Chapter 57

Inflammatory Bowel Disease

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"With good digestion all can be turned to health."
George Herbert

CLINICAL IMPORTANCE

The term inflammatory bowel disease (IBD) refers to a group of chronic, idiopathic gastrointestinal (GI) disorders characterized by histopathologic lesions of mucosal inflammation. Each IBD variant is named by the predominant cellular infiltrate within the lamina propria. Currently, IBD is considered the most common cause of chronic diarrhea and vomiting in dogs and cats (Guilford, 1996; Jergens, 1999). The generic term, IBD, encompasses lymphoplasmacytic enteritis, lymphocytic gastroenterocolitis, eosinophilic gastroenterocolitis, segmental granulomatous enterocolitis (regional enteritis), suppurative enterocolitis and histiocytic colitis. The lymphoplasmacytic form is probably the most common type of IBD (Leib, 1997; Craven et al, 2004; Hall, 2005, Hall and German, 2005).

The severity of IBD varies from relatively mild clinical signs to life-threatening protein-losing enteropathies. In particular, the Basenji and Ludenhund breeds may present with a very severe variant that has been termed immunoproliferative small intestinal disease (Breitschwerdt, 1992; Flesja and Yri, 1977; Williams, 1997).

Inflammatory infiltrates may involve the stomach, small bowel and colon. In cats, the stomach and small bowel are affected most often. In dogs, IBD is common in both the small and large intestines. In many cases, multiple segments of the bowel are involved and clinical signs may be mixed, reflecting the broad distribution of mucosal lesions.

PATIENT ASSESSMENT

History and Physical Examination

The most common clinical signs in dogs and cats with IBD are chronic vomiting, diarrhea and weight loss. The predominant GI sign varies with the portion or portions of bowel affected. Vomiting tends to be the predominant clinical sign when the stomach and proximal duodenum are affected. Loose, fluid or steatorrheic stools are most common when the small intestine is involved. Diarrhea marked by tenesmus, mucus and small scanty stools is noted with colonic lesions. Clinical signs may be intermittent or persistent. Clinical signs tend to increase in frequency and intensity as IBD progresses temporally. The presence of systemic signs is also variable. Some animals present with a history of depression, malaise and inappetence. Others are alert and active at the time they are examined.

The frequency and character of the vomitus and stools are important features. At times, vomiting will be temporally related to food intake and the vomitus will contain food particles. In other cases, animals may vomit only fluid or froth. Owners should be questioned closely about the appearance of the vomited material. Dark black or coffee grounds material may indicate gastric ulceration or erosions. The diarrhea may be small or large bowel in origin. The color of the stools should be assessed to determine the presence of GI bleeding.

Physical examination findings in dogs and cats with IBD are variable. Many patients have no abnormalities. Others present

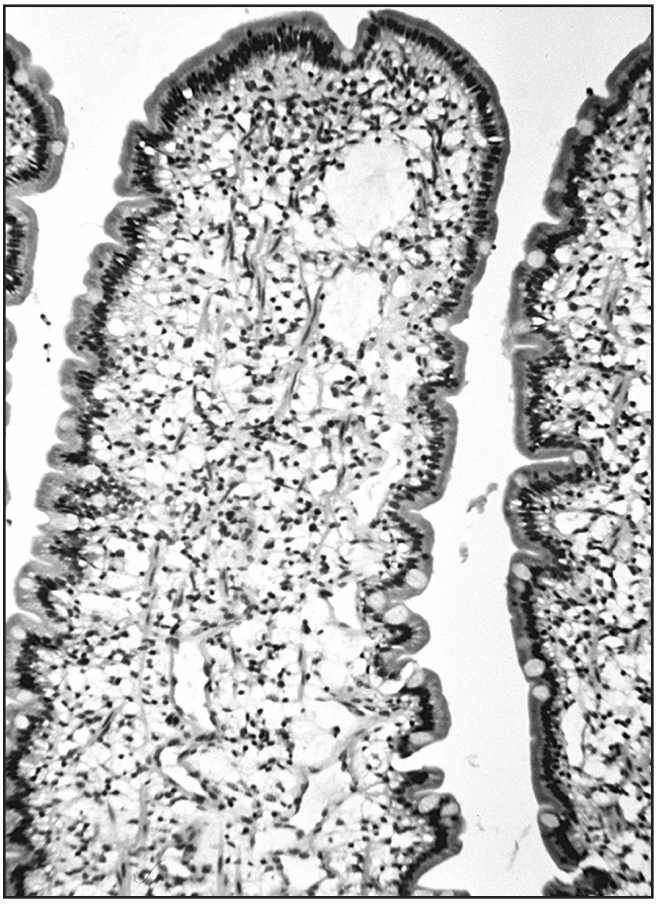

Figure 57-1. Photomicrograph of an intestinal villus showing typical monocellular infiltrates recognized in lymphoplasmacytic enteritis (original magnification 400X).

only with evidence of weight loss and poor body condition. Weight loss may be severe in longstanding cases. Mild peripheral lymphadenopathy may be detected in rare cases of IBD. This finding is most often recognized in cats with eosinophilic gastroenteritis and hypereosinophilic syndrome, which is characterized by multisystemic eosinophilic infiltrates (Moore, 1983).

Occasionally, thickened loops of bowel may be detected by abdominal palpation. This finding is more easily detected in cats. A segmental thickening of bowel may be suggestive of eosinophilic gastroenteritis in cats or granulomatous enteritis in dogs. This finding should also be distinguished from intestinal intussusceptions, foreign bodies, histoplasmosis and neoplastic lesions. Occasionally, pets with IBD present with abdominal pain, which suggests gastroduodenal ulceration (Jergens et al, 1992; Jergens, 1992).

Evidence of hemorrhage or hypoproteinemia may be noted in very severe cases. A vitamin K-dependent coagulopathy has been reported to occur in animals with marked steatorrhea but is rare. At times, IBD may cause protein-losing enteropathy. When severe, hypoalbuminemia and external manifestations of hypoproteinemia (i.e., pitting edema, ascites) may be present. Surprisingly, some animals with protein-losing enteropathy may present with only mild or no diarrhea.

Laboratory and Other Clinical Information

Laboratory findings in patients with IBD are often nonspecific. Hematologic findings are variable and may include blood loss anemia, anemia of chronic disease and/or eosinophilia. In cats with eosinophilic gastroenteritis and hypereosinophilic syndrome, eosinophil counts may exceed 100,000/µl (Moore, 1983). Patients with chronic diarrhea should be assessed with serum biochemistry profiles and urinalyses to determine the systemic effects of the GI disorder and to rule out concurrent disease. Electrolyte abnormalities, including hypokalemia, may be identified. Hypoproteinemia and hypoalbuminemia may be recognized in severe cases with protein-losing enteropathy. Prerenal azotemia may be present in dehydrated patients. In cats, IBD may be associated with pancreatitis and hepatitis, a syndrome that has been termed triaditis (Weiss et al, 1996; Steiner, 2007). In such cases, neutrophilia, increased hepatic enzyme activities, hyperbilirubinemia and increased serum pancreatic lipase immunoreactivity may be noted. IBD is often associated with a protein-losing nephropathy in soft-coated wheaten terriers. Varying degrees of azotemia and proteinuria are also common in these dogs (Vaden et al, 1998; Littman and Giger, 1990).

