THOSE TROUBLSOME CHRONIC DIARRHEA CASES
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Many gastrointestinal disorders are either frequently unrecognized or often misdiagnosed. The appropriate recognition of a specific condition and subsequent therapy may result in resolution of the disorder. The purpose of this lecture is to provide important practice tips in the diagnosis and management of various gastrointestinal disorders associated with diarrhea.

Chronic diarrhea is a common complaint and the possible potential etiologies are extensive. Parasites, dietary intolerances, metabolic disease, pancreatic disease, bacterial causes and inflammatory bowel disease are but a few etiologies of chronic diarrhea. Inflammatory bowel disease (IBD) is a common condition diagnosed in the dog and cat however it is not a specific disease but rather is a term given to describe animals having gastrointestinal (GI) signs with histological evidence of inflammation within the intestine. IBD does not however describe the etiology nor does the extent of inflammatory cells parallel severity of clinical signs. Before beginning extensive diagnostics or obtaining an intestinal biopsy in a case having chronic diarrhea there are a few diagnostics and/or trial therapies to consider. Obviously the course of action one should take is predicated in part on a good clinical evaluation and based on the severity of the clinical disease.

Every patient with chronic GI signs should have a thorough history, physical examination, CBC, biochemical profile, urinalysis, and fecal examination. In many cases this initial evaluation will determine if the diarrhea is most likely primary GI or secondary to other systemic or metabolic disease or if the diarrhea is of predominately small bowel or large bowel in origin. For example, Addison’s disease, liver disease and renal disease can all be associated with secondary GI involvement. If the initial work up fails to provide a clue as to the etiology I then begin my specific GI evaluation. The fecal examination should include standard fecal flotation, wet mount prep and stained cytology. A stained Diff-Quick cytology may reveal such things as neutrophils, eosinophils, fungal organisms clostridial spores for example and may provide clues into an etiology. At this time I will also classify the patient as to the severity of disease; minimal signs and debilitation or those cases having severe disease obviously requiring an in-depth GI work up. The animal classified as having relatively mild diarrhea without weight loss or debilitation I prefer to use trial therapy as part of the clinical evaluation. Trial therapy involves parasitic therapy, dietary food trials and antibiotic therapy. When diet, parasites and antibiotic therapy fails to resolve the diarrhea further GI evaluation is indicated and additional diagnostic testing may include such things as imaging studies (ultrasound is preferred as barium studies are rarely helpful) serology (TLI, folate, cobalamin) and endoscopy or surgery for intestinal biopsies.

ALWAYS RULE OUT PARASITES
Parasites must always be considered in any dog having chronic GI signs.\textsuperscript{1} Giardia as well as common nematodes are usually diagnosed using proper fecal examination techniques. Often it is difficult to find giardia cysts on flotation and a more accurate way to diagnose giardia is through fecal ELISA that is highly sensitive and specific. It is important to know that giardia also have antimicrobial sensitivity patterns like bacteria and so it is currently impossible to predict which anti-giardia drug will be effective in individual dogs or cats. The treatment of choice for years has been metronidazole. There are many different doses and duration of therapy reported. I currently use metronidazole at 25 mg/kg bid for 7 days. Toxicity associated with neurological signs occurs at higher doses.

Other suggested giardia therapies include febendazole or febentyl (Drontal Plus™) for 5 days.\textsuperscript{1} High fiber diets may help lessen reinfection when given during the therapy. With treatment failure one should assure that giardia is truly the problem and also that subsequent recontamination is not occurring. Infection with giardia does not confer immunity. In resistant cases combined febendazole and metronidazole therapy has been suggested. In difficult cases bathing the animal prior to therapy and decontaminating the environment using quaternary ammonium compounds is also recommended.

It is controversial whether to treat healthy dogs and cats positive for giardia because giardia is generally not considered to be a significant human health risk. I recommend treating the asymptomatic positive dog and if on recheck evaluation the patient is still positive but subclinical I will repeat therapy using a different agent. If the animal remains positive after 2 therapies I usually throw up my hands and simply recheck the patient again at the next yearly health evaluation. Some animals are chronic asymptomatic carriers and are very difficult to clear. I am more concerned when infected dogs live with immunocompromised individuals or young children.

