), Alb 1.3 (L), Glob 1.9; rest NSF A: Pancreatitis - r/o acute vs chronic Hematochezia Panhypoproteinemia - r/o PLE vs SIRS vs endocrinopathy vs hepatopathy vs neoplasia Moderate dehydration Mild leukocytosis characterized by neutrophilia Last fed between 2am -6 am - NG tube slurry Fresh frozen plasma 10mL/kg over 4 hours LRS 60mL/kg/d Cerenia 1mg/kg IV q24h Unasyn 30mg/kg IV q8h Gabapentin 15mg/kg PO q8h DiaGel Small Dog PO Feed 1/4 RER via NGT (1/2 can Recovery blended with water 1:2 - 15mL/hr CRI)

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder, trigone, cystourethral junction, and visible pelvic urethra 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 change were noted.

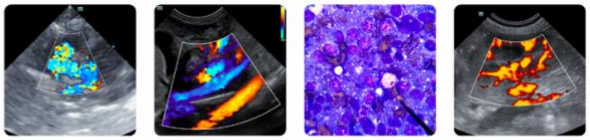
Normal size and margination was present in the kidneys. A normal 1:3 cortex / medulla ratio was maintained. The medulla and cortices were uniform in texture with some increased echogenicity and loss of corticomedullary symmetry and definition expected for the age of the patient. Mild medullary mineral was present. The left kidney measured 3.5 cm in length. The right kidney measured 3.9 cm in length.

Adrenal Glands

The left adrenal gland presented mildly enlarged at the caudal pole while the right adrenal gland was normal in size. Mild parenchyma heterogeneity and mild capsule asymmetry was present without suspicion for overt neoplasia. The left adrenal gland measured 0.78 cm width in the caudal pole. The right adrenal gland measured 0.56 cm width in the caudal pole.

Spleen

The spleen exhibited primarily finely textured and homogenous parenchyma which was hyperechoic to the liver and renal cortical parenchyma. Small perihilar hyperechoic nodule to nodules were present throughout the cranial to caudal parenchyma with an example measuring 0.26 cm in diameter. 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 or neoplastic changes were not noted. The hyperechoic nodules tend to trend benign and are most consistent with benign hyperplasia or myelolipomas.



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Liver

The liver presented with subjective mild hepatomegaly. 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. The gallbladder wall was thickened in appearance consisting of an echogenic double rim corresponding to the inner and outer portions of the wall. This is consistent with gallbladder wall edema. Possible causes may include acute inflammation, edema and anaphylaxis. The gallbladder contained mild nonorganized gallbladder debris. The common bile duct was not visualized.

Gastrointestinal

The stomach presented intact wall layering with a normal wall layer ratio. The lumen of the stomach contained echogenic, nonshadowing ingesta without signs of obstruction or foreign material.

The small intestine presented intact prominent wall exhibiting propensity for prominent to mildly thickened mucosa. Segmental to generalized mild hyperechoic intestinal mucosal speckling to mucosal fogging. Small intestine wall measured 0.40 cm wall width.

Normal visible colon wall layers were present. The colon exhibited generalized mild distention containing soft fecal matter.

Pancreas

The pancreas was mildly prominent in size with asymmetrical contour and heterogeneous parenchyma compared to adjacent hyperechoic omentum. No signs of active inflammation or neoplasia.

Free Abdomen

Generalized mild omental hyperechogenicity with mild volume of peritoneal effusion present.

Intermittent mesenteric lymph nodes were present. The lymph nodes were essentially isoechoic to adjacent omentum without evidence of peripheral inflammation and maintaining a normal width: length ratio (<0.5).

ULTRASONOGRAPHIC FINDINGS

Primary Findings

- PLE intestinal pattern.
- Mild generalized descending colon containing soft fecal matter.
- Noncongested mild hepatomegaly- subjective benign.
- Mild edematous gallbladder with nonorganized bile debris (non-mucocele).
- Mildly prominent nonhomogenous pancreas- mild inflammation versus edema.
- Mild omental hyperechogenicity, intermittent mild mesenteric lymphadenopathy and mild peritoneal effusion.

Secondary Findings

- Benign splenic nodule to nodules- consistent with myelolipomas.

