Nutritional Assessment in a Dog with Chronic Enteropathy

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THE CASE
A 4.5-year-old intact female shar-pei was presented for chronic recurrent diarrhea, which was either watery or mucoid, of more than a year’s duration. Vomiting and hyporexia developed the month before presentation and was associated with mild weight loss. The dog was the only pet in the household and was up-to-date on vaccinations and flea/tick preventives; heartworm prevention was unnecessary, as there is no heartworm disease in Sweden or northern Europe, where this dog lives.

Physical Examination
The patient had to be sedated for physical examination due to temperament. BCS was 4/9, with a muscle condition score showing mild muscle atrophy and a dull hair coat. Despite chronic diarrhea, no signs of dehydration were observed.
All other vital parameters were within normal limits. Rectal palpation was painful despite sedation.

**Dietary History**
Several therapeutic diets labeled intestinal, including a high-fiber diet, had been tried throughout the last year without clinical improvement. The protein sources of those diets included chicken, egg, and turkey, and the owners sometimes gave treats such as cold cuts and table scraps. Water intake remained the same throughout the year. Metronidazole had been prescribed on several occasions; diarrhea would cease with metronidazole but would recur each time after discontinuation of therapy.

**Diagnostic Results**
Diagnostics included screening for intestinal parasites, CBC, serum chemistry profile, and a GI panel, including trypsin-like immunoreactivity, cobalamin, and folate. No intestinal parasites were detected. Subnormal serum concentrations of folate, cobalamin, and cholesterol were detected (**Table**). CBC and serum chemistry profile were otherwise unremarkable.

Endoscopy of the stomach and small and large intestine were performed. Histopathology of biopsies of the small and large intestine revealed a moderate lymphocytic-plasmacytic enteritis, with a moderate degree of villous atrophy, and moderate lymphocytic-plasmacytic colitis.

**DIAGNOSIS:**
**CHRONIC ENTEROPATHY**

**Treatment & Follow-Up**
The dog’s diet was changed to a commercial lamb and rice novel single-source protein diet, and folate supplementation (5 mg PO q24h) was initiated. Treatment with prednisolone was initiated (initial dose, 1.5 mg/kg q24h) and slowly tapered over 6 months (maintenance dose, 0.2 mg/kg q48h). Several attempts to further taper the dose were made but would cause diarrhea to relapse. Four weekly cobalamin injections (800 µg) were administered according to Texas A&M University Gastrointestinal Laboratory recommendations (see **Suggested Reading**, page 35).

At follow-up 4 weeks after the last cobalamin injection, the dog’s stool had normalized, vomiting had stopped, and appetite returned. Serum cobalamin concentration, cholesterol, and folate had normalized (**Table**). Folate and cobalamin supplementation was stopped and prednisolone was further tapered to 0.5 mg/kg q48h.

At follow-up 3 months later, the dog had experienced 2 recurrences of diarrhea, and serum cobalamin concentrations had decreased to subnormal levels. A new parenteral cobalamin maintenance supplementation protocol was recommended; however, the owners were not interested in a new series of injections but were instead interested in oral cobalamin supplementation.

Oral cobalamin supplementation has been proven to be effective in humans with cobalamin deficiency, and recent studies have confirmed its efficacy in dogs and cats with chronic enteropathy and hypocobalaminemia. It offers an alternative to parenteral supplementation and may suit some owners better, as oral administration may be an easier and more cost-effective alternative to monthly injections, particularly for patients requiring long-term maintenance supplementation. Because oral supplementation in dogs with hypocobalaminemia had not been studied at the time of this case, the potential for failure of this therapy was carefully discussed with the owners before supplementation (1 mg PO q24h) was initiated.

At follow-up 2 months later, serum cobalamin concentration was higher than after the first series of injections and the dog was clinically stable. The dog has been on successful oral cobalamin maintenance therapy for 8 years.

**TABLE**

**SUBNORMAL SERUM CHEMISTRY RESULTS**

<table>
<thead>
<tr>
<th>Test</th>
<th>Reference Interval</th>
<th>Baseline</th>
<th>9 Weeks After Baseline</th>
<th>5 Months After Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cobalamin</td>
<td>251-908 ng/L</td>
<td>231 ng/L</td>
<td>705 ng/L</td>
<td>250 ng/L</td>
</tr>
<tr>
<td>Folate</td>
<td>7.7-24.4 µg/L</td>
<td>3.5 µg/L</td>
<td>35 µg/L</td>
<td>25 µg/L</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>158-282 mg/dL</td>
<td>124 mg/dL</td>
<td>189 mg/dL</td>
<td>N/A</td>
</tr>
</tbody>
</table>

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ASK YOURSELF ...