**Young cats with diarrhea**

The organism *Tritrichomonas foetus* (TTF) has been identified as a cause of chronic diarrhea in young cats.\textsuperscript{2} This organism appears to be genetically similar to that associated with bovine venereal disease. Most of the affected cats are under one year of age and are reported to have watery to sometimes a mucoid diarrhea. It is most often observed in cats from humane shelters or catteries and Abyssinians and Bengal cats appear over represented or have a more resistant disease. There are several ways to diagnose TTF. In some cases the diagnosis can be made by performing a wet mount fecal prep and identifying the organism. A small amount of stool is thinned with some warm saline, a coverslip applied and the feces examined at 40X. It is important that the stool is fresh for examination. A colonic flush of saline has also been described as a means of obtaining fecal material for cytology and culture. TTF is identified by its progressive forward motion (in contrast giardia moves as a falling leaf motion). One can also culture the feces in your practice using the bovine TTF culture technique using an In Pouch TF™ culture method (Biomed Diagnostic Labs). With these pouches a very small amount of stool is placed in the broth and cultured at room air temperature. The bag is then examined under the
microscope 24-72 hours later for evidence of motile organisms. Fecal PCR for TTF is offered by many commercial labs and is considered the test of choice for confirming the organism.

Ronidazole (RDZ) is the only antimicrobial having shown efficacy for treatment of TTF infection.\textsuperscript{3} RDZ is given at 30 mg/kg q24h PO for up to 14 days. RTZ has a very narrow therapeutic range and higher doses or longer duration of therapy can result in neurotoxicity. Ronidazole is not approved for use in the US and must be obtained through a reliable compounding pharmacy. It is very bitter in taste and therefore should be given via capsule; liquid solutions are not recommended. Treatment failure can occur and fecal PCR should be performed in cats failing to respond to therapy because a negative PCR makes TTF less likely the cause of the diarrhea. When left untreated many cats eventually become normal, especially young cats under a year of age. In one study 88% cats with TTF infection were reported to undergo spontaneous resolution of diarrhea within 2 years of diagnosis, however most remained infected based on PCR results when retested as long as 2-5 years after initial diagnosis.\textsuperscript{4} The role of these asymptomatic carriers in disease transmission remains unclear.

WHEN THE DIET WORKS

Over the years I have become more and more impressed with resolution of GI signs by simply changing the diet. My impression and also supported by clinical studies that would suggest that up to 50% of dogs and cats with non-specific GI disease may respond to diet alone.\textsuperscript{5,6,7} Dogs with food responsive diarrhea (FRD) tended to be younger and more have large bowel signs and higher serum albumin concentrations. However, I have recently observed a debilitated and hypoproteinemic patient respond to only a diet change. Consequently, a dietary trial should be the first step in evaluating a patient that has chronic diarrhea after systemic, metabolic and GI parasitic disease is eliminated as a potential cause. Severely debilitated or anorexic patients with GI disease would obviously require more specific and aggressive evaluation. The remainder of patients having chronic diarrhea a dietary trial is justified. A positive response to a diet trial is referred to as a food responsive diarrhea. FRD include both true dietary allergies and dietary intolerances. Allergies result to a reaction with a protein antigen while intolerances occur in response to some substance in the diet such as a preservative or food coloring as examples. Dietary trials using a test diet generally requires a 2-weeks or even less to appreciate a response; the GI seems to respond much faster than dermatologic conditions that may take as long as 8 weeks or more to improve. There is no ideal diet that will consistently resolve diarrhea. The main options for diets include diets that optimize assimilation of nutrients (e.g. highly digestible, fat restricted or low fiber) or diets that favors antigenic modification (e.g. a novel protein source or a protein hydrolysate).\textsuperscript{8,9,10} My personal favorite is the use of the hydrolyzed diets such as Purina HA™. The hydrolyzed diets are single protein sources (usually soy, rice or potato based) and have undergone digestion producing low molecular-weight protein derivatives that are thought to be highly
digestible with low antigenic potential. Perhaps their benefit might actually be due to the fact that they are very pure and contain very little else that might contribute to a dietary intolerance. These diets have now become my initial trial diet. If I observe a positive response then I know I can control the patient’s GI signs with a diet and either continue on that test diet or attempt to find for another long-term diet that works well for both the client and patient. Some recommend if there is a diet response that the patient be feed exclusively only that diet for at least 3 months at which time the diet can then be changed or sometimes even the original diet reintroduce. Only a small percentage of dogs with GI signs (~8%) relapse on challenge and are therefore likely truly food allergic. Novel protein diets containing a single protein antigen would be an alternative approach. If using the novel antigen diets one should only prescribe the veterinary diets as many of the over the counter novel protein diets are not all that novel and have been shown many other antigens not listed on the label. Highly digestible gastrointestinal diets such as Purina EN™ may improve assimilation, promote gastrointestinal health and modify the microbiota. Diets containing highly fermentable fibers such as those containing fructooligosaccharides (also referred to as prebiotics diets) are often useful for colonic disease because fermentation products are shown to have beneficial effects on mucosal function and modify enteric microbiota promoting "good" bacteria and inhibiting certain pathogenic bacteria. If a diet trial is unsuccessful, no improvement in clinical signs after 10-14 days, the next step is to institute an antibiotic trial.