Fecal examinations are very important in the evaluation of patients with chronic diarrhea. Multiple fecal examinations using concentration techniques are necessary to rule out parasitism. Radiographic findings in IBD are usually nonspecific and nondiagnostic. Occasionally, thickened bowel loops with fluid and/or ingesta are observed on survey abdominal films. In addition, ultrasonographic examination may reveal enlarged mesenteric lymph nodes, focal thickening of the gut and poor definition of the intestinal wall (Baez et al, 1999).

Endoscopic abnormalities in IBD include mucosal granularity, hyperemia, friability and inability to visualize colonic submucosal blood vessels (Jergens et al, 1992). Multiple biopsy specimens should be collected from several bowel segments because histologic changes may be present despite a normal appearance (Jergens et al, 1992; Roth et al, 1990; Marks and LaFlamme, 1998).

The definitive diagnosis of IBD is based on histopathologic examination of biopsy specimens (**Figure 57-1**) collected by endoscopic or surgical techniques (Wilcock, 1992). Expected findings include lymphocytic and plasmacytic infiltrates within the lamina propria as well as architectural abnormalities such as crypt distortion and villous blunting. Histologic grading systems have been proposed to allow objective assessment of intestinal biopsy specimens and to reduce inter-observer variation (Jergens et al, 1992; Roth et al, 1990; Yamasaki et al, 1996; Willard et al, 2002). Despite the use of formal classification schemes, interpretation of histologic changes can be difficult when the lesions are mild or suggest lymphosarcoma (Roth et al, 1990; Wilcock, 1992; Willard et al, 2002; Evans et al, 2006). The latter finding is a serious concern in cases of lymphoplasmacytic enteritis and lymphocytic enteritis.

Quantification of mucosal inflammatory markers found in colonic lavage fluid (e.g., IgG, nitrite) has been suggested for evaluation of dogs with suspected IBD (Gunawardana et al,

1997). The use of a clinical scoring index for disease activity and measurement of C-reactive protein can provide value for assessing disease burden (Jergens et al, 2003; Garcia-Sancho et al, 2007).

Risk Factors

There does not appear to be an age or gender predisposition for IBD. The condition usually arises in adult dogs and cats, but has been diagnosed in puppies and kittens (i.e., less than six months of age). In people, there is a well-recognized familial tendency toward IBD (Fiocchi, 1998). A genetic influence has also been recognized in some dog breeds: 1) the German shepherd dog, Chinese Shar-Pei and soft-coated wheaten terrier for lymphoplasmacytic enteritis, 2) the German shepherd dog and Irish setter for eosinophilic gastroenteritis, 3) the boxer and French bulldog for ulcerative colitis and 4) the Basenji and Ludenhund for immunoproliferative enteropathy (Table 55-3).

The environment may also play an important role in IBD. Animals maintained in overcrowded, contaminated quarters are at risk for development of parasitic infections, viral and bacterial enteritis and small intestinal bacterial overgrowth, all of which are speculated to play a role in the pathogenesis of IBD. The role of parasites in the pathogenesis of IBD is poorly understood; however, occult parasitism has been suggested as a cause for these disorders. For example, in German shepherd dogs, visceral larval migrans has been linked to eosinophilic gastroenteritis (Hayden and van Kruiningen, 1973). In cats, feline infectious peritonitis has been associated with granulomatous and suppurative enterocolitis (Leib et al, 1986; Tebeau, 2007). In addition, small intestinal bacterial overgrowth has been reported in association with lymphoplasmacytic infiltrates and enteritis (Rutgers, 1996). Cats with IBD have been found to have higher fecal concentrations of *Desulfovibrio* spp. and lower numbers of *Bifidobacterium* spp. as compared to healthy cats (Inness et al, 2007).

Etiopathogenesis

Despite intensive study by veterinary and medical researchers, the pathophysiology of inflammatory bowel disorders is not fully understood (Fiocchi, 1998; Hanauer, 1996; German et al, 2003; Hall and German, 2005). The disorder is undoubtedly immune-mediated, yet the pathogenesis of the various forms of IBD is poorly defined. Increased populations of plasma cells producing IgA and IgG as well as T lymphocytes have been recognized in dogs with IBD as compared to normal dogs (Jergens, 1996 et al, 1999 et al; German et al, 2001). In addition, altered cytokine expression has been demonstrated in dogs with small and large intestinal IBD (German et al, 2001). Abnormal cytokine mRNA expression has been identified within intestinal biopsy specimens from cats with IBD (Nguyen Van et al, 2006).

The fundamental pathway for the development of IBD involves hypersensitivity. However, the underlying cause for hypersensitivity reactions is unknown. Two related theories have been proposed. The first speculates that IBD patients develop a defect in the intestinal mucosal barrier. This loss of mucosal integrity results in increased gut permeability and hypersensitivity responses to antigens that are normally tolerated (**Figure 57-2**) (Guilford, 1996). Alternatively, IBD may result from aberrant immunologic responses to luminal antigens. It has been hypothesized that defects in gut-associated lymphatic tissue (GALT) suppressor function may predispose patients to development of hypersensitivity to normally tolerated luminal antigens (**Figures 57-3** and 31-2 through 31-4) (Guilford, 1996). Parasites, pathogenic organisms, normal gut flora and dietary antigens may all serve as the trigger for these immunologic reactions. Both potential pathways culminate in release of inflammatory mediators. These substances may then further damage the intestinal mucosal surface and set up a vicious cycle of inflammation and loss of barrier function.

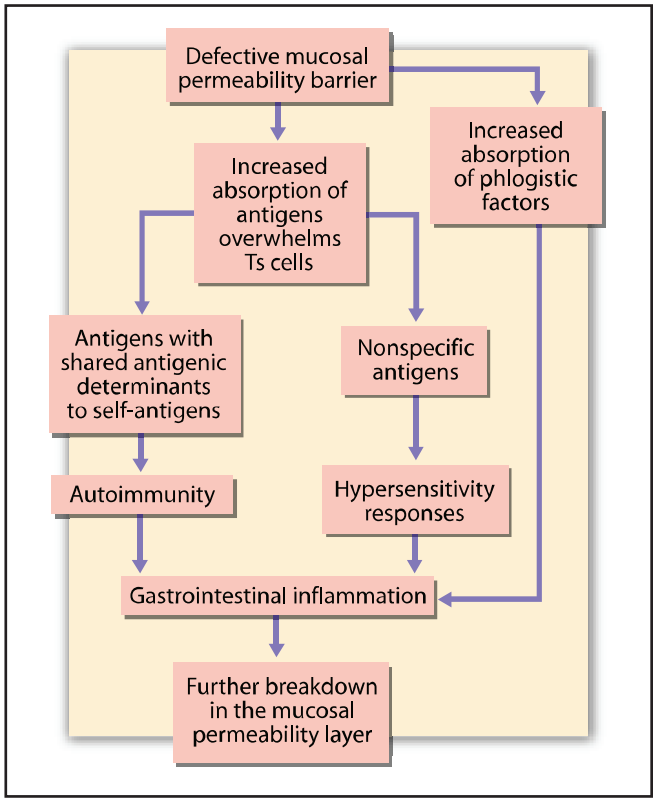

Figure 57-2. A proposed pathway for a defect in the mucosal permeability barrier as a cause of inflammatory bowel disease. (Adapted from Guilford WG. Idiopathic inflammatory bowel diseases. In: Guilford WG, Center SA, Strombeck DR, et al, eds. Strombeck's Small Animal Gastroenterology, 3rd ed. Philadelphia, PA: WB Saunders Co, 1996; 457.)

