

Inflammatory Bowel Disease

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A 16-month-old, intact male Irish setter was presented for chronic diarrhea.

History. The dog had a healthy puppyhood, although at 9 months of age it had several days of acute diarrhea after an observed episode of scavenging. *Campylobacter jejuni* had been isolated by stool culture, and the dog had been treated with a 7-day course of erythromycin.

At 12 months of age, the dog's stool had become intermittently soft and gradually worsened over several months. The dog now passed a large volume of watery diarrhea up to 3 times a day and had lost 5 kg in weight. Mucus was occasionally present in the feces, but there was no overt blood. Bilious vomiting occurred approximately once a week. The dog remained bright and had a good appetite. It had been fed a standard supermarket kibble; substitution with a home-cooked, chicken-based diet produced limited improvement.

Examination. The dog had normal stature but was underweight (23 kg; body condition score 2/5). Temperature, pulse, and respiration were within normal limits, and no physical abnormalities were detected. Primary gastrointestinal disease and malabsorption were considered most likely, although routine hematology and serum biochemistry analyses were performed to rule out nongastrointestinal disease. Aside from mild hypoalbuminemia (29 g/L; reference range, 32 to 37 g/L), no abnormalities were found. Hepatic insufficiency and urinary protein loss as causes of the hypoalbuminemia were ruled out because pre- and postprandial bile acids and urine protein–creatinine ratio, respectively, were

within reference ranges. Serum cTLI, folate, and cobalamin concentrations were within reference ranges. Zinc sulfate flotation of two stool samples found no evidence of endoparasitic ova, and stool culture isolated no known pathogens.

Abdominal radiographs and ultrasonography were unremarkable, and diffuse small intestinal disease was considered most likely. Gastroduodenoscopy was performed; the duodenal mucosa was irregular and friable (**Figure 1**). Histopathologic examination of endoscopic biopsy specimens revealed normal gastric mucosa, but moderate-to-severe inflammation of the duodenal mucosa. A diagnosis of lymphoplasmacytic enteritis was made, and treatment was initiated.



Endoscopic appearance of proximal duodenum showing marked granularity of the mucosa, consistent with diffuse inflammation.

ASK YOURSELF...

Which of the following treatments should be given?

- A. Immunosuppressive doses of prednisone
- B. Prednisone and azathioprine
- C. Budesonide
- D. Cyclosporine
- E. None of the above

continues

cTLI = canine trypsin-like immunoreactivity

INSIGHTS FROM CLINICAL CASES . DISCUSSION

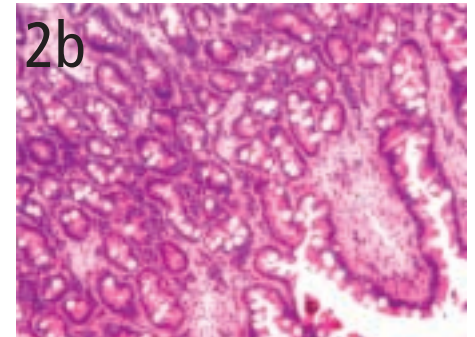
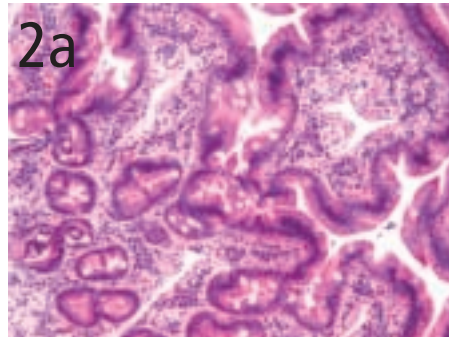
Correct Answer: E None of the above

Although the histologic definition of intestinal inflammation is sometimes debatable, this case was not: there was clear evidence of inflammation. However, this did not prove the dog had idiopathic inflammatory bowel disease. Indeed, this diagnosis would be unusual in a young dog and can only be confirmed once all known causes of intestinal inflammation have been ruled out. Thus, treatment with any of the immunosuppressive drugs listed above would be an erroneous therapeutic option at this stage.

Pinpointing the Cause

Endoscopic biopsy specimens confirmed the presence of lymphoplasmacytic enteritis; however, known causes of intestinal inflammation need to be ruled out before a diagnosis of idiopathic inflammatory bowel disease can be made. It is imperative that the clinician attempt to identify known causes of intestinal inflammation, which include bacterial and parasitic infections and dietary sensitivities.

Fecal examination was negative but empirical treatment with a 3-day course of fenbendazole in case of occult giardiasis was considered worthwhile. However, there was no response. Stool cultures were negative, but a repeated course of erythromycin for *Campylobacter* and 10 days of metronidazole for small intestinal bacterial overgrowth were tried but were also unsuccessful. The dog was then placed on a fish- and rice-based exclusion diet. The diarrhea stopped after 7 days, and the dog gained 3 kg in weight after 3 weeks. Repeated endoscopic biopsies at this time confirmed resolution of intestinal inflammation.



Photomicrographs of intestinal biopsy specimens taken from a Labrador retriever with gastrointestinal signs associated with intestinal inflammation before (a) and after (b) six weeks of treatment with a fish- and potato-based exclusion diet. Comparison shows resolution of the marked lymphoplasmacytic infiltrate with diet alone.

If the dog did not respond to the diet trial, idiopathic inflammatory bowel disease would have been the diagnosis and immunosuppressive treatment would have been justified. Of the treatments listed, immunosuppressive doses of prednisone (2 to 4 mg/kg/day) are the preferred first-line treatment. Azathioprine is only used for its steroid-sparing effect and is not a first-line treatment. Budesonide potentially has fewer side effects than prednisone because it is largely metabolized in the first pass through the liver; however, safety, efficacy, and optimal dosage have yet to be established. Use of cyclosporine in idiopathic inflammatory bowel disease has not been properly evaluated.

When Immunosuppressive Treatment is Justified

The positive clinical response to an exclusion diet suggested that this was a true dietary sensitivity. However, the response may have resulted simply from feeding a better-quality diet to a poorly functioning, inflamed intestine. Thus, a follow-up biopsy was performed and observation of normal intestine strongly indicated that the original diet was the cause of inflammation. Rechallenge of the dog with the original diet and

eliciting clinical and histologic relapse would confirm a true dietary sensitivity; however, the response to treatment made such studies difficult to justify to the client. ■

See Aids & Resources, back page, for references, contacts, and appendices.

TAKE-HOME MESSAGES

- After histologic diagnosis of intestinal inflammation, the clinician must look for underlying causes of intestinal inflammation before making a diagnosis of idiopathic inflammatory bowel disease.
- First-line therapy for truly idiopathic inflammatory bowel disease is immunosuppressive doses of prednisone. Once the patient is in remission, the dose of prednisone is gradually tapered, but if relapse occurs at unacceptably high doses, azathioprine can be introduced for its steroid-sparing effect.