

Eosinophilic gastroenteritis in basset hound dog

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ABSTRACT

An 1-year-old male Basset Hound dog was evaluated for chronic intermittent vomiting, hematemesis, and melena which had been ongoing for several months. The histopathologic examination revealed that all layers of the small intestine were thicker than normal. The lamina propria of the mucosa, including the villi, exhibited a prominent cellular infiltrate which consisted of numerous eosinophils and an increased numbers of plasma cells in addition to the normal lymphocytic component. The muscularis mucosa was invaded, and in some places disrupted, by eosinophils, which also infiltrated into the submucosa and muscularis propria. This report describes the pathological findings of a case of eosinophilic gastroenteritis (EG) in a dog.

Keywords: Eosinophilic Granuloma; Gastroenteritis; Canine

1. INTRODUCTION

Eosinophilic inflammatory bowel disease is a chronic idiopathic inflammatory disease of the intestine characterized by the presence of a mixed inflammatory infiltrate in which eosinophils predominate or constitute the most prominent cell population [1]. Inexperienced histopathologists need to realize that normal intestine has a noticeable population of eosinophils so as to avoid making a misdiagnosis of eosinophilic enteritis [2]. The causes of this disease are poorly defined, although allergens in food have been implicated. Migrating parasites can also induce EG.

Several theories exist about the pathogenesis of EG in humans, including: an autoimmune response to a luminal or mucosal antigen; a dysfunctional immune response to commensal bacteria; and an infection with a pathogenic organism that either remains in the tissues resulting in chronic inflammation, or creates ongoing deregulation of the immune response after resolution of infection [3].

Recent advances in human medicine also point out the significance of genetic factors in the predisposition, modulation and perpetuation of eosinophilic enteritis [4].

The clinical signs are similar to those of other chronic gastroenteritises and may include vomiting, small bowel type diarrhea, weight loss, and anorexia [5]. Eosinophilia is an inconsistent finding in dogs. Nevertheless, eosinophilia is common in animals with gastrointestinal diseases of all kinds and biopsies of the intestines are needed to achieve a diagnosis. Selected protein-controlled diets may be administered, but corticosteroid treatment is often still [6]. Histopathologic evaluation of biopsies from the gastrointestinal tract (GIT) is an important tool in the diagnosis of chronic GIT diseases. A definitive diagnosis can only be made based on histopathologic analysis of GIT biopsy specimens, especially in eosinophilic enteritis [7].

The treatment for canine eosinophilic enteritis is largely empirical due to the poor understanding of the etiopathogenesis and lack of therapeutic trials [8,9]. Administration of corticosteroids, sulfasalazine, azathioprine, antibiotics and specific diets, whether singly or in combination, are currently the main treatments for all histological types of human eosinophilic enteritis [10,11]. The efficacy of these approaches has not been determined in canine EG, and there is no discrimination between drugs prescribed for either induction or maintenance of remission. In humans, for example, it has been reported that sulfasalazine is more effective for maintenance therapy than induction of remission [12]. This report describes the pathological findings of a EG case in a dog.

2. CASE REPORT

An 1-year-old male Basset Hound dog was evaluated for chronic intermittent vomiting, hematemesis, and melena which had been ongoing for several months. The dog was fully vaccinated and was wormed every four months with oral fenbendazole (Panacur; Hoechst). On physical examination, no abnormalities were detected. The dog was mildly pained during abdominal palpation,

but no other signs of abnormalities were detected. The blood count revealed slight anemia (35% hematocrit, reference range 35% - 55%), marked leukocytosis (21×10^3 leukocytes/ml, reference range $6.0 - 12.0 \times 10^3$ leukocytes/ml), a decreased percentage of segmented neutrophilic granulocytes (24%, reference range 60% - 75%), 10% lymphocytes (reference range 15% - 30%), 71% eosinophils (reference range 0% - 6%), and moderate thrombocytopenia (111×10^3 platelets/ml, reference range $150 - 500 \times 10^3$ platelets/ml). Serum biochemistry showed normal values for liver enzymes (alanine aminotransferase 40 U/liter, reference range up to 50 U/liter; glutamate dehydrogenase 5.0 U/liter, reference range up to 6.0 U/liter), and total abnormal values for albumin (1.3 g/dl, reference range for dogs over 1 year 3.2 - 4.1 g/dl).

The animal had been subjected to X-ray and ultrasound. Lateral and ventrodorsal survey and contrast abdominal radiographs revealed a thickened gastric wall and a small amount of gas in the small intestine. Diagnostic imaging exams should always be performed when eosinophilic gastroenteritis is suspected. Radiographic examination and abdominal ultrasound are the most often exams requested. In this case, an exploratory laparotomy had also been performed. Few gross abnormalities were visualized during surgery. The duodenum was slightly thickened on palpation but was normal in appearance. The jejunum and ileum was considerably thickened throughout its length and the pylorus was also thickened. Because of the diffuse nature of the bowel changes, which resembled a neoplasm, euthanasia was advised.

At necropsy, there were multiple foci of omentum adhesion around the stomach. The spleen had a rough surface and one end was 9 cm wide. The greater curvature of the stomach exhibited a 7 cm whitish thickened area (**Figure 1**). The final portion of the colon was hardened, with thickening of the wall, which was 3 cm thick. Two colonic lymph nodes were enlarged and measured 3 and 4 cm in diameter. The duodenum and jejunum also exhibited some degree of wall thickening.