It is likely that the pathogenetic pathway is influenced by environmental (i.e., exposure to dietary antigens or GI parasites) and genetic factors that modulate disease expression (German et al, 2003). The predisposition for IBD in certain breeds (e.g., Basenjis, soft-coated wheaten terriers) suggests a likely role for genetic influences.

Mucosal inflammatory infiltrates and soluble factors are responsible for the clinical manifestations of IBD. Mucosal

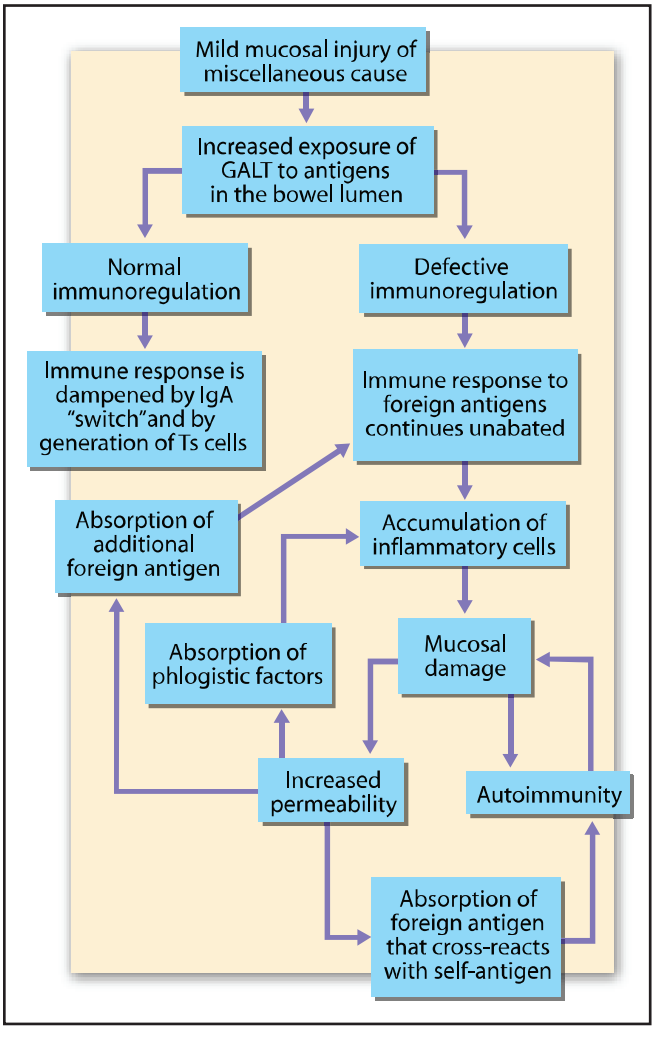

Figure 57-3. A proposed pathway for a defect in the suppressor function of the gut-associated lymphoid tissue (GALT) as a cause of inflammatory bowel disease. (Adapted from Guilford WG. Idiopathic inflammatory bowel diseases. In: Guilford WG, Center SA, Strombeck DR, et al, eds. Strombeck's Small Animal Gastroenterology, 3rd ed. Philadelphia, PA: WB Saunders Co, 1996; 453.)

inflammation disrupts normal absorptive processes resulting in malabsorption and osmotic diarrhea. Altered gut permeability can result in leakage of fluid, protein and blood into the gut lumen. Malabsorbed fats, carbohydrates and bile acids result in secretory diarrhea. Inflammatory mediators may also directly trigger intestinal secretion and mucus production by goblet cells. Mucosal inflammatory infiltrates may alter intestinal and colonic motility patterns, a mechanism attributed to the influence of prostaglandins and leukotrienes on smooth muscle. Inflammation of the proximal bowel (stomach and small bowel) may stimulate visceral afferent receptors that trigger vomiting. Delayed gastric emptying associated with gastroparesis or ileus may exacerbate vomiting.

Key Nutritional Factors

Key nutritional factors for patients with IBD are listed in **Table 57-1** and discussed in more detail below.

Water

Dehydration is a frequent problem in patients with IBD. Reduced water consumption is often aggravated by fluid losses from vomiting and/or diarrhea. Whenever possible, fluid balance should be maintained via oral consumption of fluids. However, dehydrated patients and those with persistent vomiting often need parenteral fluid administration.

Electrolytes

Serum electrolyte concentrations should be assessed regularly to allow early detection of abnormalities as vomiting and diarrhea persist. Hypokalemia is particularly common in patients with IBD. Thus, foods containing 0.8 to 1.1% dry matter (DM) potassium are preferred for dogs and cats with IBD. Initially, potassium levels should be restored with intravenous potassium supplementation. In addition, affected patients often lose large amounts of sodium through fluid feces; however, sodium deficits may be masked by dehydration.

Energy Density and Fat

Energy dense foods are preferred for managing patients with chronic enteropathies. Such foods allow the provision of smaller volumes of food, which minimizes GI distention and secretions. Unfortunately, energy dense foods are also high in fats. High-fat foods may contribute to osmotic diarrhea and GI protein losses, which complicate IBD. Thus, it is often advantageous to initially provide a food with moderate energy density (4.0 to 4.5 kcal/g [16.7 to 18.8 kJ/g] DM) for dogs and cats and fat levels of 12 to 15% for dogs and 15 to 25% for cats DM). Foods with higher fat levels can be offered if the patient tolerates them.

Fiber-enhanced foods typically have lower energy density levels than highly digestible foods because fiber-enhanced foods are usually lower in fat. The DM energy density of fiber-enhanced foods for IBD should be at least 3.2 kcal/g (13.4 kJ/g) for dog foods and at least 3.4 kcal/g (14.2 kJ/g) for cat foods. Fat content for fiber-enhanced foods for dogs and cats with IBD should be 8 to 12% and 9 to 18% DM, respectively.

There appears to be a difference in how dogs and cats are able to tolerate dietary fat in the face of GI disease. Normal cats can tolerate much higher concentrations of dietary fat than dogs (Lewis et al, 1979). Anecdotal information suggests that foods with increased fat content may actually benefit cats with small bowel disease (Guilford, 1996a). Recently, low-fat and high-fat foods were fed to cats with naturally occurring chronic diarrhea in a randomized six-week trial. Fecal scores in more than 65% of cats consuming both high- and low-fat foods improved over the course of the feeding period (Laflamme et al, 2007). The underlying cause of diarrhea in the cats was not investigated. More controlled evaluations are needed to confirm these observations.