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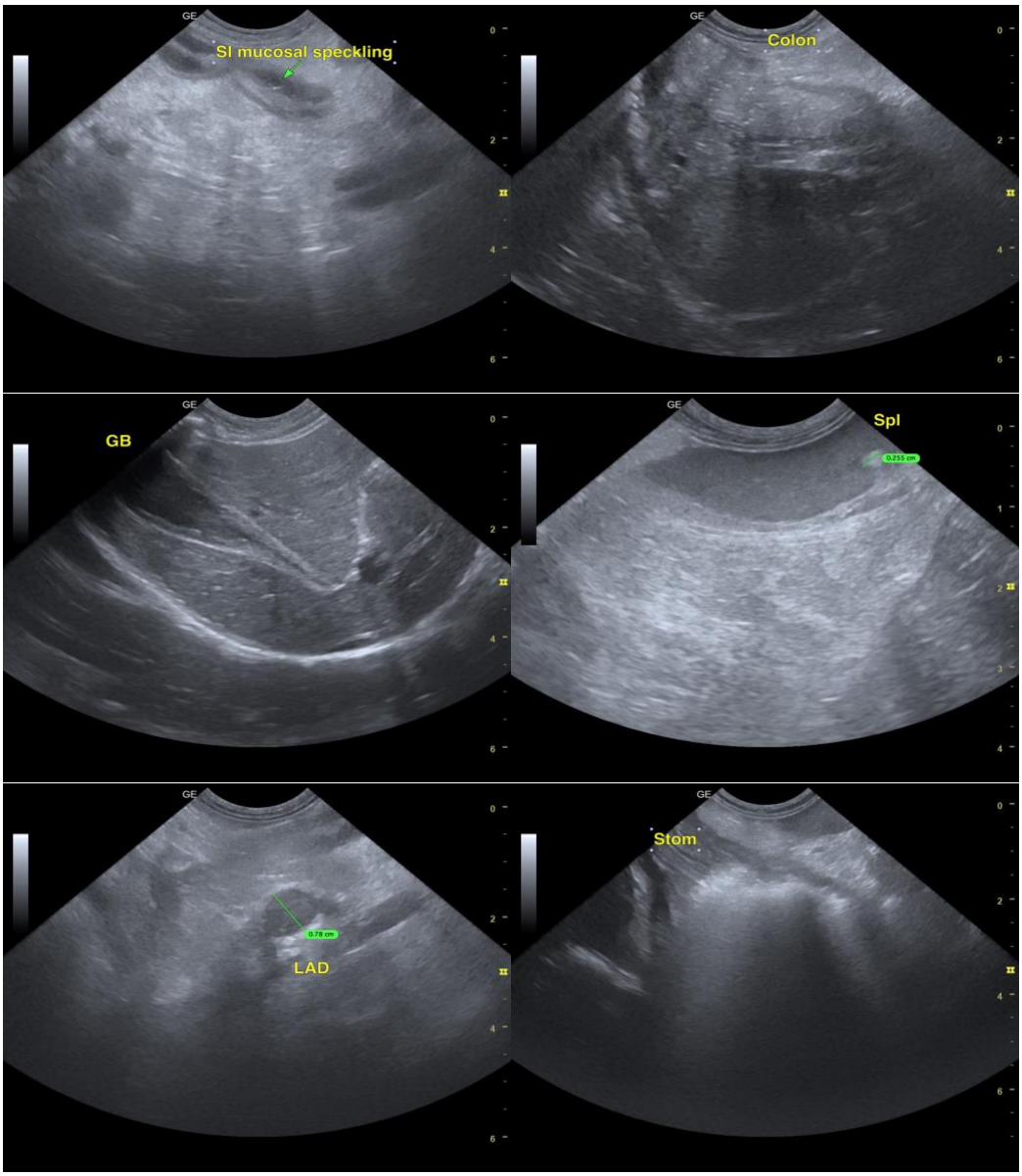
- Mild chronic renal changes exhibiting mild medullary mineral.
- Mild caudal left adrenomegaly.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

A GI panel to include PLI, TLI, cobalamin and folate is recommended. Empirical therapy for protein losing enteropathy and possible concurrent mild pancreatitis is indicated. Intestinal biopsies are required for a definitive diagnosis yet contraindicated given albumin level less than 2.0. Urinalysis is recommended if not done to assess for or rule out evidence of proteinuria. Some or all of the following protocol may be considered empirically.



