applied endoscopy

CASE STUDY OF THE MONTH. PRESENTATION



Chronic Diarrhea in a Dog

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Teddy, a previously healthy, 2.5-kg, 8-year-old intact male Yorkshire terrier, presented with chronic diarrhea, abdominal distention, and poor body condition.

History. The diarrhea was chronic (6 weeks) and was characterized by 1 to 2 bowel movements a day, "pasty" consistency, normal brown color, no mucus, no blood, and no tenesmus. The abdominal distention was first noted 1 week prior. Teddy was obtained as a puppy and lives with one other dog that was free of clinical signs. Vaccinations were current, and he was receiving heartworm preventative (ivermectin).

Physical Examination. Teddy was quiet, alert, and responsive. Temperature, respiratory rate, and heart rate were within normal limits. He

was thin (body condition score 3/9) and missing some teeth. Abdominal effusion was detected by ballottement. There was no palpable abdominal organomegaly.

Diagnostic Plan. CBC, biochemical profile, urinalysis, fecal flotation (zinc sulfate centrifugation technique), abdominal ultrasonography, and abdominocentesis were performed. CBC showed mild leukocytosis (16,700/µl) characterized by mild neutrophilia (12,191/µl) and monocytosis (1837/µl). The most significant finding on the biochemical profile was severe panhypoproteinemia (total protein, 3.5 g/dl; albumin, 1.4 g/dl; globulin, 2.1 g/dl). Mild hypocalcemia (7.6 mg/dl) was also present but was determined to be normal when corrected for hypoalbuminemia (formula: measured calcium [mg/dl] – albumin [g/dl] + 3.5 =corrected calcium [mg/dl]). Urinalysis was normal (urine specific gravity 1.020, negative for protein). Fecal flotation was negative for parasites. Abdominal ultrasonography showed moderate abdominal effusion and several bowel loops distended with luminal fluid. Fluid obtained by abdominocentesis was consistent with a transudate. Normal pre- and postprandial serum bile acids ruled out hepatic disease as a cause of the hypoalbuminemia. Thoracic radiographs were taken to rule out the presence of concurrent pleural effusion. The radiographs were normal.

Differential Diagnoses. Problems identified include chronic small bowel diarrhea, panhy-

poproteinemia, ascites, and loss of body condition (amount of weight loss could not be accurately determined because of the contribution made to the body weight by the ascites). Chronic small bowel diarrhea combined with panhypoproteinemia in the absence of proteinuria or hepatic insufficiency was consistent with protein-losing enteropathy. Hypoproteinemia associated with renal or liver disease is usually characterized by decreased albumin and normal globulin. Differentials for protein-losing enteropathy include inflammatory bowel disease (lymphocytic-plasmacytic, eosinophilic, or granulomatous enteritis), intestinal lymphangiectasia, intestinal neoplasia (lymphoma), and infectious disease

continues

New Article Series

Applied Endoscopy, a new series of articles in *Clinician's Brief*, will challenge and develop the reader's ability to apply endoscopic techniques and interpret endoscopic images. The series will cover a variety of topics, discussing a case and the use of endoscopy in its diagnosis.

CASE STUDY OF THE MONTH. PRESENTATION & DIAGNOSIS

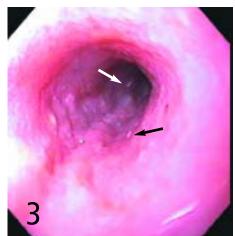
(histoplasmosis, pythiosis, phycomycosis). Ascites could result from decreased oncotic pressure, lymphatic obstruction, increased vascular permeability, and increased hydrostatic pressure, or a combination of these processes. Weight loss could result from energy loss (protein), malassimilation, increased energy expenditure (neoplasia, chronic disease), and/or decreased energy intake. A protein-losing enteropathy could account for all the clinical problems observed in this case.

Endoscopy. Dogs with protein-losing enteropathy have severe alimentary tract disease that should be diagnosed promptly to maximize the chance of successful therapy. Obtaining small intestinal biopsy specimens is essential for determining the cause of protein-losing enteropathy. Endoscopy is preferred over surgery to obtain biopsy specimens because endoscopy

allows the operator to visualize mucosal lesions and obtaining surgical full-thickness biopsy specimens in patients with a serum albumin concentration below 1.5 g/dl carries some risk for dehiscence. Upper gastrointestinal

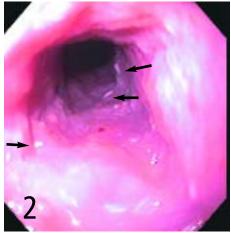
endoscopy was performed in this case. Figures 1 through 3 are images taken in the duodenum. Several biopsy specimens were obtained from the stomach and duodenum during the procedure.







