



PATIENT

Yoder Prine

SPECIES

Canine

BREED

Goldendoodle

SEX

Neutered Male

AGE

9 Years 9 Months

WEIGHT

84 pounds

INTERPRETED BY

R. McKenzie Daniel,
DVM, DABVP

IMAGING PERFORMED BY

Brandi Kurzowski

HOSPITAL NAME

Corfu Veterinary Clinic

REFERRING VET

Dr. Brooke Beatty

INVOICE

12807

DATE

12/23/25

PRESENTING CLINICAL SIGNS

P presented for vomiting (some flecks of blood seen in vomit) and diarrhea on 12/22, ADR for several days. When p arrived at the clinic, he had a seizure in the parking lot (HX of seizures, on zonisamide). P was started on Zenrelia in August, and it was discontinued in October after BW showed hypoalbuminemia. Recheck BW 2 weeks later showed albumin came up slightly. BW from 12/22 showed worsening hypoalbuminemia. AFAST showed distended intestinal loops, complete ileus, and scant free fluid in the abdomen. P was given dexamethasone and Cerenia and came back 12/23 for full abdominal ultrasound.

Abnormal PE/Chem/CBC/UA Results: 10/13/25 Chem- TP 4.2g/dL, ALB 1.6 g/dL, ALP 17 U/L, CHOL 107mg/dL UA- USG 1.009 T4- WNL CBC- Retic 144 k/uL 10/27/25 UA- USG 1.014 Chem- TP 5 g/dL, ALB 1.9 g/dL, ALP <10 U/L 12/22/25 Chem- TP 3.4g/dL, ALB 1.2 g/dL, Glob 2.2g/dL, ALP 10 U/L, CHOL 79mg/dL CBC- WNL

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder presented mildly distended in size with normal tone and mildly irregularly thickened ventral urinary bladder wall with mildly symmetrical ventral luminal surface contour measuring 0.77 cm ventral wall width. The thickened ventral urinary bladder appeared to extend caudally into the area of the cystourethral junction without evidence of obstruction to urine outflow. The urethra was normal in structure and tone to a depth of 4.0 cm.

The area of the residual prostate appeared normal and free of pathology.

No evidence of medial iliac or sublumbar lymphadenopathy or masses.

Normal size and margination was present in the kidneys. A normal 1:3 cortex / medulla ratio and normal corticomedullary definition were maintained. The echogenicity of the cortex was similar to or slightly less than normal liver parenchyma while the medulla echogenicity was hypoechoic to the cortex with no evidence of pelvic dilation. The left kidney measured 6.6 cm in length. The right kidney measured 7.1 cm in length.

Adrenal Glands

The left adrenal gland was indistinctly visualized without overt pathology. The left adrenal gland subjectively measured 0.78 cm width at the caudal pole.

The right adrenal gland was overtly normal in size, position and shape The right adrenal gland measured 0.69 cm width at the caudal pole.

Spleen

The spleen exhibited a finely textured and homogenous parenchyma which was hyperechoic to the liver and renal cortical parenchyma. The capsule was smooth and regular without apparent expansion.

The splenic vasculature at the hilus was normal in volume with no evidence of congestion or thrombosis. Acute to chronic inflammatory, neoplastic, or benign parenchyma changes were not noted.

Liver



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The liver was subjectively normal in size, structure, and contour. The liver parenchyma was uniform and hypoechoic to the spleen with a mild coarse echotexture. The hepatic and portal vasculature were subjectively normal in appearance without overt signs of congestion.

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The gallbladder was non distended in size with mild nonorganized biliary sludge. The cystic duct and common bile ducts were normal without evidence of dilation.

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Gastrointestinal

The stomach presented with mildly thickened wall exhibiting indistinct gastric mural detail. Empty lumen with mild lumen gas. Gastric body wall measured 0.74 cm wall width.

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The intestinal walls demonstrated intact thickened wall layering exhibiting intact to indistinct mural detail and increased overall intestinal mural echogenicity. Segmental mild ileus and segmental gas without evidence of mechanical obstructive pattern to the level of the colon. Small intestine wall measured 0.53 cm wall width.

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The colon walls presented intact yet prominent wall layering with mild thickened to echogenic submucosa. The colon contained generalized nonformed fecal matter consistent with patient's history.

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Pancreas

The area of the pancreas was without overt pathology with indistinct pancreatic visualization owing to peripancreatic increased omental artifact.

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Free Abdomen

Generalized mild omental hyperechogenicity and minor pockets of peri-intestinal to peritoneal free fluid was present.