**GI DRUGS AND BUGS**

There are many dogs with chronic large and or small bowel that have an antibiotic responsive diarrhea (ARD). An old terminology for ARD is small intestinal bacterial overgrowth (SIBO). However, SIBO is a poorly defined syndrome in dogs and we currently have no way to adequately and convincingly diagnose bacterial overgrowth or to know in which cases antibiotics would be beneficial short of a therapeutic trial. More recently the term gastrointestinal dysbiosis has been given to conditions associated with abnormal GI bacterial ecosystem. In simple terms GI dysbiosis refers to an imbalance in GI bacteria with the loss of the “good bacteria” coupled with an increase in the so-called “bad bacteria”. The chronic diarrhea cases that do respond to antibiotic therapy it is likely the antibiotics are not specifically eliminating a specific pathogen but rather work by changing the overall bacterial echo system promoting a more normal bacterial make up. Some dogs with gastrointestinal dysbiosis have decreased serum cobalamin (vitamin B12) concentrations. The cobalamin deficiency can be due to lack of intrinsic factor production, abnormal increased intestinal bacterial utilization or ileal disease causing inadequate cobalamin absorption. Serum folate concentrations are usually variable in cases having dysbiosis.

Metronidazole is frequently used in GI cases but long-term administration and potential side effects making it less desirable. Metronidazole has been shown to cause DNA damage to feline lymphocytes in vitro and there is also evidence in laboratory animals that it has some carcinogenic potential. A suggested GI dosage for metronidazole is 7.5-10 mg/kg given bid. A commonly used alternative intestinal antibiotic and my first choice is tylosin. Tylosin was
first reported useful for chronic diarrhea in the early 1970s and more recently has generated resurgence of interest and use. Tylosin (Tylan Soluble™, Elanco) is a macrolide, bacteriostatic antibiotic that is currently marketed over the counter for the treatment of respiratory disease in chickens. Tylosin has activity against most gram-positive and gram-negative cocci, gram-positive rods, and mycoplasma however, but the gram-negative bacteria *Escherichia coli* and *Salmonella spp.* are intrinsically tylosin resistant. Tylosin works by transiently changing the GI enteric bacterial population likely by promoting the growth of beneficial commensal bacteria while suppressing deleterious bacteria. Once tylosin is discontinued the original bacterial population often returns to its pre-treatment state. There is also a suggestion that tylosin may also have anti-inflammatory properties as well. Tylosin appears to have almost no systemic or toxic side effects. The initial dose recommendation for tylosin is 15 mg/kg PO q 12 hr. mixed with the food (has a bitter taste) or given via gelatin capsule. [Note: it comes as a powder and a # 3 gelatin capsule holds 130 mg, #1 capsule holds 240 mg, #0 capsule holds 345 mg and #00 capsules hold 430 mg]. Cases that respond one can reduce the long-term dose to as low as 5 mg/kg q 24 hours. Tylosin is considered by many to be the treatment of choice for suspected Clostridial diarrhea.

