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PLE: Protein Losing Enteropathy

Anorexia, weight loss, vomiting, diarrhea are often associated with protein losing enteropathy mainly in canine patients. In light of these clinical signs combined with subnormal albumin levels, the clinician should consider ultrasound to evaluate the big 4 (GI, Renal, Liver, Adrenals) to identify the potential sources of albumin loss (GI/Renal) or lack of production (Liver) or linked to hypoadrenocorticism (Adrenal) that may also be associated with this clinical profile (isoechoic flattened adrenals < 0.4 cm study under way at SonoPath.com).

Typical diagnostics for protein losing enteropathy (PLE) reveal include a sonogram and clinical profile of hypoalbuminemia (but may also be within normal serum levels), suggestive intestinal wall prominence and mucosal striations noted on the sonogram, and clinical signs of vomiting, weight loss, diarrhea, or lack of GI signs altogether and potentially weight loss alone. **PLE** is preferably definitively diagnosed by full thickness or endoscopy guided biopsies after feeding a fatty meal the night before in order to dilate lacteals to adequately diagnose lymphangiectasia. PLE can be associated with:

1. Inflammatory bowel disease (IBD)
2. Granulomatous disease
3. Neoplasia
4. Immunoproliferative enteropathy

Often GI blood loss may occur (anemia +/- azotemia) potentially caused by lymphosarcoma, ulcerative disease, or intussusception. A dynamic sonogram testing procedure feeding corn oil (0.5-1 cc/kg) 45 minutes prior to the sonogram of an NPO patient will enhance the presence of mucosal striations in the small intestine during the sonogram. This is a reliable test founded by Marks et al. at UC Davis (NAVC 2011).
Endoscopy both anterior and posterior approaches are ideal in order to access the stomach/duodenum in the former case and colon/ileum in the latter in order to maximize the biopsy information. However, this will not detect transmural disease such as lymphoma affecting the muscularis and submucosa that are not typically obtained readily via endoscopy. Ultrasound evaluation of the GI tract can help decide whether the pathology is luminal, and available for sampling through endoscopy, or mural and necessitating US-guided FNA or core biopsy or surgical biopsy ideally guided by intra-operative ultrasound. More information regard intra-operative ultrasound may be found in "resources" at www.sonopath.com.

B12, Folate, Ionized calcium, ionized magnesium and antithrombin levels should all be measured in the clinical PLE patient and levels should be corrected with Calcium gluconate (50-150 mg/kg Iv over 12-24 hours) or 1 tablet extra strength Tums (300 mg calcium) PO tid>bid>sid as needed to maintain normal calcium levels over 3-4 weeks. Mg sulphate (1mEq/kg/day IV) or MgOxide 10-20 mg/kg PO BID (Milk of magnesia) may be utilized for magnesium supplementation but milk of magnesia may cause diarrhea (ironically).
Yorkshire Terriers are 10X more likely to develop IBD and 9X more likely to suffer hypocalcemia and hypomagnesemia with IBD. Therefore magnesium and calcium supplementation may be in order.
Other differentials for Hypoalbuminemia include:

1. PLE
2. Liver failure
3. Glomerulonephritis/amyloidosis/protein losing nephropathy (PLN) (typically with elevated serum globulins)
4. Vasculitis
5. Exocrine pancreatic insufficiency (EPI)
6. Addison's disease (typical or atypical).

MettaSound
Veterinary Imaging

Robyn Roberts RDMS
Appointments: 512-585-6698
Fax: 512-686-3149
Email: scheduling@mettasound.com

Hypocalcemiamay be present owing to albumen loss (carrier proteins). If depressed cholesterol is also present with hypoalbuminemia then either liver failure or PLE should be suspected. Hypocalcemia may also be an issue with PLE and general causes of Hypoalbuminemia.
Hypocalcemia differentials include:

1. Hypoalbuminemia
2. Hypovitaminosis D
3. Hypomagnesemia
4. Hypoparathyroidism
5. Pancreatitis
6. Spurious/idiopathic

Given that a significant (not a majority) fraction of PLE cases is caused by a food allergy--> IBD--->lymphangiectasia, leading to PLE. I suggest, as does my colleague Mark Taylor DVM (Resident nutritionist), **Purina HA** is low (not restricted in fat) in fat; low enough to use for lymphangiectasia cases. Plus, it is hydrolyzed, meaning there is nothing in which the body can respond antigenically. No other hydrolyzed or novel protein diets are as low in fat. (Z/D RC HP, and IVD diets are actually pretty high in fat).
It is a good idea to rule out parasitism and/or utilizing empirical treatment with **Fenbendazole** 50 mg/kg SID for 5 days & repeat in 2 weeks combined with **Metronidazole** 15mg/kg BID for 10 days. Also ruling out neoplasia with FNA or biopsy, if a target is available or full thickness may be performed. Occasionally GI lymphoma or mast cell disease may be emerging in these cases.
Feeding normal levels of fat with lymphangiectasia patients causes further dilation of the lacteals with rupture, exacerbating protein losses. Many recommend a low fat diet with lymphangiectasia, however, if the primary cause was a food allergy, this recommendation could be counterproductive.

I recommend Purina HA for a few weeks and monitor total protein and albumin. If the levels are still low, I would transition to a novel protein diet that is ultra restricted in fat (ostrich, kangaroo, crab, or something). Because none exist commercially, the diet would have to be homemade, expensive for the owner to make, but balanced. Home made diet with a base of low fat cottage cheese or chicken breast may also be suggested through a nutritional consultation.

Low dose **lasix** (1-2 mg/kg BID) and/or **spironolactone** (2-4 mg/kg BID) may be utilized for ascites until oncotic pressures are restored with colloid, or even better, plasma therapy. Abdominocentesis should be utilized only to keep the patient comfortable owing to excessive abdominal distention. Excessive drainage will further deplete the protein supply that we are trying to restore.
B12supplementation of 250-1,000 ug (cat/small dog lower end, Large breed higher end dose or 1/4cc-1cc) SC weekly x 6 weeks would be recommended owing to loss of B12 in the ileum regardless of serum levels.

Aspirintherapy is suggested (1 mg/kg sid) to assist in potential thromboembolic episodes that are the often the source of sudden death in these cases owing to antithrombin III loss. This dose will not cause side effects in GI tract or elsewhere.
Azothiaprine for cyclosporine may also be considered for refractory cases.

In a last ditch effort if this doesn't work and biopsies are still not possible then a prednisone trial would be in order but I always prefer utilizing cortisones in these patients based on biopsy results but realize that this is not feasible in many cases.
References:
ACVIM 2006-2010
NAVC 2011

Eric C. Lindquist DMV (Italy)
DABVP (K9 & Feline Practice)
CERT. IVUSS, Founder of SonoPath.com

For mobile appointments in the Austin, TX region contact:

Robyn Roberts RDMS
Appointments: 512-585-6698
Fax: 512-686-3149
Email: scheduling@mettasound.com

This Communication has Been Fueled By

SonoPath LLC. 31 Maple Tree Ln. Sparta, NJ 07871 USA
Via Costagrande 46, MontePorzio Catone (Roma) 00040 Italy
Tel: 800 838-4268
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