ULTRASONOGRAPHIC FINDINGS

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Brandi Kurzowski

- Thickened empty stomach.
- Enterocolonopathy exhibiting increased intestinal mural echogenicity and nonformed fecal matter in colon.
- Normal volume liver.
- Omental hyperechogenicity and minor peri-intestinal/peritoneal free fluid.
- Mildly thickened ventral urinary bladder wall.

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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

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Dr. Brooke Beatty

In conjunction with hypoalbuminemia, PLE intestinal criteria is suspected with considerations including IBD or other inflammatory disease, lymphangiectasia, while potential for gastroenterocolic neoplasia is not excluded. A GI panel to include PLI, TLI, cobalamin and folate is recommended. Gastroenterocolic biopsy is required for a definitive diagnosis yet contraindicated if albumin level is 2.0 or less. Some or all of the following protocol may be considered empirically. Empirical therapy for protein losing enteropathy with clinical monitoring may prove beneficial.

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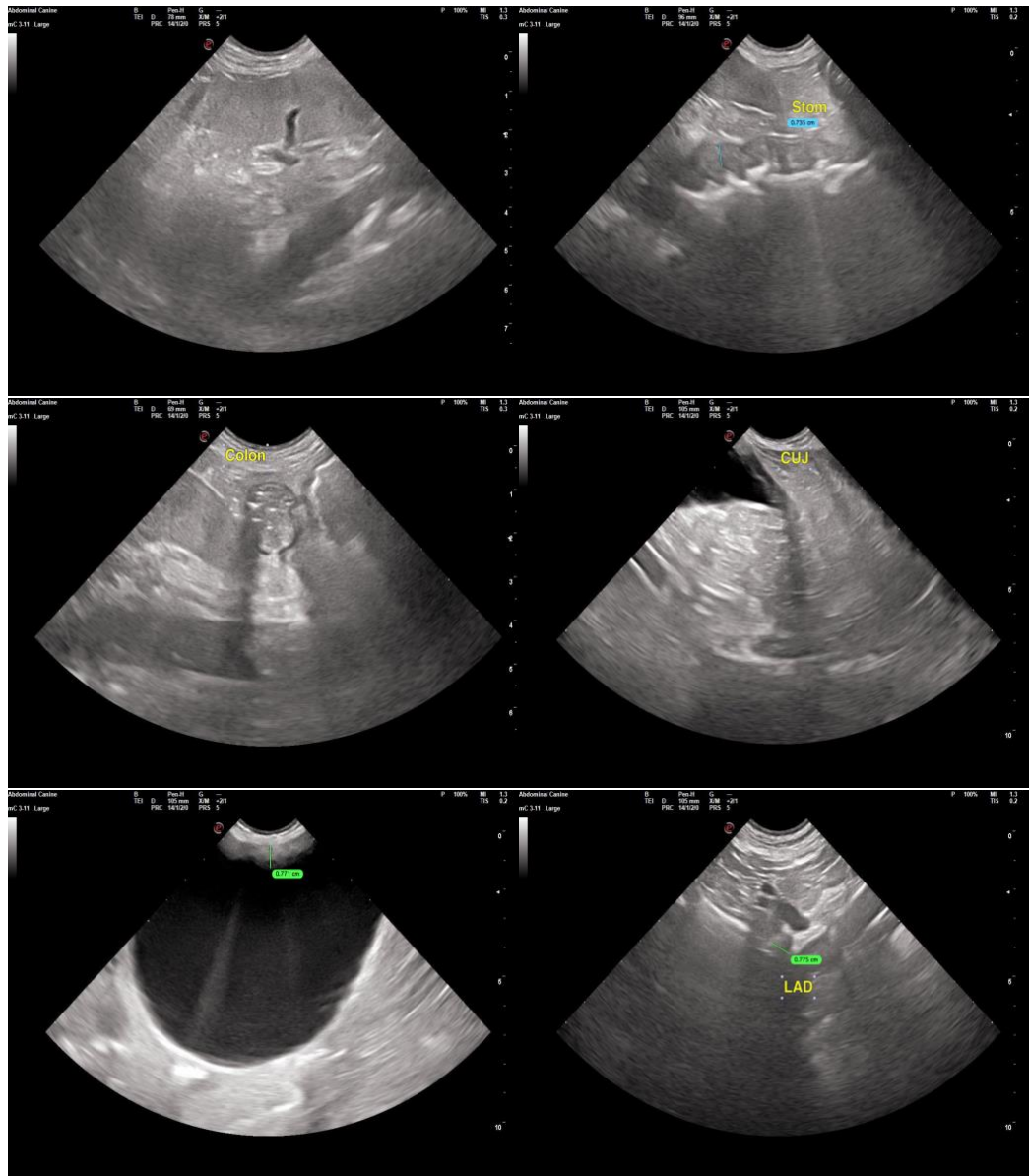
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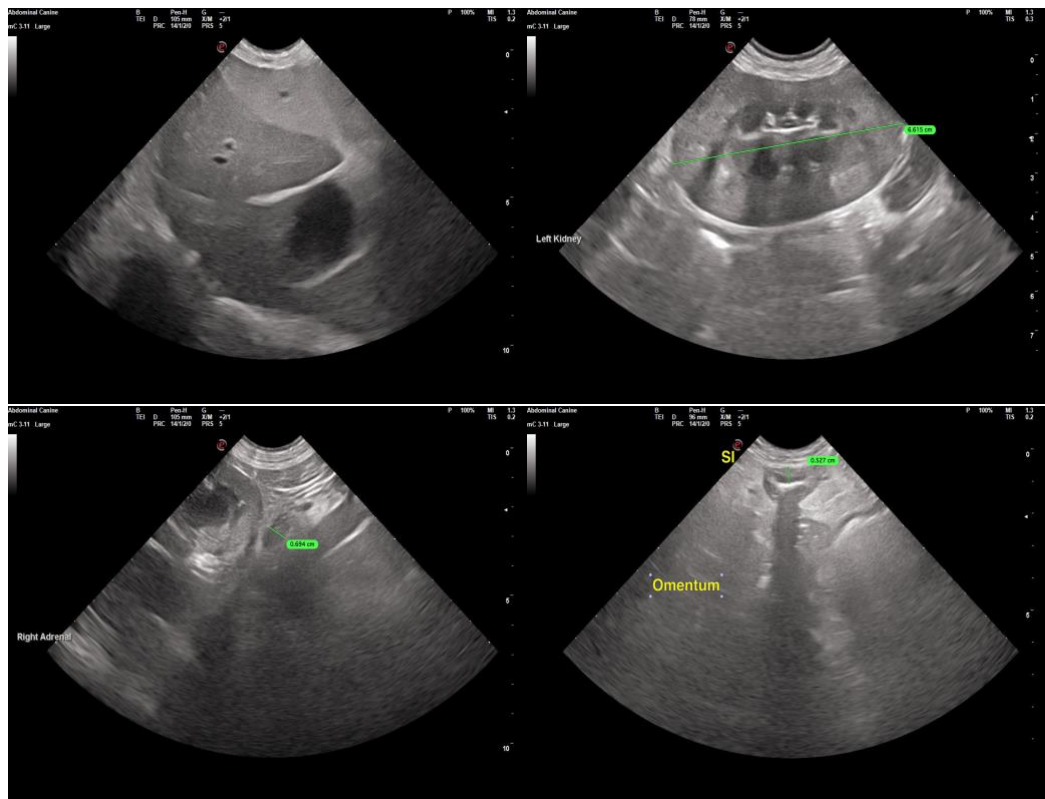
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

