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DIAGNOSIS AND MANAGEMENT OF CANINE INFLAMMATORY BOWEL DISEASE

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Definition

Inflammatory bowel disease (IBD) by definition is a chronic inflammatory process involving the gastrointestinal (GI) tract, with no identifiable underlying cause. Inflammation can be centred on the stomach, small intestine or large intestine and clinical signs can either be persistent or intermittent. The underlying aetiology of IBD is complex and involves interaction between the host gut flora, environmental factors and genetic predispositions. For the purposes of this presentation, an approach to dogs with small bowel diarrhoea will be discussed.

Diagnosis

Clinical presentation

Clinical signs of IBD in dogs are highly dependent on the site of the GI tract that is affected. Involvement of the stomach or proximal small intestine results in vomiting, and small bowel involvement is associated with diarrhoea and weight loss. With severe disease, GI haemorrhage can cause melena and signs associated with anaemia, and protein loss may also result in ascites and/or pleural effusion due to decreased vascular oncotic pressure. When the large bowel is involved, mucoid faeces, tenesmus and haematochezia develop, whilst weight loss is rare. Clinical signs may also be triggered by stress or be episodic in nature.

There are reported breed predispositions that may increase the index of suspicion of IBD, such as Boxer dogs with granulomatous colitis and Soft-Coated Wheaten terriers with protein-losing enteropathy, but the condition can affect any breed or gender. It is very important to determine the dietary history, any previous medication and to establish the parasitic prophylactic regime given to the dog. The presence of concurrent pruritis in a young dog increases the index of suspicion for a dietary aetiology.

Possible differentials for a dog with weight loss and small bowel diarrhoea include exocrine pancreatic insufficiency, food responsive or antibiotic responsive diarrhoea, liver disease and hypoadrenocorticism. When there is concurrent vomiting, melena, ascites or haematochezia other differentials to consider include infiltrative
neoplasia (lymphoma, adenocarcinoma, leiomyosarcomas, and gastrointestinal stromal tumours), lymphangiectasia, partial intestinal obstruction or severe parasitism.

**Laboratory assessment**

Clinical pathology testing is important to establish if there is evidence of anaemia or hypoproteinaemia. The presence of electrolyte abnormalities or eosinophilia may alert the clinician to the possibility of hypoadrenocorticism or parasitism. Further testing such as an ACTH stimulation test may be of benefit in those dogs. In dogs with weight loss, measurement of serum trypsin-like immunoreactivity (cTLI) is essential to rule out exocrine pancreatic insufficiency. Serum cobalamin is often reduced in chronic GI disease in dogs, and alongside measurement of cTLI may be beneficial.

Faecal analysis can be considered in dogs with large bowel diarrhoea (to evaluate for Trichurus), or in young dogs with poor antihelminthic prophylaxis. Pythiosis should also be considered in young, large breed dogs with granulomatous inflammation causing obstruction. Serology detecting antibodies against P insidiosum is more diagnostically useful than faecal analysis.

**Imaging**

Abdominal radiographs are seldom useful in the investigation of chronic diarrhoea in dogs. Abdominal ultrasound, preferably performed by a qualified radiologist, can however be of great benefit. In particular, the size, shape and echogenicity of mesenteric lymph nodes and organs such as liver, kidney and pancreas should be assessed. If lesions are present then aspiration may be indicated. Free abdominal fluid should also be aspirated if present. The GI tract itself should be screened for wall thickness, layering and the distribution of those changes. IBD can cause focal or diffuse thickening of the intestinal wall, with or without loss of wall layering. It is not possible to differentiate between inflammatory changes and neoplastic changes on abdominal ultrasound, and equally the absence of intestinal changes does not rule out IBD. However, if there is a focal area of intestinal wall thickening with loss of wall layering, it is more likely to be neoplastic than inflammatory. Some studies have also detected differences in Doppler-derived resistance in dogs with food allergy.

**Intestinal histopathology**

Regardless of the imaging or clinical findings, diagnosis is dependent on demonstration of inflammation histologically. Samples for analysis can be obtained either via endoscopy or exploratory surgery. Both have their advantages and disadvantages—the former being less invasive and expensive, but only obtains mucosal samples from a limited area of the GI tract; whilst the latter is invasive, expensive and carries the risk of poor wound healing. Evaluation of the intestinal biopsies should be carried out consistently, using a standardised process such as the WSAVA guidelines. The presence of inflammatory infiltrate, villus atrophy and lacteal dilation are assessed and graded in this system.

**Assessing severity**

Before embarking on a management plan, assessment of disease severity should be made. This can be done by assessing a variety of clinical signs (frequency of vomiting, presence of weight loss, anaemia, melena etc) with or without measurement of inflammatory markers such as C-reactive protein (CRP).
**Management**

All dogs prior to treatment, and often prior to invasive investigation such as endoscopy should ideally receive a 5-day course of fenbendazole at 50 mg/kg once daily to treat for Giardia and other intestinal parasites. In severely affected dogs however, investigation and further management should not be delayed for this.

If reduced cobalamin is documented in individual dogs then treatment with parenteral cobalamin should be given until serum levels normalise, in conjunction with other treatment.

In mildly affected dogs, the next step is to provide a new diet for 2-4 weeks. If this is successful, then this can be continued for up to 6 months before re-challenging with the dog’s previous diet. A large percentage of dogs with IBD will be food responsive within a short period of time (2-3 weeks), but the exact mechanism by which this occurs is unknown as even in food responsive animals, the immunological response is rarely true food allergy (hypersensitivity). Some dogs maintain long-term responsiveness to dietary modification, whilst others will relapse after a certain period of time. Diets of benefit for dogs with IBD have reduced allergenic exposure (use of novel or hydrolysed proteins/carbohydrates), are highly digestible and in many cases will have added fatty acids or prebiotics such as inulin. One study suggests superiority when feeding a hydrolysed soy diet compared to a non-hypoallergenic intestinal diet9, however large comparative studies are lacking.

If dietary therapy is unsuccessful, then an antibiotic trial should be undertaken, and continued for 4 weeks if helpful. Antibiotics can include tylosin, oxytetracycline or metronidazole. If this is successful, then discontinuing the antibiotics may result in dietary management alone being sufficient after this period. After this step, if there is a poor response then immune suppression should be used. Prednisolone is most commonly used at a dosage of 1.0-2.0 mg/kg/day tapering down every 2-4 weeks. The addition of azathioprine or cyclosporine should be considered if initially glucocorticoids are helpful, to allow the glucocorticoid dosage to be reduced. If initial use of glucocorticoids is not helpful, the diagnosis should be reconsidered prior to increasing immune suppression in case the initial diagnosis is incorrect.

When the dog is severely affected, concurrent dietary, antibiotic and prednisolone therapies should be initiated. Azathioprine is often started early in these dogs to allow early reduction of glucocorticoids. Again, additional immune suppression should be reserved for when the diagnosis is definitive. When ascites is present additional measures such as abdominocentesis and diuretic therapy may be required.

There is currently a lot of interest in the role of both prebiotics and probiotics, which may in the near future be shown to be of benefit to dogs with IBD. Overall, the prognosis for dogs that are mildly to moderately affected is reasonable although individual and targeted treatment strategies may be needed.

**References**