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Treatment Options for Gastrointestinal Disease in Cats

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There is a cynical view that all cats with chronic vomiting and/or diarrhea have inflammatory bowel disease (IBD) and should be treated with steroids. Whilst not denying that IBD is relatively common, such an approach is simplistic, prone to error, and potentially harmful. It is important to reach as accurate a diagnosis as possible so that treatment can be tailored to the individual cat's needs. However, it is also true that the armamentarium for treating GI disease in cats is limited, and it is important that the clinician uses every available modality to ensure successful treatment, including diet.

DIET

Dietary therapy is an essential part of any treatment, but can present more problems in cats than dogs with their less fastidious appetites. With our greater understanding of the unique characteristics of feline nutritional requirements, the dangers of home-cooked diets or feeding inappropriate foods such as baby food are now recognised. There are now specific commercial diets available with adequate protein and taurine content.

Digestibility

If bowel function is compromised then a highly digestible diet is likely to be beneficial, reducing the "workload" with which an already compromised intestinal mucosa must cope. In mild cases of malabsorption such "hypoallergenic diets" can be sufficient to abolish signs of diarrhea even without resolution of the IBD. Furthermore, improved digestibility is likely to enhance gastric emptying and reduce the frequency of vomiting in chronic gastritis and IBD.

Low fat diet

Fat restriction may reduce the tendency for bacterial fermentation of luminal fat especially if there is steatorrhoea and thereby reduce the volume of diarrhea. However, fat restriction is likely to reduce palatability. Also fat is the densest source of calories, and if food intake is restricted either by disease or palatability, then fat restriction may cause weight loss. This is usually undesirable in cats with GI disease as intestinal integrity deteriorates in malnutrition. Furthermore there may be malabsorption of fat-soluble vitamins (A, D, E, K) and vitamin K responsive coagulopathy has been documented in cats with fat malabsorption.

Exclusion diet

Restriction of the protein and carbohydrate sources to single foodstuffs allows production of a true exclusion/elimination diet. This can be fed as the sole source of food for a trial period to determine whether the GI signs are due to dietary sensitivity. A recent study by Guilford indicated that 20% of cats with chronic GI signs ultimately respond to an exclusion diet. This study didn't prove a food allergy as follow-up biopsies were not taken to show resolution of the problem, but clearly indicates the value of dietary manipulation.

It has been claimed that a home-cooked diet trial is preferable for an exclusion diet as all the ingredients can be controlled and that the any nutritional deficits are unlikely to be harmful during a 3-week trial. However, it is now clear that some cats can take 10 weeks to completely respond and so unbalanced diets cannot be recommended. Furthermore, commercial exclusion diets will be nutritionally balanced and are more likely to elicit compliance both from the owner, through its convenience, and from the cat, because of its palatability. The chance of a dietary sensitivity to a food additive in a particular commercial cat food compared to its home-cooked equivalent is too small to be the first concern.

Of greater concern is that cats will actually eat the exclusion diet, and that they can be stopped from eating other foods next door or hunting. A number of palatable exclusion diets based on chicken, venison, and fish, usually with rice as the cereal source are available. Corn and potato starch are other suitable carbohydrate sources. Barley is theoretically a poor choice as it contains proteins that are antigenically similar to wheat gluten. However, in cats most reports of dietary sensitivities are to chicken and fish and not gluten. No single brand can be recommended as the most appropriate diet depends on the cat's dietary history and its preferences.

Hydrolysed diets

These diets are pre-digested so that native proteins are broken down into short peptides that are hopefully non-antigenic. They are proposed as a diet that is useful in identifying and treating food allergies.

However, completely non-antigenic peptides would have to be very short, and this unfortunately often imparts a bitter taste. Thus a compromise is usually reached with hydrolysed diets having average molecular weights of 5000 to 13000 daltons. These molecules are potentially antigenic, and indeed, they may contain antigenic epitopes that are normally exposed during digestion and to which the cat is actually sensitised. Alternatively, the hydrolysis may have destroyed the important epitopes, and the diet will be efficacious. An alternative proposed mechanism of action is that the small peptides are unable to cross-link the IgE molecules on sensitised mast cells. Whilst this is theoretically feasible, the diets would only be effective in type I hypersensitivities. Thus the concept of hydrolysed diets is novel, but proof of efficacy and not just anecdote is still awaited.