**QUESTION 1**
Changing the diet to a novel protein is most likely to be successful in patients with chronic enteropathy if:
A. Albumin is below the normal reference interval
B. The dog is younger than 3 years
C. Large-bowel diarrhea is present
D. The dog weighs less than 22 lb

**Most Accurate Answer:** B

In a study, dogs with food-responsive chronic enteropathy were significantly younger, weighed more than 22 lb, and had a higher albumin than those with steroid-responsive chronic enteropathy. Differentiating food-responsive from steroid-responsive chronic enteropathy based on clinical signs was not possible.

**QUESTION 2**
Which of the following statements regarding cobalamin is true?
A. All cells in the body require cobalamin.
B. Cobalamin deficiency is a negative prognostic factor in canine chronic enteropathy.
C. Significant weight gain in cats with hypocobalaminemia after supplementation of cobalamin has been observed.
D. All of the above

**Most Accurate Answer:** D

Cobalamin is required as a cofactor for all DNA and protein synthesis; thus, all cells in the body require cobalamin. In 3 separate studies, cobalamin deficiency has been identified as a negative prognostic factor in dogs with chronic enteropathy, exocrine pancreatic insufficiency, and chronic diarrhea. In addition, cats with cobalamin deficiency experienced significant weight gain after cobalamin supplementation with no other changes in treatment.

**QUESTION 3**
What is the most likely mechanism behind the cobalamin deficiency in this patient?
A. Dietary insufficiency
B. Exocrine pancreatic insufficiency with decreased production of intrinsic factor
C. Chronic enteropathy affecting the cobalamin-intrinsic factor receptors in the ileum
D. Familial cobalamin malabsorption

**Most Accurate Answer:** C

Congenital cobalamin malabsorption in shar-peis has been described, and these dogs often have GI signs. However, shar-peis with familial cobalamin malabsorption usually have undetectable serum cobalamin at diagnosis.

**QUESTION 4**
In which breeds has congenital cobalamin malabsorption been reported?
A. German shepherd dog, shar-pei, and Staffordshire bull terrier
B. Beagle, giant schnauzer, shar-pei, border collie, and Australian shepherd dog
C. West Highland white terrier, Labrador retriever, Bichon Havanese, and shar-pei
D. Giant schnauzer, border collie, miniature schnauzer, Bedlington terrier, and Basenji

**Most Accurate Answer:** B

Congenital cobalamin malabsorption has been reported in beagles, giant schnauzers, shar-peis, border collies, and Australian shepherd dogs. German shepherd dogs and Staffordshire bull terriers have a predisposition for hypocobalaminemia, but congenital malabsorption in these breeds has not been proven.

**QUESTION 5**
A middle-aged intact female cocker spaniel with a history of lethargy and reduced appetite of 2 months’ duration has a subnormal serum cobalamin concentration. The dog has been fed a homemade diet due to hyporexia for 6 weeks. How should the cobalamin deficiency be interpreted?
A. Because no diarrhea is present, chronic enteropathy or exocrine pancreatic insufficiency is not the most likely cause of cobalamin deficiency; a dietary imbalance is more likely.
B. The dog could possibly be suffering from chronic enteropathy or exocrine pancreatic insufficiency, as neither condition needs to be associated with diarrhea.
C. Congenital cobalamin malabsorption is the most likely diagnosis.
D. A lipemic serum sample may have caused a false low serum cobalamin concentration.

**Most Accurate Answer:** B
A lack of diarrhea would not exclude a diagnosis of chronic enteropathy in this dog.6,8 In another study, 5% of dogs with exocrine pancreatic insufficiency did not have diarrhea. Dietary insufficiency is less likely, as there have been no reports on naturally occurring cobalamin deficiency in dogs due to a poor diet. Breed and age further make congenital cobalamin supplementation on clinical and biochemical variables in cats with gastrointestinal insufficiency.33,35

References