Protein

Protein malnutrition may occur in dogs and cats with IBD due to fecal losses. High biologic value, highly digestible (≥87%) protein sources should be used. Protein should be provided at levels sufficient for the appropriate lifestage for patients not experienc-

Table 57-1. Key nutritional factors for dogs and cats with inflammatory bowel disease.*

Factors	Recommended levels
Potassium	0.8 to 1.1%
Energy density	4.0 to 4.5 kcal/g (16.7 to 18.8 kJ/g) for highly digestible foods for dogs and cats ≥3.2 kcal/g (≥13.4 kJ/g) for fiber-enhanced foods for dogs and ≥3.4 kcal/g (≥14.2 kJ/g) for cats
Fat	12 to 15% for dogs and 15 to 25% for cats for highly digestible foods For fiber-enhanced foods: 8 to 12% for dogs 9 to 18% for cats
Protein	≥25% for dogs ≥ 35% for cats If using a limited protein (elimination food) approach, restrict protein to one or two sources and use protein sources to which the patient has not been exposed previously or feed a protein hydrolysate (Chapter 31); also use lower protein levels (16 to 26% for dogs and 30 to 45% for cats)
Crude fiber	≤5% for highly digestible foods (mixed fiber) for dogs and cats 7 to 15% for increased-fiber foods (insoluble fibers are best) for dogs and cats
Digestibility	≥87% for protein and ≥90% for fat and digestible carbohydrate for highly digestible foods ≥80% for protein and fat and ≥90% for carbohydrate for fiber-enhanced foods

*Nutrients expressed on a dry matter basis.

ing excessive GI protein loss (at least 25% for adult dogs and 35% for adult cats [DM]). Suggested protein levels for patients being managed with "hypoallergenic foods" can be lower.

Because dietary antigens are suspected to play a role in the pathogenesis of IBD, "hypoallergenic" novel protein elimination foods or foods containing a protein hydrolysate are often recommended (Nelson et al, 1984; Nelson and Stookey, 1988; Davenport et al, 1987; Guilford, 1996a; Guilford et al, 2001). In some cases, elimination foods may be used successfully without pharmacologic intervention (Hall and German, 2005; Allenspach et al, 2006). Ideal elimination foods should: 1) avoid protein excess (16 to 26% for dogs and 30 to 45% for cats), 2) have high protein digestibility (≥87%) and 3) contain a limited number of novel protein sources to which the patient has never been exposed or contain a protein hydrolysate. Chapter 31 discusses elimination foods in detail. The suspected pathogenesis of IBD involves an increase in gut permeability; therefore, the use of "sacrificial" dietary antigens in the treatment of IBD has been also suggested, but proof of the concept using controlled dietary trials is lacking (Guilford, 1996) (**Box 57-1**).

The evidence regarding the efficacy of elimination foods in people with IBD is conflicting (Husain and Korzenik, 1998). Although specific foods provoking symptoms may be identified in as many as 80% of human patients with Crohn's disease, double-blinded rechallenges suggest that food hypersensitivity may be identified consistently in fewer than 10% (Husain and Korzenik, 1998). Similarly, positive reactions to food antigens applied topically to the gastric mucosa (i.e., gastroscopic food sensitivity test) have been recognized in canine patients with IBD (Vaden, 2000; Vaden et al, 1998a; Guilford et al, 1994; Elwood et al, 1994). Gastroscopic food sensitivity test findings, however, often do not correlate with the results of provocative food challenges or clinical responses (Guilford et al, 1994).[a] A protein hydrolysate-based elimination food has been used successfully in refractory canine IBD cases (Marks and LaFlamme, 1998; Hannah et al, 2000).

Box 57-1. Sacrificial Proteins in Inflammatory Bowel Disease.

Oral tolerance is difficult to maintain in the inflammatory milieu; therefore, animals with inflammatory bowel disease (IBD) are at risk for becoming rapidly sensitive to undigested food proteins entering the lamina propria. This theoretical concern has led to the concept of feeding a "sacrificial protein" source. The first novel protein fed to patients in the early phase of therapy is referred to as a sacrificial protein because it is being offered when the bowel is inflamed and the mucosal barrier porous. The dietary protein source is then changed after the first six weeks of therapy. For animals receiving concurrent prednisone therapy, this diet change is made just before the prednisone dose is decreased from the immunosuppressive to the antiinflammatory range, by which time it is hoped that the mucosal inflammation has been controlled and the mucosal barrier has markedly recovered. As a result, the second dietary protein source is less likely to result in acquired food hypersensitivity and delayed recovery from IBD. The potential benefit of this recommendation is currently under investigation. This type of nutritional management is likely to be of most value in those patients in which IBD has resulted from a transient injury to the gut-associated lymphoid tissue or the mucosal barrier (e.g., from a viral infection) rather than those in which IBD is due to an inherent (i.e., permanent) defect in these structures.

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The Bibliography for **Box 57-1** can be found at www.markmorris.org.

Fiber

It has been recommended that people with IBD eat small quantities of soluble or mixed fiber sources (Fiocchi, 1998). In fact, short-chain fatty acid and butyrate enemas induce clinical improvement in people with ulcerative colitis (Harig et al, 1989; Breuer et al, 1991). A number of substrates including beet pulp, soy fiber, inulin and fructooligosaccharides have been demonstrated by in vitro fermentation to produce volatile fatty acids that may be beneficial in IBD that involves the distal small intestine and colon (Sunvold et al, 1995, 1995a, 1995b; Jamikorn et al, 1999). In addition, these fermentable fibers may serve as prebiotics and foster the growth of beneficial bacterial organisms such as *Bifidobacterium* and *Lactobacillus* at the expense of more pathogenic microbes such as *Desulfovibrio* and *Clostridium* spp. (Chapter 5). These fibers are usually incorporated at rates of 1 to 5% DM in commercial products.

A second approach is to increase dietary fiber content to normalize intestinal motility, water balance and microflora. Fiber has several physiologic characteristics that are beneficial in managing small bowel diarrhea. Moderate levels (7 to 15% DM) of insoluble fiber (e.g., cellulose) add nondigestible bulk, which buffers toxins, holds excess water and, perhaps more important, provides intraluminal stimuli to reestablish the coordinated actions of hormones, neurons, smooth muscle, enzyme delivery, digestion and absorption. Fiber can help normalize transit time through the small bowel, which means slowing a hypermotile state, but also improving a hypomotile state to reestablish normal peristaltic action. However, this level of fiber reduces the energy density and digestibility of a food.

Digestibility

Feeding highly digestible (fat and digestible [soluble] carbohydrate at least 90% and protein at least 87%) foods provides several advantages in the management of dogs and cats with IBD. Nutrients from low-residue foods are more completely absorbed in the proximal gut. Furthermore, these highly digestible foods are associated with: 1) reduced osmotic diarrhea due to fat and carbohydrate malabsorption, 2) reduced production of intestinal gas due to carbohydrate malabsorption and 3) decreased antigen loads because smaller amounts of protein are absorbed intact. Ideal foods for IBD patients are free of lactose to avoid the complication of lactose intolerance. If fiber-enhanced foods are used, the digestibility will be reduced. Digestibility of protein, fat and carbohydrate of fiber-enhanced foods should be at least 80, 80 and 90%, respectively.