Tissues were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 5μ , and stained with hematoxylin and eosin for routine evaluation. Selected tissues were stained with Warthin-Starry, Gram, and

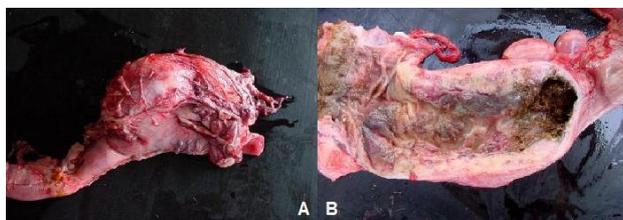


Figure 1. Eosinophilic gastroenteritis, stomach and duodenum. Was also observed small wall thickening, necrosis and hemorrhagic foci.

acid-fast stains to reveal bacteria. Giemsa, Gomori's methenamine-silver, and periodic acid-Schiff stains were used to stain fungi or parasitic remnants. The histopathologic examination revealed that all small intestine layers were thicker than normal. The lamina propria of the mucosa, including the villi, exhibited a prominent cellular infiltrate which consisted of numerous eosinophils (**Figure 2**) and increased numbers of plasma cells, in addition to the normal lymphocytic components. The muscularis mucosa was invaded, and in some places disrupted, by eosinophils which also infiltrated into the submucosa and muscularis propria. The inner muscular was the most severely affected of the layers and was permeated with numerous eosinophils; therefore, this layer was greatly thickened. A diagnosis was made on the basis of the dog's clinical signs, necropsy and the histopathological findings.

3. DISCUSSION

EG is a rare, poorly understood condition characterized by presence of eosinophilic leukocytes. The histologic hallmark of EG is infiltration of the gastrointestinal tract mucosa by eosinophils. The cause of eosinophilic gastroenteritis is unknown. Several factors have been shown to be involved in the pathogenesis of this disease [13]. The clinical signs, laboratory findings and type of disease reported in this case are similar to previous studies [7,8]. Dogs of five years and under are most commonly affected; there is no sex predisposition, and German Shepherd dogs and Rottweilers are more predisposed.

Vomiting, weight loss and abdominal discomfort signs are usually observed early in the course of the disease and tenesmus, hematochezia and inability to urinate are seen in the final stages as the disease progresses. Although eosinophilic gastroenteritis is the most common cause of chronic vomiting and diarrhea in the dog, diagnosis may be difficult [5]. The clinical history usually includes chronic or intermittent vomiting, diarrhea, inappetence, and weight loss. Hematemesis, hematochezia, or melena may be present as a result of gastrointestinal ulceration [13,14].

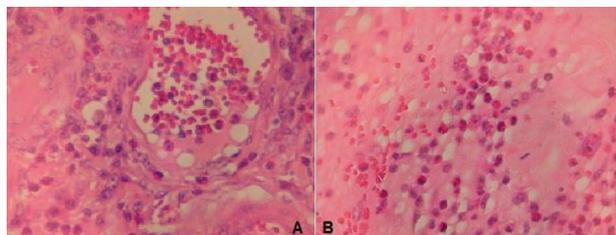


Figure 2. The lamina propria of the mucosa, showed a prominent cellular infiltrate consisting of numerous eosinophils. HE (40 \times).

The dog of this report presented thrombocytopenia and hypoalbuminaemia. It is reported in humans that the thrombocytopenia may or may not resolve with treatment of inflammatory bowel disease [15]. The pathophysiology of hypoproteinaemia may involve reduced appetite, malabsorption due to a reduction in intestinal surface area, such as with villus atrophy or fibrosis, haemorrhage or exudation of protein into the gastrointestinal tract, and increased intestinal permeability [2].

EG is defined based on the following criteria: 1) upper gastrointestinal tract symptoms, 2) eosinophilic infiltration in the gastrointestinal tract (more than 20 eosinophils per high power field), and 3) exclusion of other general causes of eosinophilia (parasitic infestations, connective tissue disease, hypereosinophilic syndrome, lymphoma and other intestinal tumors, primary amyloidosis, celiac disease, vasculitic syndromes). Inflammatory bowel disease may mimic infiltrative neoplastic formations, especially when there is severe thickening of the walls and loss of individualization of its layers. The ultrasound scan is not sufficient to differentiate between these conditions [10].

At necropsy, the pylorus and the entire length of the small intestine were markedly thickened; additionally, luminal hemorrhages were observed beneath the mucosa. There was no gross evidence of helminth infestation. Eosinophils were also present in the outer (longitudinal) muscle layer but the muscle fibers exhibited much less fragmentation. Both muscle layers had degenerated muscle fibers and hemorrhage areas. The serous layer was greatly thickened owing to the presence of numerous eosinophils. Some necrotic foci and polymorphonuclear collagen were observed in the colonic submucosa. The same infiltration was observed in two colonic and splenic lymph nodes. No fungal structures were observed in periodic acid-Schiff stain (PAS).

4. CONCLUSION

The diagnosis of EG was based on clinical information, necropsy and histological findings. The causes of this syndrome often can not be determined, as in this case. In dogs, granulomatous inflammatory lesions of the gastrointestinal tract may have noninfectious causes or result from chronic infections.

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