Nutritional supplements

The cat's essential requirement for taurine is well recognised, and this and other essential micronutrients are catered for in all commercial diets.

In cats with pancreatic or intestinal disease there may be cobalamin and vitamin E deficiency. Oral supplementation with vitamin E (100 IU per 5 kg per day) is recommended. Vitamin B_{12} must be supplemented by subcutaneous injection. Cats have limited body stores of cobalamin and can become rapidly cobalamin depleted. Cobalamin deficiency is metabolically significant, causing methyl malonic aciduria, can impair the integrity of the intestinal mucosa, and have a significant appetite suppressant effect. Therefore, cobalamin deficiency alone may perpetuate the clinical signs of GI disease, and supplementation is considered to be most important. A dose of up to 1 mg once every two weeks and long-term monitoring are recommended for effective treatment of feline exocrine pancreatic insufficiency and IBD.

The ratio of n3:n6 fatty acids in the diet is considered important, and a ratio of 1:5 is recommended for its anti-inflammatory activity. However, there is as yet virtually no evidence that this is of significant value in intestinal inflammatory diseases.

Finally, the presence of some fiber is considered essential; fiber-free diets cause deterioration of the intestinal mucosa and diarrhea. An insoluble fiber such as wheat bran and cellulose provide bulk, retaining some water and making a stool of desired consistency. Partially fermentable fibers (e.g. beet pulp, ispaghula) are broken down to volatile fatty acids that enhance colonocyte viability. Highly fermentable fibers, such as gums, in excess amounts are likely to cause flatulence and diarrhea.

TUBE FEEDING

It is now well recognised that the intestine obtains most of its nutrition from the gut lumen and that starvation compromises the intestinal mucosal integrity and, in cats, can predispose to hepatic lipidosis. Therefore the maxim that "if the gut works, use it" is most apposite for cats.

However, in many GI diseases cats can't or won't eat. Tube feeding via naso-esophageal, esophagostomy, gastrostomy and enterostomy tubes should always be considered in all anorexic cats. The choice of tube depends on the site of the lesion and the amount and duration of feeding required. The type of formula used will depend on the route of feeding, the tube diameter, the functional state of the GI tract, and the patient's requirements. Patients with naso-esophageal and enterostomy tubes are limited to liquid enteral diets.

Most human liquid formulas contain less than 20% protein and lack sufficient taurine, precluding their long-term use. Higher protein (up to 30%) formulas do exist but still require supplementation with 250 mg taurine per 8 fl oz. Veterinary enteral diets are available and do not require additional supplements. Most liquid diets provide 1 kcal/ml, but commercial petfoods can be blenderized for feeding through esophagostomy and gastrostomy tubes.

Meals can be given as boluses 4–6 times per day after warming to room temperature. However, bolus feeding may provoke nausea, abdominal cramps, vomiting and diarrhea, and trickle feeding from a syringe pump can be tried. An initial flow rate of 1 ml/kg/hr is used. Every eight hours the infusion is stopped and the residual food retrieved by suction. If the residual volume exceeds twice the volume infused in one hour, feeding should be suspended for 2 hours and then re-instituted at 75% of the previous rate. Metoclopramide (1 mg/kg/day continuous infusion) may help stop vomiting and enhance gastric emptying.

SPECIFIC THERAPIES VERSUS SUPPORTIVE CARE

In many cases of acute vomiting and diarrhea, the problem is self-limiting and only fluid and electrolyte support is necessary, with no specific diagnosis or treatment. Hypokalemia is likely to develop in GI disease through anorexia and GI losses, and potassium supplementation is often required

However, if a definitive diagnosis can be a made, a specific treatment is then instituted. Foreign bodies, linear foreign bodies, intussusception etc are all treated surgically and are not discussed.

BACTERIAL INFECTIONS

Helicobacter

Gastric infections with spiral organisms (*Helicobacter heilmanii*, *H. felis* and perhaps *H. pylori*) are frequently found in cats, but their significance remains unclear. If organisms are found in association with gastritis then triple therapy is recommended although the evidence for successful treatment is lacking. The combination of two antibiotics such as amoxicillin or a macrolide (clarithromycin, azithromycin) and metronidazole with an acid blocker (e.g. ranitidine, famotidine or omeprazole) is considered most effective at eliminating these organisms.