The use of monomeric liquid foods and total parenteral nutrition to provide a period of "bowel rest" for people and animals with IBD is controversial (Griffiths et al, 1995; Ling and Griffiths, 2000). Bowel rest has been recommended as a means of reducing or eliminating antigenic stimuli while minimizing GI secretions. The greatest benefit appears to be for human patients with Crohn's disease (Lewis and Fisher, 1994; Jeejeebhoy, 1995). Placebo controlled trials of monomeric foods have not been performed in people but response rates in clinical practice have been convincing (Ling and Griffiths, 2000). Monomeric feedings provide energy and nitrogen in a readily available, nonantigenic form. Monomeric liquid foods are also supplemented with glutamine. In pediatric human patients, a recent meta-analysis demonstrated that enteral nutritional support was as efficacious as corticosteroid therapy in acute Crohn's disease (Henschkel et al, 2000). Parenteral nutrition does not appear to provide any advantage over monomeric foods and is not recommended except in those patients unable to tolerate enteral feeding (Hanauer, 1996). Complete bowel rest may theoretically worsen GI mucosal lesions by depriving mucosal epithelial cells of nutrients such as glutamine and short-chain fatty acids (Husain and Korzenik, 1998). Veterinary experience with parenteral feeding and monomeric and hydrolysate-based foods in the management of IBD is limited (Marks and LaFlamme, 1998; Guilford, 1996a; Hannah et al, 2000). Most often, these therapies have been used in refractory cases in which other therapeutic modalities have failed.

Other Nutritional Factors

Vitamins

Adequate intake of water-soluble and fat-soluble vitamins is critical for patients with IBD. In many cases, the limited stores of water-soluble vitamins have been depleted by diarrheic losses and the large fluid flux through the animal. Thiamin deficiency, in particular, occurs commonly and can profoundly affect appetite. Cobalamin (vitamin B_{12}) deficiency has been recognized in dogs and cats with chronic enteropathies and can result in severe metabolic abnormalities including increased serum methylmalonic acid and disturbances in serum amino acid levels (Ruaux et al, 2001). Dogs and cats appear to more susceptible to cobalamin depletion than people because they have a more rapid cobalamin turnover as a consequence of biliary excretion of cobalamin (Simpson et al, 2001; Simpson, 2003). In addition, dogs and cats lack cobalamin binding protein TC1, which facilitates long-term cobalamin storage in people (Simpson, 2003). Hypocobalaminemia typically occurs when specific cobalamin receptors in the ileum are damaged as a consequence of inflammatory disease (Suchodolski and Steiner, 2003). Deficiency is accelerated by reduced cobalamin consumption and ongoing GI losses. A recent case control study demonstrated that parenteral cobalamin supplementation in cats with undetectable serum cobalamin values (<100 ng/l) normalized serum cobalamin and methylmalonic acid values and improved clinical indices such as body weight, vomiting and diarrhea (Ruaux et al, 2005). For that reason, serum cobalamin should be assessed in patients with chronic small intestinal disease and those with hypocobalaminemia (<300 ng/l) should receive weekly subcutaneous cobalamin therapy (250 μg in cats and 500 μg in dogs) for four to six weeks or until serum levels return to the normal range (Ruaux et al, 2005). Once or twice monthly therapy may be required for longer term maintenance. Disease of the proximal small intestine can inhibit absorption of dietary folate, which is present in foods in the polyglutamate form. Folate absorption requires the jejunal brush border enzyme, folate deconjugase, and specific folate monoglutamate carriers

(Suchodolski and Steiner, 2003). Chronic inflammatory disease of the small bowel can result in low serum folate values due to jejunal mucosal damage, reduced folate absorption and depletion of folate stores.

Loss of fat-soluble vitamins can be significant in patients with steatorrhea (e.g., vitamin K-deficient coagulopathies may occur in patients with IBD). Initially, parenteral administration of fat-soluble vitamins may be necessary. Administering 1 ml of a vitamin A, D and E solution,[b] divided into two intramuscular sites, is simple and cost effective. This should supply fat-soluble vitamins for approximately three months. Vitamin K_1 at a dosage of 0.5 to 1 mg/kg subcutaneously is recommended if a vitamin K-responsive coagulopathy is suspected. Dietary intake of vitamins is often sufficient when the disease responds to treatment and fat absorption is reestablished.

Zinc

Zinc deficiency is well recognized in people as a complication of IBD (Hendricks and Walker, 1988). The small intestine is the primary site of zinc homeostasis and there are several potential mechanisms for zinc deficiency in IBD (**Table 57-2**). In Crohn's disease, oral zinc supplementation improves clinical signs and normalizes intestinal permeability (Sturniolo et al, 2001). Zinc may provide benefits by enhancing brush border enzyme activity, water and electrolyte absorption and regeneration of the gut epithelial surface. Supplemental dietary zinc intake should be considered if dogs and cats with IBD have poor coat quality or dermatitis (Chapters 6 and 32).

Magnesium

Hypomagnesemia has been reported to occur in 30% of dogs and cats hospitalized for GI disorders (Martin, 1994; Toll et al, 2002). Anorexia and malabsorption complicated by the use of magnesium-free fluids are likely causes of low serum magnesium. Magnesium repletion can be accomplished via the use of intravenous fluids.

Omega-3 Fatty Acids

Omega-3 (n-3) fatty acids derived from fish oil or other sources have been hypothesized to have a beneficial effect in controlling mucosal inflammation in IBD. The rationale for the use of omega-3 fatty acids in inflammatory GI disorders first arose from the epidemiologic observation that Japanese and Eskimo populations consuming diets rich in fish sources of these fatty acids have a low prevalence of IBD (Ling and Griffiths, 2000). Some clinical evidence suggests that dietary supplementation with these fatty acids can modulate the generation and biologic activity of inflammatory mediators. More recently, it has been suggested that omega-3 fatty acids may act as competitive agonists of bacterial Toll-like receptor 4 (lipopolysaccharide receptor complex). Because aberrant immune responses to enteric flora have been speculated to play a role in the pathogenesis of IBD, this inhibitory effect may provide another rationale for the use of omega-3 fatty acids in IBD (Lee et al, 2003).

Foods supplemented with fish oil have been used in a limited number of human trials with mixed results (Belluzi et al, 1996, 2000; Mate et al, 1991; Lorenz-Meyer et al, 1996; Lorenz et al, 1989; Stenson et al, 1992). To date, there are no published therapeutic trials investigating the efficacy of omega-3 fatty acid supplementation in dogs or cats with IBD. Although use of omega-3 fatty acids warrants further consideration in veterinary gastroenterology, there is no well-established effective dose for dogs and cats. A reasonable starting dose estimated from human and animal trials is approximately 175 mg (range 50 to 300 mg) omega-3 fatty acids/kg body weight/day.