**Probiotics**

It is estimated that up to 26% of dogs treated for diarrhea receive alternative therapies usually including probiotics. By definition probiotics are live microorganisms when given in adequate amounts confer a health benefit to the host. The microorganisms most frequently used are lactic acid bacteria (i.e., *Lactobacillus, Enterococcus, Streptococcus,* and *Bifidobacterium spp.*). They are believed to impart a beneficial effect but the mechanism remains poorly understood. Some probiotic strains have been shown to modulate the immune system, others help to restore or normalize the function of the mucosal barrier, or others protect the normal microbiota from pathogenic bacteria through the production of antimicrobial substances or from competitive exclusion of pathogens. To date there have been very few controlled clinical studies evaluating probiotic success. However, in a large double-blinded placebo control study of shelter animals developing diarrhea found significantly fewer cats that received *Enterococcus faecium* (FortiFlora™, 2.1 x 10^9 cfu/day) developed diarrhea for greater than a 2-day duration. Probiotics exert their effects as long as they are being given but once stopped the GI flora generally returns to a state before treatment. It also may seem counterintuitive to give GI antibiotics with probiotics however clinical improvement is often seen when given in combination with each other. Probiotics are considered a safe adjunct therapy for both acute and chronic diarrhea in both dogs and cats as well as for the prevention of stress induced diarrhea. Recommendations of the ideal probiotic containing adequate type and number of viable organisms for specific GI disorders becomes difficult to make. Some over the counter preparations were actually found not contain the label claims. My recommendation is to use a product produced by a reputable veterinary company that has done research on their product.
German Shepherds with Chronic Diarrhea

A clinical syndrome frequently encountered in the German Shepherd dog (GSD) is chronic GI signs and weight loss. Exocrine pancreatic insufficiency (EPI) is common in the breed requiring pancreatic enzyme supplementation and must first be ruled out. The diagnosis is made by documenting a subnormal TLI concentration followed by improvement with pancreatic enzyme replacement. A second group of GSDs are those with similar clinical signs but normal TLI concentrations. Many of these dogs turn out to have an antibiotic responsive diarrhea due to GI dysbiosis. Testing should include measurement of folate and cobalamin (serum B12) concentrations. A low cobalamin and high folate are characteristic of both EPI and GI dysbiosis. Dogs having subnormal cobalamin concentrations will require parenteral supplementation (approximately 500 µcg SQ weekly initially) as part of the therapy. The cause of the GI dysbiosis in the GSD is unknown. Researchers have investigated IgA concentrations suggesting they may have an inherent deficiency leading to altered GI immunity. More recently researchers have measured toll-like receptors (TLR) in the GI tract of these dogs documenting an abnormal expression of the receptors. Using candidate gene analysis, polymorphisms in toll-like receptor TLR 4 and TLR5 were recently shown to be significantly associated with IBD in GSDs. Furthermore, the same polymorphisms in TLR5 were also associated with IBD in a heterogeneous population of dogs consisting of 38 different breeds. These mutations could well play an important role in the pathogenesis of IBD in dogs, as a mutated receptor will lead to misrepresentation of commensal bacteria as pathogens, therefore signaling “danger” to the host and initiating the characteristic inflammatory response seen in this disease. Management of affected GSD involves diet, antibiotics, and cobalamin supplementation. Prebiotics and probiotics are also often given as additional adjunctive therapy. This therapy tends to be a life long management.

WHEN IS IT IBD?

I generally consider IBD as the probable diagnosis when GI parasites have been ruled out and when the patient fails to respond to appropriate dietary and antibiotic trials or if the clinical severity is such that trial therapies should be precluded. The classification of IBD is generally based on the region of the GI tract affected and on the predominant cell type in the inflammatory infiltrate. Lymphocytic-plasmacytic enteritis is the most common type of IBD observed in dogs and cats. Other forms include eosinophilic, neutrophilic and granulomatous enteritis. There are also breed specific forms of IBD best recognized in Soft-Coated Wheaten Terriers having a protein-losing enteropathy (PLE), in Basenjis with an immunoproliferative enteropathy, in Norwegian Lundehunds with IBD and PLE and Boxers having histiocytic ulcerative colitis.

Although the exact etiology of IBD is unknown it is widely accepted that the pathogenesis involves a complex interplay among host genetics, the intestinal mucosal immune system, the environment and the intestinal microbiota. Studies using both histochemical and immunohistochemical techniques to describe immune cell populations and cytokine expression in the intestinal
mucosa have had variable results precluding making a general description of abnormalities in IBD. Histopathology confirms IBD; more appropriately inflammatory changes in the intestine. However, architectural changes (such as villous atrophy) seem to be more important than solely identifying the inflammatory infiltrates present. Also the lesions can be variable throughout the GI tract so multiple biopsies from different areas are suggested.

