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VCA MidWest Veterinary Referral & Emergency Center

A CASE OF CHRONIC DIARRHEA IN A DOG

PATIENT HISTORY

SIGNALMENT:

Mooshi, 2.5 yr FS American Bulldog (32 kg)

PRESENTING CONCERN:

2-month duration of weight loss and small bowel diarrhea

PERTINENT HISTORY:

Mooshi's history included:

- Small bowel diarrhea of 3 month duration approximately 1 year prior to the current presentation that was minimally responsive to metronidazole therapy and dietary manipulation.
- Her current diet is novel protein (venison) formula with occasional apple slices for training treats.
- Mooshi has always lived in eastern Nebraska, has never travelled out of Nebraska/Iowa, and received RV, DA2PP/CV, and bordatella vaccinations 6 months prior to presentation.

PHYSICAL EXAMINATION

On examination, Mooshi was bright, alert, responsive, well-hydrated, and afebrile. Her body condition score (BCS) was 2/9 and generalized cachexia was noted. Rectal examination identified soft, light brown feces. After rectal examination, the patient was observed to defecate a large volume of malodorous, similarly colored feces.

INITIAL DIAGNOSTIC EVALUATION

THE INITIAL DIAGNOSTIC PLAN INCLUDED:

- *Complete blood count (CBC), serum biochemical profile, and urinalysis*
- *Fecal flotation by zinc sulfate sedimentation to evaluate for ova and parasites*

RESULTS

The CBC identified an absolute mature neutrophil count of 15,265 (reference range: 2060-10600) and an absolute monocyte count of 1011 (reference range: 0-840). Serum biochemical profile documented an alanine aminotransferase (ALT) of 132 U/L (reference range: 12-118 U/L) and asparagine aminotransferase (AST) of 88 U/L (reference range: 15-66 U/L). Urinalysis, Giardia ELISA, and fecal flotation were all negative.

Given the patient's history, signalment, and cumulative test results, differential diagnoses for his weight loss and chronic small bowel diarrhea included:

- Intestinal parasitism (i.e.: giardiasis, hookworms, roundworms)
- Inflammatory gastrointestinal disease (inflammatory bowel disease)
- Lymphangiectasia
- Small intestinal bacterial overgrowth
- Histoplasmosis
- Stagnant loop syndrome
- Occult gastrointestinal parasitism
- Neoplasia
- Exocrine pancreatic insufficiency (EPI)
- Highly digestible diet responsive

UPDATED DIAGNOSTIC PLAN

The updated diagnostic plan included:

- *Abdominal ultrasound (AUS) examination to look for any changes in the intra-abdominal organs, particularly the gastrointestinal tract, liver, and pancreas*
- *Fasting trypsin-like immunoreactivity assay to screen for EPI*
- *Fasting cobalamin and folate levels to screen for deficiencies and/or hypersyntheses*

UPDATED RESULTS

The AUS examination identified no architectural abnormalities. Fasting TLI was 1.2 ug/dL (reference range: 5.7-45.2 ug/L) and the cobalamin level was 175 ng/L (reference range: 251-908 ng/L). The serum folate level was normal.

DIAGNOSIS

Exocrine Pancreatic Insufficiency (EPI)

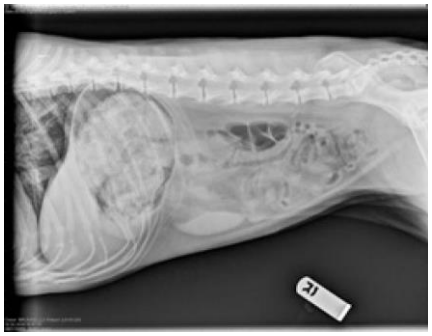
TREATMENT

Mooshi was prescribed a powdered pancreatic extract (Pancreazyme; 2 teaspoons per 20 kg body weight), parenteral cobalamin (1000 mcg SQ q7 days), and vitamin E (10-15 IU/kg PO q24 hr). At a 2-week recheck examination, her weight had slightly increased, her appetite had normalized, and her fecal consistency was markedly improved.

