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THE APPROACH TO CHRONIC DIARRHEA IN THE DOG

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OVERVIEW

Intestinal parasites are the most common cause of chronic diarrhea in dogs worldwide. They usually include nematodes (e.g., hookworms and whipworms) and/or protozoa (e.g. *Giardia* spp.), but other locally prevalent parasites must also be considered. Ruling out the presence of parasites and initiating proper anthelminthic treatment if they are present are the essential first steps in approaching dogs with chronic diarrhea.

After parasites have been ruled out, “chronic enteropathies” (CE) or “chronic inflammatory enteropathies” (CIE) need to be considered. The term CIE is currently preferred over other terms, such as IBD, to describe dogs with chronic intestinal diseases of unknown origin.

GLOSSARY

The following definitions apply to diseases affecting the small and/or the large intestine:

- Chronic inflammatory enteropathies (CIE): a term encompassing all chronic inflammatory intestinal diseases of unknown origin.
- Diet-responsive CIE: a form of CIE that responds to a dietary trial with an elimination diet based on a novel protein source or a hydrolyzed peptide diet. They are by far the most common type of CIE.
- Idiopathic/cryptogenic intestinal dysbiosis: a form of CIE associated with a severe imbalance of intestinal microbiota that does not respond to a dietary approach. It has been described in young to middle-aged large-breed dogs.
- Immunosuppressant-responsive CIE: a more severe form of CIE that does not respond to a dietary elimination trial and requires the use of immunosuppressive drugs. IR-CIE is usually associated with moderate to severe infiltration of the intestinal mucosa with inflammatory cells (e.g., lymphocytes, plasma cells, eosinophils, neutrophils, macrophages and/or a combination of 2 or more types). In addition, changes in mucosal architecture, such as stunting of the small intestinal villi, are also common. Severe IR-CIE may cause protein-losing enteropathy (PLE).
- Granulomatous colitis of boxers and bulldogs is a particular form of CIE that affects young boxers and bulldogs and responds to fluoroquinolone antibiotics.

ETIOLOGY

Canine CIE occur following dysregulation of the intestinal mucosal immune system and its interactions with intestinal microbiota and/or dietary components, and compromised integrity of the intestinal mucosal barrier. Risk factors for developing CIE later in life include experiencing severe GI injury, such as parvovirus infection, acute hemorrhagic diarrhea syndrome (AHDS), or giardiasis in early life. In addition, in a large population of Finnish dogs, feeding non-processed meat-based diets and giving the dog human meal leftovers and table scraps during puppyhood and adolescence were protective against CIE. However, feeding ultra-processed diets (e.g., dry kibbles) and raw-hide bones during that same period appeared to be significant risk factors for the development of CIE later in life.

CLINICAL PRESENTATION

CIE are associated with chronic or chronic-intermittent diarrhea of more than 3 weeks duration. Information from the patient’s history is useful in evaluating which segments of the intestine are likely involved. Deciding if the diarrhea of small bowel, large bowel, or mixed origin is helpful in tailoring the further diagnostic workup and the initial treatment approach to each patient.

Mild CIE may cause intermittent clinical signs, whereas progressive and severe clinical signs are common in severe cases. Poor body condition with poor hair coat is frequent with severe disease. Some animals may regularly vomit, and dehydration is possible. Animals may show pain or discomfort on abdominal

palpation. Ascites, hydrothorax, and peripheral edema may occur in cases with significant protein loss (PLE).

Use of a clinical grading system, such as CIBDAI or CCECAI, is recommended in dogs with CIE, particularly if moderate to severe clinical signs are present. It allows a more objective semi-quantitative assessment of disease severity and is useful to evaluate treatment success. The form used at the author's institution can be downloaded from the following website: https://drive.google.com/file/d/1yepOFQimxjZlXKRu47X3XJObFQCTMPRr/view?usp=sharing.

DIFFERENTIAL DIAGNOSIS

General differentials for chronic diarrhea include intestinal parasitic diseases, bacterial enteritis (rare in dogs, but a consideration in dogs eating raw diets or other sources of enteropathogens), and diseases originating outside the GI tract such as eukalemic, eunatremic hypoadrenocorticism (also known as "atypical Addison's disease", which was diagnosed in between 1 and 4% of dogs with chronic diarrhea in European clinical studies), exocrine pancreatic insufficiency (EPI), and chronic pancreatitis. In addition, chronic kidney disease and chronic liver disease could also be associated with chronic diarrhea.