The diagnosis of IBD requires a complete laboratory evaluation to rule out other diseases. A CBC, biochemical profile, urinalysis and fecal cytology and parasite evaluation is required in all cases. An eosinophilia or hypoproteinemia may provide clues to IBD. Abdominal radiographs or ultrasound may be helpful. However ultrasounds showing increased wall thickness is neither specific or sensitive for the diagnosis of IBD. Specific testing may include measurement of fecal 1-proteinase inhibitor concentrations for documenting GI protein loss and measurement of serum folate and cobalamin concentrations. Cobalamin deficiency is a common complication of feline gastrointestinal disorders and complete improvement in GI function is not possible until cobalamin deficiency is corrected.

An overall impression is that most cases of IBD can be managed but unless the underlying etiology can be identified and removed it can become a long term proposition. A retrospective study demonstrated that only 26% of canine IBD cases progress to complete remission, with intermittent clinical signs remaining in approximately half of cases and 4% were completely uncontrolled with 13% being euthanized because of poor response to treatment. Another study found 8% of the dogs were euthanized because of their disease. Poor prognostic indicators are hypoalbuminemia and hypocobalaminemia.

**Treatment of IBD**

Patients that do not respond to a diet or an antibiotic trial are usually administered glucocorticoids. It is estimated that about 30% of the dogs that fail diet and antibiotics will respond to corticosteroids. Generally oral prednisolone 1-2 mg/kg q 24h PO is given that is then tapered over an 8-week period. However, the side-effects of glucocorticoids can be marked and I never try to exceed a total of 40 mg per day in large breed dogs. Budesonide is a novel glucocorticoid that is reported to have a high first-pass hepatic metabolism and exerts a “local effect” on the intestine with minimal systemic effects. An enteric-coated formulation is used for humans with IBD but a non-enteric coated formulation made by a compounding pharmacy should be used. There is apparent efficacy using budesonide in dogs and cats but the systemic steroid effects are also present and consequently may have no benefit over traditional corticosteroid therapy in most cases. Recommended dose is 1 mg q 24h in cats and toy breeds and up to 2 mg q12h for large breed dogs.

If there is poor response to glucocorticoids after the first 3-4 weeks or if the side effects are severe then I recommend oral cyclosporine at 5-10 mg/kg q 24h, PO for at least 2 months. Many dogs with IBD that are steroid refractive are reported to respond to cyclosporine. In cats, the use of chlorambucil, 2-6 mg/m² q 24h, PO (alternatively 2 mg/cat given 3 times a week) with prednisolone
is preferable if there is inadequate response to glucocorticoid treatment alone. Hematologic parameters should be monitored regularly if chlorambucil is used. Cyclosporine blood concentrations do not need to be monitored regularly, unless there is a suspicion of side effects induced by the cyclosporine treatment or an inadequate response to treatment is observed. If measuring cyclosporine serum concentrations, it is recommended to take blood samples 1-2 hours after giving the medication to ensure that peak concentrations are measured. If the cyclosporine serum concentration is above 700 ng/ml at peak level, then halving the dosage for the first 2 weeks of treatment can reduce the side effects. If the patient responds to cyclosporine, then the medication can either be tapered slowly or stopped after 10 weeks.

Sulfasalazine (20 to 50 mg/kg q 8h, PO for 3-6 weeks) and related drugs are often used in dogs when IBD is limited to the large intestine. However, side-effects include keratoconjunctivitis sicca so tear production should be monitored regularly when using these drugs. I rarely ever prescribe sulfasalazine for large bowel disease as most get better with diet and antibiotics in my experience.

Other novel or adjunct therapy could include omega 3 fatty acids for anti-inflammatory effects and various antioxidants. Probiotics have also been suggested to be beneficial for IBD due to multiple mechanisms described above.