DISCUSSION

Exocrine pancreatic insufficiency (EPI) is caused by insufficient synthesis and secretion of exocrine pancreatic digestive enzymes.[1] Subsequently there is insufficient digestive enzyme activity in the lumen of the small intestine. Pancreatic acinar atrophy (PAA) is the most common cause of EPI in dogs. Canine PAA occurs secondary to subclinical immune-mediated pancreatitis in German Shepherd Dogs and Border Collies.[2,3]

Clinical signs of EPI develop following loss of approximately 90% of the exocrine pancreas. Nutrient malabsorption may lead to both to protein-calorie malnutrition and vitamin deficiencies. Serum cobalamin concentrations are markedly decreased in many dogs with EPI, and serum folate concentrations are often increased, suggesting concurrent small intestinal bacterial overgrowth (SIBO).[4] Vitamin K deficiency and a resultant coagulopathy may develop rarely.[5] In patients with EPI caused by chronic pancreatitis, pancreatic tissue destruction may not be limited to the acinar cells, and concurrent diabetes mellitus may be present.



Clinical signs most commonly reported in dogs with exocrine pancreatic insufficiency are polyphagia, weight loss and diarrhea. Feces from dogs with EPI are commonly pale, loose, voluminous and malodorous. However, dietary modification may mask these expected fecal changes.1 Results of routine blood/urine tests are within the normal range in most cases, although lymphopenia, lymphocytosis, neutrophilia, eosinophilia and elevations of hepatic enzymes may be seen. [6,7] Abdominal radiography and ultrasonography are frequently unremarkable. The serum trypsin-like immunoreactivity (TLI) is recognized as the most sensitive and specific non-invasive screening assay for EPI. Serum TLI concentrations are markedly subnormal in affected patients.[8]

Most dogs with EPI may be successfully managed by supplementing each meal with pancreatic enzymes present in commercially available dried pancreatic extracts.[9] Adding two teaspoons of powdered pancreatic extract per 20 kg of body weight to each meal is generally an effective starting dose, and this can be mixed with a maintenance food immediately prior to feeding. When available, use of 85-115 g/20 kg of body weight of chopped raw ox, pig or other pancreas obtained from animals certified as healthy following appropriate inspection is a more economical alternative.[10] As soon as clinical improvement is apparent owners can determine a minimum effective dose of enzyme supplement. Rarely oral bleeding has been correlated with pancreatic enzyme replacement therapy with resolution of clinical signs with dose reduction.[11]

Only a small proportion of the oral dose of each enzyme is delivered functionally intact to the small intestine. As such, attempts have been made to increase the effectiveness of enzyme supplementation, including pre-incubation of enzymes with food prior to feeding, bile salts supplementation, inhibition of gastric acid secretion and use of enteric-coated preparations. None have been shown to consistently improve clinical response.

Nutrient absorption does not return to normal despite appropriate enzyme therapy. As such, patients usually compensate by eating more than usual in order to maintain ideal body weight. Feeding a highly digestible, low-fiber diet helps to compensate for residual digestive deficits.[12]
Dogs with EPI may have severely subnormal concentrations of serum cobalamin and Vitamin E.[1,3,13] Clinical signs associated with naturally occurring Vitamin E deficiency and/or hypocobalaminemia have not been

well documented in the dog, but supplementation of these vitamins if serum levels are decreased is prudent. Administration of vitamin E once daily generally normalizes serum concentrations. Long-term monitoring of serum cobalamin concentration is warranted. Parenteral cobalamin replacement at intervals as frequently as every 1 week may be required for a long-term optimal clinical response.

Dogs with PAA commonly have SIBO, but in most cases this is a subclinical abnormality and affected individuals respond very well to treatment with oral enzyme replacement alone even though the overgrowth usually persists.[1,3,6,13] In those patients who do not respond to oral enzyme supplementation, antibiotic therapy may be appropriate.

Dogs with EPI that do not respond adequately to standard therapies (enzyme supplementation, cobalamin supplementation when hypocobalaminemia is present, antibiotic therapy when SIBO is suspected) should be evaluated further for additional gastrointestinal disease(s), most commonly lymphoplasmacytic gastroenteritis.

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