Table 57-2. Potential causes of zinc deficiency in patients with inflammatory bowel disease.*

Decreased absorption
Intestinal inflammation
Supplemental iron and/or copper
Surgical resection of distal duodenum
Inadequate dietary intake
Anorexia
High fiber or phytate intake
Parenteral nutrition
Increased losses
Chronic blood loss
Increased metabolism
Increased requirements
Growth
Lactation
Pregnancy
Wound healing

*Adapted from Hendricks KM, Walker A. Zinc deficiency in inflammatory bowel disease. Nutrition Reviews 1988; 46: 401-408.

FEEDING PLAN

The justification for nutritional management of IBD is twofold. First, dietary factors may contribute to the initiation or perpetuation of the disease. Second, malnutrition is a common sequela to IBD due to anorexia, malabsorption and increased nutrient losses. Thus, dietary intervention should be aimed at controlling clinical signs while providing adequate nutrients to meet requirements and compensate for ongoing losses through the GI tract. Some dogs and cats with IBD may only require dietary manipulation (Hall and German, 2005; Allenspach et al, 2006). In other cases, dietary therapy is better used in concert with pharmacologic agents. Antibiotics (e.g., tylosin, tetracycline, enrofloxacin, metronidazole), anthelmintics (e.g., fenbendazole) and immunosuppressive agents (e.g., corticosteroids, budesonide, cyclosporine, azathioprine, cyclophosphamide) are often used for managing IBD.

Assess and Select the Food

Selection should focus on foods that reduce intestinal irritation/inflammation and normalize intestinal motility. Three types of foods may be useful in managing diarrhea associated with IBD: 1) highly digestible, low-residue foods formulated

Table 57-3. Key nutritional factors in selected highly digestible veterinary therapeutic foods marketed for dogs with inflammatory bowel disease compared to recommended levels.* (See Table 31-5 if foods with novel protein sources or protein hydrolysates are desired.)

Dry foods	K (%)	Energy density (kcal/g)	Fat (%)	Protein (%)	Fiber (%)	Protein digestibility (%)	Fat digestibility (%)	Carbohydrate digestibility (%)
Recommended levels	**0.8-1.1**	**4.0-4.5**	**12-15**	**≥25**	**≤5**	**≥87**	**≥90**	**≥90**
Hill's Prescription Diet i/d Canine	0.92	4.2	14.1	26.2	2.7	92	93	94
Iams Veterinary Formula Intestinal Low-Residue	0.90	3.8	10.7	24.6	2.1	na	na	na
Medi-Cal Gastro Formula	0.8	na	13.9	22.9	1.9	na	na	na
Medi-Cal Vegetarian Formula	0.8	na	10.5	20.9	3.2	na	na	na
Purina Veterinary Diets EN GastroENteric Formula	0.66	4.2	12.6	27.0	1.5	84.5	91.4	94.4
Royal Canin Veterinary Diet Digestive Low Fat LF 20	0.88	3.7	6.6	24.2	2.3	na	na	na
Royal Canin Veterinary Diets Intestinal HE 28	0.88	4.5	22.0	33.0	1.6	na	na	na

Moist foods	K (%)	Energy density (kcal/g)	Fat (%)	Protein (%)	Fiber (%)	Protein digestibility (%)	Fat digestibility (%)	Carbohydrate digestibility (%)
Recommended levels	**0.8-1.1**	**4.0-4.5**	**12-15**	**≥25**	**≤5**	**≥87**	**≥90**	**≥90**
Hill's Prescription Diet i/d Canine	0.95	4.4	14.9	25.0	1.0	88	94	93
Iams Veterinary Formula Intestinal Low-Residue	0.84	4.6	13.2	35.9	3.9	na	na	na
Medi-Cal Gastro Formula	0.6	na	11.7	22.1	1.0	na	na	na
Medi-Cal Vegetarian Formula	0.7	na	11.5	26.4	1.9	na	na	na
Purina Veterinary Diets EN GastroENteric Formula	0.61	4.0	13.8	30.5	0.9	85.1	95.6	92.2
Royal Canin Veterinary Diet Digestive Low Fat LF	0.74	4.0	6.9	31.9	3.0	na	na	na
Royal Canin Veterinary Diet Intestinal HE	0.80	4.3	11.8	23.1	1.4	na	na	na

Key: K = potassium, Fiber = crude fiber, na = information not available from manufacturer.
*Manufacturers' published values. Nutrients expressed on a dry matter basis. To convert kcal to kJ, multiply kcal by 4.184.

for GI disease, 2) fiber-enhanced foods and 3) elimination foods. Unfortunately, no physical examination finding, laboratory test result or historical fact will dictate which method will be successful in any one patient. Dietary trials are often needed to find which food type works best.

The most commonly used strategy is to feed a highly digestible, low-residue GI food. There are several commercial veterinary therapeutic foods marketed for treatment of GI diseases. **Tables 57-3** and **57-5** list selected highly digestible foods for dogs and cats, respectively, and compare them to the recommended levels of key nutritional factors for IBD. When possible, choose the food that most closely matches the recommendations for key nutritional factors. Recipes for highly digestible homemade foods are also available (Table 10-6). Besides being the most common initial approach for dietary management of IBD, this strategy has also been effective in cats with chronic nonspecific diarrhea (Laflamme and Long, 2004).

A second approach is to increase dietary fiber content to normalize intestinal motility, water balance and microflora. **Tables 57-4** and **57-6** list selected fiber-enhanced commercial veterinary therapeutic foods for dogs and cats with IBD, respectively, and compare them to the recommended key nutritional factors for this approach. These foods typically have a lower energy density and IBD patients may have difficulty maintaining a normal body weight and body condition. Also, foods with 10 to 15% DM fiber usually have lower digestibility. The third dietary option in IBD cases is the use of an elimination food with a limited number of highly digestible, novel protein sources or one containing a protein hydrolysate. Commercial veterinary therapeutic foods (Tables 31-5 and 31-6) or homemade foods that contain novel protein sources often combine lamb, rabbit, venison, duck, fish or game meats with a highly digestible or novel carbohydrate source. All other possible dietary sources of protein and carbohydrate should be eliminated including treats, snacks, table foods, vitamin-mineral supplements and chewable/flavored medications. Clinical signs should abate within the first three weeks of strict dietary management (e.g., feeding only the novel ingredient or protein hydrolysate food). After signs abate, owners may add individual specific ingredients previously fed in an effort to identify the allergen. Clinical GI signs may recur within 12 hours after the offending ingredient is fed. In many cases, owners elect to continue feeding the elimination food if clinical signs abate.

Assess and Determine the Feeding Method

If the patient has a normal body condition score (BCS [2.5/5 to 3.5/5]), the amount of food previously fed (energy basis) was

Table 57-4. Key nutritional factors in selected fiber-enhanced veterinary therapeutic foods marketed for dogs with inflammatory bowel disease compared to recommended levels.* (See Table 31-5 if foods with novel protein sources or protein hydrolysates are desired.)