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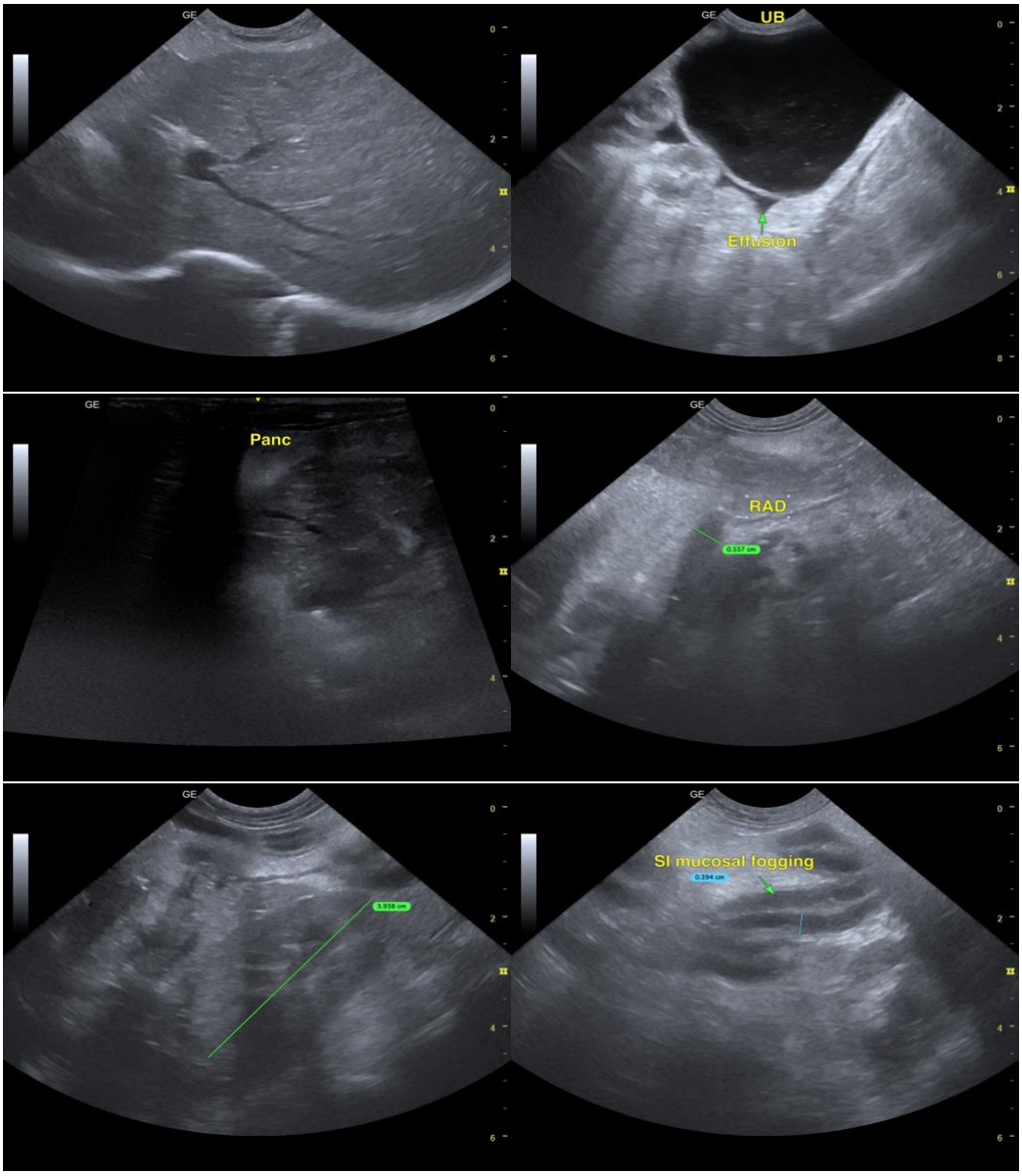


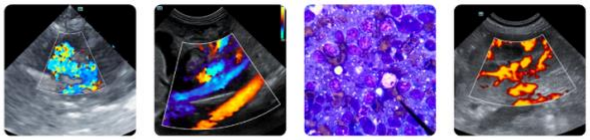
The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

R. McKenzie Daniel, DVM, DABVP (Canine / Feline Practice)

info@SonoPath.com

Protein-Losing Enteropathy (PLE)



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http://www.sonopath.com/PLE

Description: Protein-losing enteropathy (PLE) is characterized by conditions or disease processes that cause protein loss through the gastrointestinal (GI) mucosa. Clinical signs related to hypoalbuminemia will occur when albumin levels drop below 1.5 g/dl; a loss of oncotic pressure will ensue and precipitate ascites, thoracic effusion, and peripheral edema. Causes of PLE may include: inflammatory changes to the gastrointestinal mucosa or inflammatory bowel disease (IBD); food allergies resulting in IBD; ulcerative disease; granulomatous disease (fungal disease); immunoproliferative enteropathy; neoplasia (lymphoma being most common); and lymphangiectasia. Intussusception and parasitic infection can result in PLE in young animals. Lymphangiectasia typically occurs as a secondary disease process, with lymphatic duct dilation secondary to underlying inflammation or neoplastic cells. 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.