DIAGNOSIS

Diagnosis of CIE consists of an elimination process. The aim is to rule out other diseases of known etiology that may cause similar clinical signs: fecal flotation and *Giardia* antigen test, or parasite panels using nucleic acid amplification testing should be performed in all dogs. Empiric parasiticide treatment may contribute to benzimidazole resistance in hookworms and other parasites and is not recommended anymore. Notably, several avermectins administered for heartworm prevention also offer protection against hookworm and whipworm infestation, thereby making these parasites less likely in dogs receiving these products. Signalment and history are useful to decide, for instance, if a resting serum cortisol concentration should be obtained to rule out hypoadrenocorticism or if TLI should be assessed in dogs satisfying the criteria for EPI.

- Dogs with mild to moderate disease can undergo a treatment trial with a novel protein or hydrolyzed peptide diet (see below).
- Severely affected dogs showing significant systemic repercussions of their intestinal disease should be evaluated more thoroughly with the collection of a minimal database including CBC, serum biochemistry, and urinalysis. These investigations aim to gauge the severity and extent of the intestinal inflammation and assess the systemic impact of the intestinal disease on the patient. Serum cobalamin concentrations should be evaluated. The sensitivity of abdominal ultrasound is intermediate, and scans may be normal or show focal or diffuse loss of wall layering, presence of mucosal striations or spicules, wall thickening, or enlarged and/or hypoechoic mesenteric lymph nodes. If lesions are present, localization to a specific intestinal segment may be helpful.

Changes in the gut microbiome may be the primary driver of CIE or result from mucosal inflammation. Evaluation of the gut microbiome in canine patients is commercially available via different laboratories. As an example, the fecal dysbiosis index (FDI) relies on quantitative PCR testing for 8 bacterial taxa in the feces and was shown to accurately reflect changes in bacterial diversity and other health parameters of the gut microbiome. Preliminary evidence suggests that a severely abnormal FDI (6 or above) in dogs with CIE may be a negative prognostic factor.

Procurement of biopsy samples with upper GI endoscopy or exploratory celiotomy may be helpful in further evaluating disease severity in dogs undergoing an in-depth workup. Importantly, there is no direct correlation between the severity of clinical signs and the type or severity of mucosal infiltration, and the impact of histopathologic findings on treatment decisions is questionable in most cases. Therefore, sampling of GI biopsies is not performed as often as it used to be, even in specialty hospitals. The most important justification for histology is to rule out a neoplastic infiltrate such as lymphoma.

Current standardized histopathologic scoring systems are available and take both architectural and inflammatory mucosal changes into account. Inflammatory infiltration may be of varying severity and consist of lymphocytes and plasma cells (lymphoplasmacytic enteritis), eosinophils, neutrophils,

macrophages, or combinations thereof. Examples of small intestinal mucosal architectural changes include villus stunting, surface epithelial injury, crypt distension, lacteal dilation, and mucosal fibrosis.

MANAGEMENT

After endoparasites have been ruled out, the standard approach for a dog with mild to moderate chronic recurrent diarrhea of unknown origin without significant systemic repercussions is to initiate a food trial with a novel protein or hydrolyzed peptide diet. Collecting a detailed dietary history is essential to identify the best-suited diet for each animal.

Many clinicians prefer hydrolyzed diets over novel protein diets to treat their patients with diet-responsive CIE, although there is no evidence to support their superiority. A thorough discussion with the owner about how to implement the dietary trial is essential. During the trial, the dog should not receive any food or treats other than the prescribed diet. All dogs with diet-responsive CIE usually show at least a partial response within 10 to 14 days.

- In dogs with partial or complete response, the dietary trial should be pursued for several months.
- In dogs with continued mild to moderate clinical signs that do not respond to the elimination diet, the next step usually consists of a second food trial with a different novel protein or hydrolyzed peptide diet. Based on recent studies, up to 3 consecutive dietary trials have been recommended for dogs with intestinal disease with no or only minor systemic impact. Generally, the 2nd or 3rd trial should consist of a commercially available, extensively hydrolyzed diet such as Purina Elemental® or Royal Canin Ultamino®.
- In refractory cases and in dogs that did not initially qualify for a dietary trial due to moderate to severe disease with systemic impact (e.g., PLE), a more thorough workup with acquisition of a minimal database and targeted additional testing should be initiated (see diagnosis above). The next therapeutic options include modulation of the gut microbiome using prebiotics, probiotics, or fecal microbiota transplantation, or modulation of mucosal inflammation with glucocorticoids and other immunosuppressants (cyclosporine, chlorambucil, and others).