Dry foods	K (%)	Energy density (kcal/g)	Fat (%)	Protein (%)	Fiber (%)	Protein digestibility (%)	Fat digestibility (%)	Carbohydrate digestibility (%)
Recommended levels	**0.8-1.1**	**≥3.2**	**8-12**	**≥25**	**7-15**	**≥80**	**≥80**	**≥90**
Hill's Prescription Diet w/d Canine	0.70	3.3	8.8	18.9	16.4	84	92	95
Medi-Cal Fibre Formula	1.0	na	10.6	26.2	14.3	na	na	na
Purina Veterinary Diets DCO Dual Fiber Control	0.7	3.7	12.4	25.3	7.6	79.9	80.4	90.6
Purina Veterinary Diets OM Overweight Management	0.83	2.9	7.2	31.1	10.3	81.9	78.9	72.3
Royal Canin Veterinary Diet Calorie Control CC 26 High Fiber	0.9	3.1	10.4	30.9	17.6	na	na	na
Moist foods	**K (%)**	**Energy density (kcal/g)**	**Fat (%)**	**Protein (%)**	**Fiber (%)**	**Protein digestibility (%)**	**Fat digestibility (%)**	**Carbohydrate digestibility (%)**
Recommended levels	**0.8-1.1**	**≥3.2**	**8-12**	**≥25**	**7-15**	**≥80**	**≥80**	**≥90**
Hill's Prescription Diet w/d Canine	0.64	3.5	12.7	17.9	12.4	88	90	92
Medi-Cal Fibre Formula	0.7	na	9.1	24.8	15.0	na	na	na
Purina Veterinary Diets OM Overweight Management	1.06	2.5	8.4	44.1	19.2	80.9	89.8	62.9
Royal Canin Veterinary Diet Calorie Control CC High Fiber	0.82	3.6	12.5	25.9	8.8	na	na	na

Key: K = potassium, Fiber = crude fiber, na = information not available from manufacturer.
*Manufacturers' published values. Nutrients expressed on a dry matter basis. To convert kcal to kJ, multiply kcal by 4.184.

Table 57-5. Key nutritional factors in selected highly digestible veterinary therapeutic foods marketed for cats with inflammatory bowel disease compared to recommended levels.* (See Table 31-6 if foods with novel protein sources or protein hydrolysates are desired.)

Dry foods	K (%)	Energy density (kcal/g)	Fat (%)	Protein (%)	Fiber (%)	Protein digestibility (%)	Fat digestibility (%)	Carbohydrate digestibility (%)
Recommended levels	**0.8-1.1**	**4.0-4.5**	**15-25**	**≥35**	**≤5**	**≥87**	**≥90**	**≥90**
Hill's Prescription Diet i/d Feline	1.07	4.3	20.2	40.3	2.8	88	92	90
Iams Veterinary Formula Intestinal Low-Residue	0.66	3.9	13.7	35.8	1.8	na	na	na
Medi-Cal Hypoallergenic/Gastro	0.8	na	11.5	29.8	3.1	na	na	na
Purina Veterinary Diets EN GastroENteric Formula	0.99	4.4	18.4	56.2	1.3	94.0	93.1	79.7
Royal Canin Veterinary Diet Intestinal HE 30	0.97	4.4	23.7	34.4	5.8	na	na	na
Moist foods	**K (%)**	**Energy density (kcal/g)**	**Fat (%)**	**Protein (%)**	**Fiber (%)**	**Protein digestibility (%)**	**Fat digestibility (%)**	**Carbohydrate digestibility (%)**
Recommended levels	**0.8-1.1**	**4.0-4.5**	**15-25**	**≥35**	**≤5**	**≥87**	**≥90**	**≥90**
Hill's Prescription Diet i/d Feline	1.06	4.2	24.1	37.6	2.4	91	89	91
Iams Veterinary Formula Intestinal Low-Residue	0.93	4.0	11.7	38.4	3.7	na	na	na
Medi-Cal Hypoallergenic/Gastro	1.1	na	35.9	35.5	1.2	na	na	na
Medi-Cal Sensitivity CR	1.1	na	35.1	34.5	2.5	na	na	na

Key: K = potassium, Fiber = crude fiber, na = information not available from manufacturer.
*Manufacturers' published values. Nutrients expressed on a dry matter basis. To convert kcal to kJ, multiply kcal by 4.184.

probably appropriate. If the patient has a low BCS (1/5 or 2/5), the amount of food previously fed may have been inappropriate or significant malassimilation may be occurring due to IBD.

Initially, IBD patients should be fed multiple small meals per day as indicated by their acceptance and tolerance for the food. Meal size can be increased and meal frequency can be reduced as tolerated by the patient after the clinical signs have been successfully managed for several weeks.

REASSESSMENT

Regaining or maintaining optimal body weight and condition, normal levels of activity and alertness and absence of clinical signs are measures of successful dietary and medical management. Serial measurement of the clinical IBD activity index (CIBDAI) offers a more rigorous method of assessing response

Table 57-6. Key nutritional factors in selected fiber-enhanced veterinary therapeutic foods marketed for cats with inflammatory bowel disease compared to recommended levels.* (See Table 31-6 if foods with novel protein sources or protein hydrolysates are desired.)

Dry foods	K (%)	Energy density (kcal/g)	Fat (%)	Protein (%)	Fiber (%)	Protein digestibility (%)	Fat digestibility (%)	Carbohydrate digestibility (%)
Recommended levels	**0.8-1.1**	**≥3.4**	**9-18**	**≥35**	**7-15**	**≥80**	**≥80**	**≥90**
Hill's Prescription Diet w/d Feline	0.84	3.5	9.8	39.0	7.6	90	87	86
Hill's Prescription Diet w/d with Chicken Feline	0.80	3.5	9.9	39.9	7.6	91	85	94
Medi-Cal Fibre Formula	0.9	na	12.2	34.2	14.9	na	na	na
Purina Veterinary Diets OM Overweight Management	0.89	3.6	8.5	56.2	5.6	91.1	87.7	66.8
Royal Canin Veterinary Diet Calorie Control CC 29 High Fiber	0.88	3.3	10.2	33.5	14.0	na	na	na

Moist foods	K (%)	Energy density (kcal/g)	Fat (%)	Protein (%)	Fiber (%)	Protein digestibility (%)	Fat digestibility (%)	Carbohydrate digestibility (%)
Recommended levels	**0.8-1.1**	**≥3.4**	**9-18**	**≥35**	**7-15**	**≥80**	**≥80**	**≥90**
Hill's Prescription Diet w/d with Chicken Feline	0.89	3.5	16.6	39.6	10.6	92	na	na
Medi-Cal Fibre Formula	0.8	na	17.1	40.0	16.7	na	na	na
Purina Veterinary Diets OM Overweight Management	0.91	3.9	14.6	44.6	10.2	87.3	88.6	84.0
Royal Canin Veterinary Diet Calorie Control CC High Fiber	0.77	4.1	21.3	33.5	7.7	na	na	na

Key: K = potassium, Fiber = crude fiber, na = information not available from manufacturer.
*Manufacturers' published values. Nutrients expressed on a dry matter basis. To convert kcal to kJ, multiply kcal by 4.184.

to treatment (Jergens et al, 2003; Garcia-Sancho et al, 2007). Endoscopically obvious gastric and intestinal lesions often respond to therapy (Garcia-Sancho et al, 2007), but underlying histopathologic changes typically remain unchanged (Schreiner et al, 2005; Allenspach et al, 2006; Garcia-Sancho et al, 2007). Laboratory tests to assess serum cobalamin and folate levels are recommended for patients receiving parenteral cobalamin injections for hypocobalaminemia and/or have a history of low serum folate levels to assess the adequacy of and necessity for continued supplementation. The feeding method and amount fed can be adjusted as needed to maintain body weight and condition.

