

INFLAMMATORY BOWEL DISEASE IN A GERIATRIC DOG

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Introduction

Inflammatory Bowel Disease (IBD) can be frustrating to diagnose and to treat, as the clinical signs can be variable. The case described below is one of many diverse clinical presentations seen with this disease.

History

Libby, a 13 YO FS Labrador, presented for a history of chronic intermittent vomiting which began 8 months prior to presentation. Episodes last for a few days and then seem to resolve on their own. Initial workup, including radiographs, did not reveal a cause for vomiting. Libby's diet is Flint River Ranch dry food. Current medications include Prednisone 5 mg twice daily and DES 1 mg once weekly. She was receiving Carprofen, but this was discontinued prior to the Prednisone administration, which has improved Libby's vomiting.

Diagnostics and Treatment

On presentation, Libby was bright and alert. Abnormalities on her physical examination included palpable cranial abdominal organomegaly, thickened fused tarsal joints, symmetrical muscle wasting, and lick granulomas. Diagnostics included repeat CBC and chemistry, T4, urinalysis, urine culture, B12 and folate levels as well as an abdominal ultrasound. A diet trial was instituted with Purina HA and Libby was dewormed with Fenbendazole.

Abnormalities on bloodwork included an elevated ALT at 356 U/L (ref 12-118), elevated ALP of 1,646 U/L (ref 5-131), and an elevated GGT at 22 U/L (ref 1-12). Libby's total T4 was at the low end of the normal range, 0.8 ug/dL (ref 0.8-3.5), however a free T4 was within normal limits. Urinalysis revealed isosthenuria with a quiet sediment. The B12 level was low normal at 340 pg/mL (normal 249-733). The folate was slightly decreased at 5.23 ng/mL (normal 6.5-11.5). Liver enzyme elevations and isosthenuria were attributed to Prednisone administration and possibly Cushing's disease. Low normal B12 and folate were concerning for primary gastrointestinal disease.

Abdominal ultrasound abnormalities included a heteroechoic liver and adrenal glands at the upper limits of the normal range. The stomach wall was possibly thickened and there was speckling in the duodenal mucosa (Fig 1), although the wall layering of the small intestine was normal and the thickness was also within normal limits.

The diet trial decreased the frequency of vomiting, however there was some inappetence and mild weight loss. The vomiting also improved with the administration of Famotidine and Sucralfate. Due to the weight loss and thickening in her stomach, Libby's owner elected to pursue endoscopic biopsies.

The appearance of the stomach on endoscopy was grossly abnormal. It contained a poorly-marginated area in which the mucosa appeared thickened and hyperemic with a loss of rugal folds (Fig 2). There were two polypoid masses immediately adjacent to the pylorus (Fig 3). The appearance of the duode-

num was also abnormal with areas of hyperemic tissue and some areas of pale tissue (Fig 4). Pinch biopsies were taken of the duodenum, the polypoid masses, as well as abnormal and normal appearing regions of the stomach. Biopsy results were consistent with chronic, proliferative, lymphoplasmacytic and eosinophilic gastritis as well as mild, lymphoplasmacytic and eosinophilic enteritis.

Libby is continuing to do well on her hydrolyzed diet and intermittent Famotidine. If her clinical signs recur, the next therapeutic step will be a trial of an antibiotic.

Discussion

IBD is a term applied to a group of diseases characterized by chronic gastrointestinal signs and inflammation within the wall of the bowel on histopathology. Diagnosis of IBD requires exclusion of other causes of GI inflammation, as well as biopsy confirmation of characteristic inflammatory cell infiltrate. Exclusion of other causes involves full workup and a prophylactic course of a broad spectrum dewormer. It is recommended that the full workup (complete bloodwork and urinalysis, imaging including thoracic radiographs and abdominal ultrasound, and GI specific bloodwork such as B12 level and possibly cPLI) be performed prior to considering biopsy.

The etiology of IBD is unknown, but genetics, environment, and diet may all play a role. IBD is classified according to the inflammatory cell type on histopathology, with lymphoplasmacytic being most common. Eosinophilic is the second most common, with granulomatous being the least common and seen often in young Boxers and French Bulldogs.

IBD may also be characterized based on response to treatment. Positive response to a diet trial with a hydrolyzed diet or novel protein diet warrants a diagnosis of food-responsive IBD. Failure to respond to diet trial warrants an antibiotic trial, usually with Tylosin or Metronidazole. Patients that do not respond to either of these treatments may require immunosuppressive therapy but biopsy is always recommended prior to beginning immunomodulatory therapy. Approximately one quarter of canine IBD patients can be expected to progress to complete remission. The remainder will have some clinical signs and/or require some form of lifelong treatment. Factors that are related to a worse prognosis include hypoalbuminemia and hypocobalaminemia.

Summary

IBD is a diagnosis which should be considered in any patient with chronic gastrointestinal signs. A full workup should be performed prior to biopsy. Lesions on histopathology can be variable and are not related to prognosis. Although Libby is a geriatric dog, thus warranting a careful workup for potential neoplasia, IBD can be seen in any age patient and has a variable clinical course.



Figure 1: Ultrasound image of duodenum with speckled mucosa (a non-specific finding).



Figure 2: Abnormal region of stomach wall, with loss of normal rugal folds.



Figure 3: Polypoid masses near the pylorus.

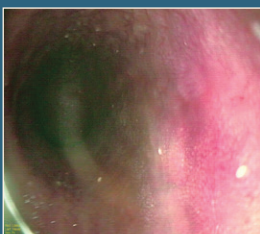


Figure 4: Abnormal appearance of duodenal mucosa.

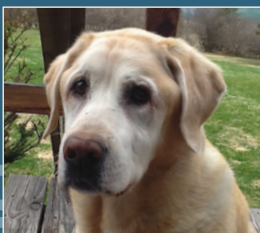


Figure 5: Libby, doing well on her hydrolyzed diet and symptomatic treatment.





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