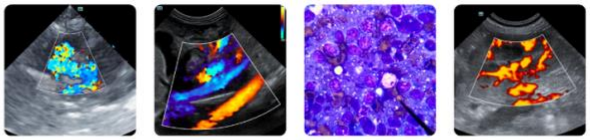
Clinical Signs: Canine patients are typically the most susceptible to PLE (cats are less commonly affected), and will often display anorexia, weight loss, vomiting, and diarrhea. Interestingly, some patients may present with pleural or peritoneal effusion secondary to severe hypoalbuminemia, but may 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. Signs of thromboembolic disease, such as dyspnea due to pulmonary thromboembolism, can occur secondary to a lack of anti-thrombin III (AT-III).

Diagnostics: Typical laboratory abnormalities include hypoalbuminemia and/or hypoglobulinemia. If globulin levels are within normal limits, they are usually at the lower end of normal. 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 prothrombotic state. Thus, AT-III levels should be measured in severely hypoalbuminemic patients.

The clinician should consider ultrasound as a non-invasive method to help determine the cause of hypoalbuminemia. Ultrasound 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 (adrenal), which may also be associated with hypoalbuminemia (the ultrasound may reveal 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 Alpha 1-Proteinase Inhibitor test to assess for GI protein loss. An ACTH stimulation test may be indicated if hypoadrenocorticism is clinically suspected.

One should measure serum TLI, folate, and B_{12} levels to evaluate for evidence of small intestinal bacteria overgrowth or to establish the presence of small intestinal disease due to cobalamin loss and elevated folate levels. The TLI will also confirm exocrine pancreatic insufficiency as a differential diagnosis for diarrhea and weight loss. A fecal exam should be submitted to rule out parasites.

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 non-specific 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 should ideally 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.



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A definitive diagnosis of PLE can only be obtained via histopathology. This is preferably 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—via the stomach to biopsy the duodenum, and via the colon to biopsy the ileum—thereby maximizing 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 of the GI tract can help determine whether the pathology is luminal and thus available for sampling through endoscopy, or mural or serosal and therefore necessitating surgical biopsy.

Treatment: Therapy for PLE is dependent on the underlying disease process. Given that a significant fraction of PLE cases are the result of a food allergy causing IBD, whether or not lymphangiectasia is concurrent, dietary trials with a hydrolyzed protein diet or a novel protein diet are a good choice, especially if IBD has been confirmed on biopsy. If, however, 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.

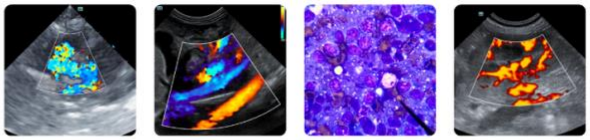
Empirical broad-spectrum deworming should be pursued using fenbendazole at 50 mg/kg PO Q24hr for 5 days; repeat in 2 weeks. Treating for small intestinal bacterial overgrowth can also be considered, especially if there is evidence of elevated folate levels. In such cases, one should consider high colony count probiotics such as Proviable.

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 30-40mg/m^2 for large breed dogs. Concurrently administering azathioprine (Immuran) (2mg/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) 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 3-5mg/kg PO Q12-24hr to start. Blood cyclosporine levels should be evaluated 7 days after initiating treatment; one can adjust the dosage at that point if need be. Concomitant use of ketoconazole (2.5-5 mg/kg PO BID) inhibits some metabolism of cyclosporine, leading to higher blood concentrations of the latter without increasing the overall dose (or cost to the owner). Typically, the dose of cyclosporine can be cut in half when dosed in conjunction with ketoconazole.

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; do not to exceed 20 ml/kg/24 hours). 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 BID) and low-dose lasix can be added if necessary (1-2 mg/kg PO BID). 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, leading to acute hypovolemia and hypotension.

Anticoagulant therapy is suggested in the face of severe hypoalbuminemia (less than 1.5 g/dl). Therapeutic options include clodiprogel (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.



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Neutered Male

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Patients should be supplemented with cobalamin (vitamin B_{12}) at 25-50 ug/kg once weekly 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 12-24 hours). 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 hypomagnesiemia, magnesium sulphate (1mEq/kg/day IV) or magnesium oxide 10-20 mg/kg PO BID (milk of magnesia) may be utilized for magnesium supplementation; however, the latter may cause diarrhea.

Conclusion: PLE can be a challenging disease syndrome to treat given the multiple possible underlying etiologies and the severity of clinical sequelae characteristic of severe hypoalbuminemia. It is important, if possible, 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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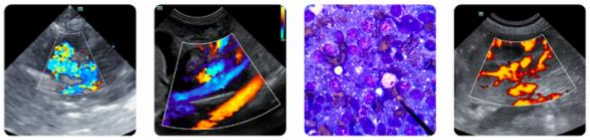

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