The prognosis for IBD varies with the specific entity present, severity of the condition at the time of presentation and owner compliance. The hypereosinophilic form of eosinophilic gastroenteritis in cats and segmental granulomatous enterocolitis (regional enteritis), immunoproliferative enteropathy and histiocytic colitis in dogs may be refractory to treatment (Breitschwerdt, 1992; Moore, 1983; van Kruiningen, 1967). Likewise, response to therapy may be poor when animals present late in the course of disease and with evidence of protein-losing enteropathy.

In most cases, judicious use of dietary and medical regimens controls the disease. Often, medical measures can be withdrawn after three to six months; thereafter, animals maintain remission with appropriate foods. In some cases, however, pharmacologic treatment may be required for the life of the patient.

The most common causes for failure to respond include noncompliance on the part of the owner and failure of the clinician to tailor a program incorporating dietary and pharmacologic measures for each patient (Guilford, 1996). Intercurrent illnesses such as triaditis in cats, small intestinal bacterial overgrowth or exocrine pancreatic insufficiency may also result in a poor response to treatment. Occasionally, treatment failures occur because of misdiagnosis of alimentary lymphosarcoma or progression of IBD to lymphosarcoma. This progression has been previously reported to occur in dogs (Breitschwerdt, 1982) and cats (Davenport, 1987, 1991).

ENDNOTES

a. Davenport DJ. Unpublished data. 1991.
b. Vital E-A+D containing 100 IU of D and 300 IU of alpha-tocopherol per ml. Schering-Plough Animal Health Corp., Kenilworth, NJ, USA.

REFERENCES

The references for **Chapter 57** can be found at www.markmorris.org

CASE 57-1

Chronic Diarrhea in a Cat

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Patient Assessment

A 10-year-old castrated male domestic shorthair cat was examined for a three-month history of intermittent diarrhea. The owner described the feces as being abnormal, three to five times per week; feces were usually fluid to semi-formed and occasionally black. No tenesmus or blood or mucus in the feces had been noted. The cat had not vomited although the owner felt that its appetite had decreased in the last few days. The cat lived in an apartment and no other pets were in the household.

Physical examination was normal except for mild accumulation of dental calculus and a somewhat "doughy" abdomen. Body weight was 4.4 kg with normal body condition (body condition score [BCS] 3/5). The medical record indicated that a body weight of 4.6 kg was recorded six months previously.

Diagnostic evaluation included a complete blood count (mild eosinophilia, 1,170/µl), serum biochemistry profile (normal), urinalysis (normal), serum T_4 concentration (normal), zinc sulfate fecal flotation (negative for *Giardia* cysts but positive for coccidia ova) and a Sudan black stain for fecal fat (positive). Two weeks of treatment with sulfadimethoxine for coccidiosis improved the diarrhea.

The owner returned with the cat six weeks after completion of sulfadimethoxine treatment because the diarrhea had worsened. The cat was thinner (BCS 2/5) and weighed 3.8 kg. Feces were soft and still positive for fat; however, fecal flotation was negative for coccidia and other parasites. A complete blood count revealed more severe eosinophilia (3,500/µl).

Endoscopic examination of the upper gastrointestinal (GI) tract revealed a normal esophagus and stomach but a coarse, granular, friable mucosa in the duodenum. Histopathologic examination of biopsy specimens collected during endoscopy revealed a normal esophagus, mild lymphoplasmacytic infiltration of the stomach and severe lymphoplasmacytic infiltration in the duodenum. Diagnosis was inflammatory bowel disease (IBD) (lymphoplasmacytic gastroenteritis).

Assess the Food and Feeding Method

The cat was fed a commercial dry grocery brand cat food. The food and water were offered free choice.

Questions

1. Outline a feeding plan for this cat.
2. What other medical therapy can be used in this patient?

Answers and Discussion

1. Several different types of foods may benefit patients with IBD. One strategy involves using a highly digestible, low residue food in conjunction with medical management to control inflammation. (See Answer 2.) Another strategy uses foods with mild to moderate levels of fiber to alter intestinal motility in conjunction with medical management. A third strategy uses an elimination ("hypoallergenic" or one containing a protein hydrolysate) food to decrease mucosal exposure to potential antigens. Although the etiopathogenesis of IBD is unknown, limiting exposure of the GI mucosa to potential antigens is considered an important part of the feeding plan. Use of an elimination food is often the first choice in these cases although a combination of various dietary strategies can also be tried. Access to table food and snacks should be avoided. Therapeutic trials with several different food types and careful monitoring are necessary for optimal case management. The food should be fed in an appropriate amount for the patient's body condition and activity level. For this cat, the daily energy requirement (DER) was estimated to be 1.4 x resting energy requirement for an ideal body weight of 4.5 kg (DER = 290 kcal [1.21 MJ]).
2. Medical therapy is indicated along with dietary management in most moderate to severe cases of IBD. Mild to moderate cases may respond to dietary management alone. Although clinical remission can be obtained in some cases without medical therapy, many gastroenterologists believe that remission will be more rapid, complete and prolonged if the patient is given a short course of antiinflammatory drugs. The rationale for this recommendation is that the more rapidly intestinal inflammation can be controlled, the more rapidly the intestinal permeability barrier will be restored and the less exposure the animal will have to intestinal luminal antigens, including the antigens in the new food. A large variety of medications have been used in cats with this condition including oral corticosteroids, parenteral corticosteroids (i.e., nonresponsive patients with severe disease), azathioprine, cyclophosphamide, metronidazole, tylosin, miscellaneous antibiotics and motility modifiers.

Progress Notes

The owner was offered several therapeutic options but elected to try an elimination food alone. The cat was started on a commercial moist veterinary therapeutic food (Prescription Diet Feline d/d[a]) that contained highly digestible ingredients (lamb and rice) to which the cat had not been exposed previously. The food was offered as two meals per day (one-fourth of a 14.25-oz. can twice daily). Four weeks later, the owner reported that the diarrhea had resolved completely and the cat weighed 4.2 kg. The feeding plan was continued.

The cat did well for more than a year; however, lethargy, vomiting and weight loss were noted 16 months after the initial diagnosis of IBD. Physical examination revealed a thin cat (body weight 3.5 kg, BCS 2/5) with palpably thickened bowel loops. Persistent eosinophilia and elevated liver enzyme activity were present. Evaluation of intestinal biopsy specimens obtained endoscopically revealed GI lymphosarcoma. The cat was euthanized at the owner's request.

Endnote

a. Hill's Pet Nutrition Inc., Topeka, KS, USA. This product is currently available as Prescription Diet d/d Feline.

Bibliography

Dimski DS. Therapy of inflammatory bowel disease. In: Bonagura JD, ed. Current Veterinary Therapy XII. Philadelphia, PA: WB Saunders Co, 1995; 723-728.