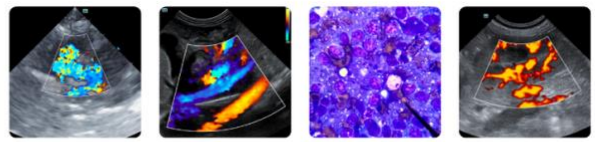
R. McKenzie Daniel, DVM, DABVP (Canine / Feline Practice)

info@SonoPath.com

Protein-Losing Enteropathy (PLE)

<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



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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.

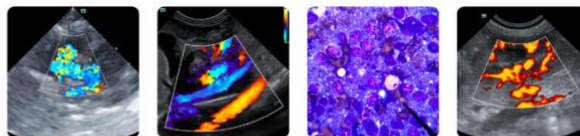
The clinician should consider ultrasound as a non-invasive method to help determine the cause of hypoalbuminemia. Ultrasound can be utilized to evaluate the GI tract, kidneys, liver, and adrenals. It will also help identify the potential sources of albumin loss (GI or renal), whether there is a lack 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 B₁₂ 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.

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.



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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. High colony count probiotics such as Provable are recommended.

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/m² for large breed dogs. Concurrently administering azathioprine (Immunan) (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.

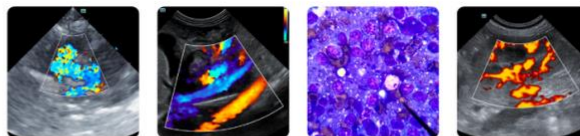
Diuretics can be utilized in the face of severe ascites, but they are not particularly effective. Spironolactone is preferred (2 mg/kg PO BID) and low-dose lasix can be added if necessary (1-2 mg/kg PO BID). Abdominocentesis should only be pursued if the patient is experiencing discomfort due to exaggerated 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.

Patients should be supplemented with cobalamin (vitamin B₁₂) 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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References:

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