Enteric pathogens

Small intestinal bacterial overgrowth has never been documented in the cat, perhaps because of the high number of organisms found normally. Enteric pathogens such as *Clostridium perfringens*, *Clostridium difficile* and *Campylobacter* are considered uncommon causes of GI disease in cats, compared with dogs. Simple antibiotic therapy is indicated: amoxicillin for *Clostridium perfringens*, metronidazole for *Clostridium difficile*, and erythromycin or fluoroquinolones for *Campylobacter*.

Salmonella is a cause of subclinical disease in some cats but probably should not be treated because it may develop carrier status. However, some cats can become bacteremic and even die of septicemia. Parenteral quinolones are most effective in bacteremic cats.

PROTOZOAL INFECTIONS

Giardia

Giardia infection can be a significant cause of GI disease in cats, especially in the cattery setting. Treatment with fenbendazole at 50 mg/kg/day for 3–5 days is generally more effective than metronidazole, which must be used at 25 mg/kg BID, near to its neurotoxic dose in cats.

Toxoplasma

This can cause self-limiting diarrhea in cats but should be treated if pregnant or immunocompromised owners are at risk. The feces should be collected daily until the oocyst-shedding period is complete as it takes several days for the infective spores to form. Administration of clindamycin (25–50 mg/kg q24h PO), sulfonamides (100 mg/kg q24h PO), or pyrimethamine (2.0 mg/kg q24h PO) can reduce levels of oocyst shedding.

Cryptosporidium

Paromomycin has been recommended for cryptosporidiosis. More recently sequential administration of clindamycin followed by tylosin blocked oocyst shedding and resolved diarrhea in one cat with chronic, clinical cryptosporidiosis.

PARASITIC INFECTIONS

Nematode and tapeworm infections are rare causes of significant GI disease but are readily treated with fenbendazole or praziquantel respectively, in conjunction with appropriate flea control measures.

IDIOPATHIC INFLAMMATORY BOWEL DISEASE

Dietary manipulation, as discussed above, is an important part of managing idiopathic IBD. An exclusion diet is often recommended, partly because the underlying cause may be a food allergy, but also because it provides a "sacrificial protein" that will not be important if the cat becomes sensitised to it, as it is not part of the cat's staple diet.

Metronidazole (10–15 mg/kg q 12h) is effective in some cases of feline IBD, but the mainstay of treatment of idiopathic IBD is immunosuppression. Prednisolone is the drug of first choice: 1–2 mg/kg PO q 12h for 2–4 weeks is given and then tapered to q 24h, and ultimately stopping or at least reducing to a maintenance dose EOD after several months.

In cats, steroid side effects are of less concern than in dogs, but there is a risk of diabetes mellitus. Cats that cannot be pilled daily can be treated with intermittent injections of depot methylprednisolone (20 mg per cat IM or SC q 2–4 weeks).

A locally active steroid, budesonide (Rhinocort), has been successful in maintaining remission in cats with IBD at 1mg per day. It produces less hypothalamo-pituitary-adrenal suppression, as it is destroyed 90% first-pass through the liver. The correct dosage, efficacy and safety of budesonide await further evaluation.

If the prednisolone dosage can't be reduced without relapse, alternative drugs are introduced for long-term management. Azathioprine (0.3 mg/kg PO SID or EOD) has good steroid-sparing properties, but bone marrow suppression is a significant problem in cats because they have low levels of the enzyme (thiopurine methyltransferase) necessary to degrade the active metabolite of azathioprine. In cats, chlorambucil (2 mg/cat every 4–5 days) may be a safer alternative.

Colitis is rarely an isolated cause of diarrhea in cats and although 5-ASA derivatives (e.g. sulfasalazine) can be used in cats it is rarely indicated. As there is concern about salicylate toxicity in cats, the dose (10–20 mg/kg q12h, or 250 mg total daily dose) is half the canine dose.

ALIMENTARY LYMPHOSARCOMA

Although the prognosis is guarded, cats with alimentary lymphosarcoma respond better to chemotherapy than dogs. The standard COP protocol is often adequate, although some recommend substituting chlorambucil for cyclophosphamide.

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