**IS IT FELINE IBD OR LYMPHOMA**

With a failure to respond traditional therapy for IBD in the cat one must consider GI lymphoma. Lymphoma is the most common neoplasia in the cat and approximately 10% of feline lymphomas involve the gastrointestinal tract. GI lymphoma has been classified histologically as B or T cell in origin. The most common is the low grade T cell lymphoma that is typically characterized by the mucosal and sub-mucosal infiltration of small well-defined lymphocytes. These lymphomas are usually FeLV negative. The low-grade lymphocytic form usually responds very well to chemotherapy. The infiltration in the bowel by the tumor generally results in malabsorption. In contrast lymphoblastic (generally B cell origin) usually present as a mass like lesion (most often stomach and colon) and generally has a poor prognosis and will not be discussed further.

Most cats with GI lymphoma (small cell) are middle aged or older DSH cats. Weight loss, vomiting, chronic small bowel diarrhea and progressive inappetance are common features of GI lymphoma. Some cats may only present with weight loss as the chief complaint. On physical examination the bowel may be normal or feel diffusely thickened. Mesenteric lymphadenopathy may also be identified. Less commonly organomegaly may occur.

Routine laboratory testing is often unremarkable or may reveal hypoalbuminemia. Anemia may also be present. Most all cats having GI lymphoma have significantly subnormal serum concentrations of cobalamin. Serum folate concentrations may also be reduced. Some cats may also have concurrent pancreatitis with increases in pancreatic lipase or cholangitis with increased liver enzymes. Ultrasound is useful for evaluating intestinal thickness or loss of normal layering and detecting mesenteric lymphadenopathy. The diagnosis can sometimes be made by demonstrating neoplastic lymphocytes in
aspirates from enlarged intestinal or peripheral lymph nodes. Frequently though, lymph nodes show only reactive changes. Endoscopic visualization and biopsy can enable the accurate diagnosis of many cases of GI lymphoma. Endoscopy biopsies can however miss submucosal and serosal lesions or yield a diagnosis of lymphoplasmacytic enteritis (IBD) because of inadequate tissue. One report suggests that GI lymphoma is more common in ileum than in duodenum and so ileal biopsies are indicated in suspected cases. Because of concurrent liver and pancreatic disease and possible location of tumor in ileum a surgical exploratory may be indicated in some cases.

In a study of 41 cats with low-grade lymphoma, the lymphoma was confined to the gastrointestinal tract in 68% of cats. Eighty-nine percent of the lymphomas were determined to be of T-cell origin via immunohistochemistry, while 8% (3 of 36) were of B-cell origin. Fifty-five per cent of cats achieved a complete response to therapy and 37% achieved a partial response. The majority of cats (76%) received prednisolone at a dose of 5-10 mg, PO, q 12-24 hrs and most (85%) received chlorambucil at a dose of 2 mg, PO, every other day. Overall median remission duration was 948 days. Partial response to therapy was associated with shorter remission duration. Overall median survival time was 704 days. No factors were significantly associated with survival time. Eight percent of the cats experienced no response. Hypocobalaminemia was found in 78% of cats tested and supplemental cobalamin (250µg SC q weekly) should be given as required. IV pulse chlorambucil or other chemotherapy may be also tried however in most cases every other day oral therapy is often easier for the client.

The differentiation of IBD and lymphoma histologically can sometimes be difficult and it is also possible IBD may progress to lymphoma over time. PCR evaluating for clonality PARR (neoplastic cells have one basic clone type) my help and is thought to be about 90% accurate for detecting lymphoma. Fresh tissue from a biopsy or an unstained histology slide is needed for this test. PARR is available at Colorado State University (http://www.cvmbs.colostate.edu/ns/departments/mip/cilab/faq_parr.aspx). More recently a blood test measuring thymidine kinase activity used to distinguishing feline IBD from GI lymphoma has become commercially available. Increased thymidine kinase activity results with proliferative neoplastic disease. As yet there is limited information on this test.

References:
6. Allenspach K, Wieland B, Gröne A, Gaschen F. Chronic enteropathies in dogs:

**Drugs for the treatment of giardia.**

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<tr>
<th>Drug</th>
<th>Dose</th>
<th>Comments</th>
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<td>Metronidazole</td>
<td>15-25 mg/kg q24h for 5-7 days PO</td>
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<td>Febendazole</td>
<td>50 mg/kg q 24h for 5 days PO</td>
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