Use of antibiotics to modulate the gut microbiome is discouraged due to their often lasting negative impact on microbiome recovery. Other microbiome modulation techniques include the use of probiotics, the administration of prebiotics/dietary fibers (see other presentation at this conference), and fecal microbiota transplantation (see other presentation at this conference).

The benefits of probiotics in the management of dogs with CIE have yet to be fully elucidated. Recent studies using highly concentrated multi-strain probiotics (e.g., 225 billion CFU per 10 kg body weight) suggest that these products may contribute to treatment success in dogs with moderate to severe forms of CIE that failed other treatment trials. There is no evidence documenting the value of less concentrated multi-strain or single-strain probiotics in the clinical setting. However, there is a need for additional clinical studies to provide clearer evidence of how to best use probiotics in dogs with CIE.

Immunosuppressive treatment is a reasonable option to manage dogs with CIE that have failed all other trials. Immunosuppressives are also commonly used in sick dogs with severe CIE and dogs with PLE. Initial treatment consists of prednisone or prednisolone at 2 mg/kg q24h. For dogs > 25 kg, a daily dose of 50-60 mg/m^2 body surface area is recommended, with a maximum daily dose of 60 mg/dog. If the dog improves, the dose can be slowly decreased after 2-4 weeks by approx. 25% every 2-4 weeks until the smallest dose that controls the disease is identified. Addition of other immunosuppressives, such as cyclosporine and chlorambucil, may be necessary in severely affected, prednisone-refractory cases, or to allow a quicker decrease of the steroids over time (particularly in dogs with severe prednisone side-effects).

Dogs with severe forms of CIE may show metabolic complications such as vitamin B12 deficiency (hypocobalaminemia) or hypocalcemia due to vitamin D deficiency. These complications occur most commonly in dogs with PLE and moderate to severe hypoalbuminemia. Supplementation of the lacking vitamins is an essential part of these dogs' management.

PROGNOSIS

A recent paper highlights the impact of CIE on the quality of life of dogs and their owners. It provides helpful information to understand how managing a dog with CIE can have extensive repercussions on the life of the adoptive (human) family.

The prognosis for diet-responsive CIE is good. It appears that 77-80% of these dogs remain controlled only with an elimination diet one year after diagnosis. However, several studies showed that the prognosis of dogs with CIE that do not respond to a dietary treatment trial is significantly worse, with up to 1/3 of patients euthanized within 3 months of diagnosis. Moreover, severe hypoalbuminemia (serum albumin < 20 g/l) and deficiency in vitamin D at the time of presentation were both identified as independent negative prognostic factors.

SELECTED REFERENCES

1. Graarup-Hansen Lyngby J, Nielsen LN, Ankerkilde S et al. (2025): Chronic enteropathy in dogs affects the quality of life in both dogs and their owners—are veterinarians proficient in handling the caregiver burden? Frontiers In Vet Sci. https://doi.org/10.3389/fvets.2024.1488917
2. Jergens AE and Heilmann RM (2022): Canine chronic enteropathy—Current state-of-the-art and emerging concepts. Frontiers in Vet Sci. https://doi.org/10.3389/fvets.2022.923013
3. Makielski K et al. (2019): Narrative review of therapies for chronic enteropathies in dogs and cats. J Vet Intern Med. 2019,33:11-22 https://doi.org/10.1111/jvim.15345
4. Rudinsky AJ, Row JC and Parker VJ (2018): Nutritional management of chronic enteropathies in dogs and cats. JAVMA, 253, 570-578, https://doi.org/10.2460/javma.253.5.570
5. Tolbert MK, Murphy M, Gaylord L et al. (2022): Dietary management of chronic enteropathy in dogs. J Small Anim Pract 1-10. https://doi.org/10.1111/jsap.13471
6. Rodrigues SD, Mendoza B, Dias MJ et al. (2025): Association of Diet With Treatment Response in Dogs With Chronic Enteropathy: A Retrospective Multicenter Study. J Vet Intern Med 39 https://doi.org/10.1111/jvim.70071
7. Volkmann M et al. (2017): Chronic Diarrhea in Dogs - Retrospective Study in 136 Cases. J Vet Intern Med., 31:1043-1055 https://doi.org/10.1111/jvim.14739
8. Vuori KA, Hemida M, Moore R et al. (2023): The effect of puppyhood and adolescent diet on the incidence of chronic enteropathy in dogs later in life. Sci Rep https://doi.org/10.1038/s41598-023-27866-z