- In addition to panhypoproteinemia and hypocalcemia, what other clinical pathologic abnormalities have been associated with lymphangiectasia?
- Is a plasma transfusion indicated?
- Describe the gross endoscopic appearance of the duodenal mucosa in the Figures.
- Based on the gross endoscopic appearance, which differential diagnoses would you consider most likely?



at a Glance

- Highly digestible, hypoallergenic (protein hydrolysate), moderately low fat diet
- Immunosuppressive drug therapy
 - Prednisone—2 mg/kg Q 12 H
 - Azathioprine—2.2 mg/kg Q 24 H Both drugs were prescribed due to disease severity.

Diagnosis: Severe lymphocytic-plasmacytic enteritis with marked intestinal lymphangiectasia and crypt distention

Endoscopic biopsy specimens from the duodenum showed an intense plasmacytic infiltrate with smaller numbers of lymphocytes and occasional neutrophils. Several dilated duodenal crypts containing amorphous matter and cellular debris were found throughout the biopsy specimens. The villar lacteals were dilated and contained a flocculent eosinophilic material. These findings were consistent with marked lymphocytic-plasmacytic enteritis and marked lymphangiectasia and crypt distention.

Intestinal lymphangiectasia is characterized by intestinal lymphatic obstruction (dilatation) and dysfunction. Hypoproteinemia results from leakage of protein-rich lymph into intestinal lumen. The disease can be primary or secondary. In both types, the pathogenesis seems to be associated with inflammation of the intestinal lymphatics. The primary form is considered congenital, although clinical signs may not manifest for vears. Progression is believed to be associated with pyogranulomatous lymphadenitis superimposed on the congenital lymphatic malformation. Small terrier breeds (e.g., Yorkshire, soft-coated wheaten) and lundehunds are most commonly affected. The secondary form is caused by lymphatic infiltration or obstruction resulting from inflammatory (inflammatory bowel disease, fungal disease) or fibrosing processes, thoracic duct obstruction, or rightsided heart failure.

The signalment and clinical findings in this case could be consistent with either primary lymphangiectasia with secondary inflammation or lymphangiectasia secondary to lymphocytic-plasmacytic enteritis. Because of the severity of the inflammatory process, the latter diagnosis was considered more likely.

DID YOU ANSWER ...

- Lymphopenia and hypocholesterolemia
- Plasma transfusions are rarely of significant benefit because the added albumin is often quickly lost into the intestinal lumen. In addition, large amounts of plasma are usually needed to significantly increase serum albumin (except in small dogs), which can make this a costly approach. If increasing oncotic pressure is considered important before endoscopy or surgery, the use of a synthetic colloid (e.g., hetastarch) may be more cost-effective.
- Moderate mucosal irregularity with scattered mild erosive lesions and dilated lacteals (see arrows on Figures) are observed.
- The gross finding of dilated lacteals is suspect for lymphangiectasia. Often, more dilated lacteals are visible in cases of lymphangiectasia than are observed in this case. In addition, the mucosal irregularity is consistent with an inflammatory or infiltrative disease process; however, histopathologic evaluation is necessary to determine the underlying cause.

Treatment. Treatment consisted of a highly digestible hypoallergenic diet (protein hydrolysate) combined with immunosuppressive drug therapy. The hypoallergenic diet was used because of the likelihood that food sensitivity is involved in the pathogenesis and perpetuation of inflammatory bowel disease. The diet was also sufficiently low in fat—dietary fat is poorly assimilated and may exacerbate leakage of protein-rich lymph from the dilated lacteals. Initial immunosuppressive drug therapy consisted of prednisone (2 mg/kg Q 12 H) and azathioprine (2.2 mg/kg Q 24 H); the combination was prescribed because of the severity of the disease process.

Follow-up. After 2 weeks, the ascites (confirmed by ultrasonography) and diarrhea had resolved. Body weight was 2.2 kg (the lost body weight is attributable to the resolution of the ascites). Total protein level was 5.1 g/dl, with an albumin level of 3.1 g/dl and a globulin level of 2.0 g/dl. Despite the dramatic initial improvement, the long-term prognosis for dogs with intestinal lymphangiectasia is guarded. However, the prognosis is improved in cases of secondary intestinal lymphangiectasia-associated inflammatory bowel disease (IBD) when IBD is effectively controlled. If a favorable clinical response continues, the prednisone dose will be gradually decreased (monthly intervals) until a dose of 0.5 mg/kg Q 48 H is achieved. The azathioprine dose interval will be reduced to Q 48 H after one month. Treatment may need to be continued indefinitely at these doses to control the disease process.

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