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Nutritional management of pancreatitis and concurrent disease in dogs and cats

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ABSTRACT
Nutrition is considered a key part of the management of pancreatitis in dogs and cats. While limited prospective research exists, experimental studies, retrospective studies, and anecdote allow for formulation of nutritional guidelines. Historically, fat has been considered the key nutrient of interest in pancreatitis; however, other nutrients and dietary factors, including energy density, digestibility, protein, carbohydrates, and fiber, are all of importance in these patients. Indeed protein particle size may be of greater significance than dietary fat in the management of pancreatitis in cats. Low-fat gastrointestinal diets are frequently recommended in the initial management of pancreatitis in dogs, while hydrolyzed diets are often considered first-line diets in cats with pancreatitis. The presence or absence of comorbid disease may also alter nutritional recommendations. When diseases occur concurrently, the dietary strategies for the most life-threatening illness, or the illness with the greatest impact on quality of life, is recommended to be prioritized. Many dogs and cats with pancreatitis can be transitioned back to their prediagnosis diet or another commercial maintenance diet, provided that significant comorbid disease is absent. Use of a low-fat diet in the long term may be prioritized in dogs with recurrent episodes of pancreatitis.

Keywords: enteral nutrition, assisted feeding, fat, protein, carbohydrate

Introduction

The role of nutrition in pancreatitis is increasingly recognized in both human and veterinary medicine. Historically, "resting the gut" was proposed to prevent stimulation of pancreatic secretions and thus minimize autodigestion and inflammation. It has now been shown that pancreatic secretions are decreased during pancreatitis, and it is proposed that injured acinar cells are unable to fully respond to physiologic stimuli.[1–3] Thus the principles that underpinned the "rest the gut" theory were based in physiology and are thought to be of reduced significance in clinical disease. Fasting may also have detrimental effects, including intestinal mucosal atrophy, enterocyte apoptosis, gut barrier dysfunction, and bacterial translocation.[4–6] Many of these effects are mitigated via provision of enteral nutrition (EN).[7–10] Nutrition, either enterally or parenterally, has also been shown to reduce risk of death in humans with pancreatitis.[11] It has also been shown to reduce abdominal pain, opioid requirements, and risk of food intolerance as compared to nil per os.[12] When considering route of nutrition, EN is preferred.[13,14] This is likely also true for veterinary patients, leading to a new dogma of "feed the gut."

While many articles have provided information on the medical management of pancreatitis, few have focused on the nutritional management of the disorder, in addition to how this may vary when comorbid disease is present. In this review we aim to describe best practices in the nutritional management of pancreatitis based on data from experimental models, clinical studies, and, where necessary, anecdotal recommendations based on our clinical experience.

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Nutritional Assessment and Detailed Feeding Instructions

To provide nutritional recommendations, a veterinarian must understand each patient's dietary history, its body composition (body condition score [BCS] and muscle condition score),[15] and the presence or absence of comorbid disease.

Management Principles in Dogs and Cats

Pancreatitis—no comorbid disease

Clinical studies on nutritional management of pancreatitis are limited. We are reliant on low-quality

evidence to support our recommendations. These recommendations are based on theory, retrospective studies, and clinical experience. Low-fat diets are frequently used in the initial management of dogs with pancreatitis, while hydrolyzed protein diets are often used in the initial management of cats with pancreatitis. Low- to moderate-fat hydrolyzed diets may also be used in dogs. Our definitions for low, moderate, and high concentrations of each nutrient are provided in **Supplementary Material S1**.

Energy—It is important to meet each patient's daily energy requirement to avoid negative energy balance, particularly in the long term, as it can have deleterious effects on the pancreas due to increased protein turnover. This is of increased importance in cats due to their susceptibility to hepatic lipidosis, which worsens prognosis.[16] In the short term, provision of some nutrition if full energy needs cannot be met should be prioritized over no nutritional intake.

Fat—Noncommercial high-fat diets have been shown to induce and worsen experimentally induced pancreatitis in dogs.[17,18] In contrast, the effect of high dietary fat in naturally occurring disease is less clear, and it is the authors' opinion that fat is overemphasized in the management of pancreatitis.

Previous studies[19,20] assessing ketogenic diets and struvite dissolution diets, some of which are high in fat, demonstrated that 3 of 9 and 2 of 50 dogs developed pancreatitis, respectively. Therefore, it seems prudent to avoid high fat-diets and potentially reduce dietary fat intake, at least initially, in dogs with pancreatitis. Decreasing dietary fat also has the potential advantage of decreasing delayed gastric emptying. This is important because motility support may be a prerequisite to return of normal appetite. Particle size and food volume play major roles in gastric emptying, and focusing on low fat alone may not be sufficient to affect gastric emptying. Some nutritionists recommend reducing dietary fat by 50% of what the dog was originally eating prior to diagnosis of pancreatitis. There is no scientific basis behind this recommendation, but anecdote suggests this approach is effective in many cases. Interestingly, 1 study in healthy dogs did not show a significant effect of dietary fat on serum biomarkers of pancreatic injury (eg, canine pancreatic lipase immunoreactivity), questioning the focus on dietary fat in diets for dogs with pancreatitis.[21] However, it is unknown whether similar results would be found in dogs with pancreatitis. The role of dietary fat is even less certain in cats.[22] Dietary fat should be considered on a case-by-case basis in cats.

Protein—Dietary protein and amino acids are a stimulus for pancreatic secretion in cats, whereas products of protein digestion have been shown to stimulate pancreatic secretions in dogs.[23,24] Therefore, excess dietary protein should be avoided, while the diet should still provide sufficient protein for tissue repair and recovery. In cats, intact protein is more of a pancreatic stimulant than free amino acids.[25] Therefore, hydrolyzed protein diets are often recommended for cats, as they may result in less pancreatic secretion. This may be due to the fact that intact proteins are able to serve as substrates for proteases that break down cholecystokinin-releasing factors.[26] Another advantage of hydrolyzed protein diets in cats is their increased digestibility and reduced antigenicity, the latter of which may be beneficial if chronic enteropathy (CE) is present. It is also important to note that there are limited feline therapeutic hydrolyzed protein diets on the market at this time. Thus, if a cat does not accept this category of diet, some nutrition should be prioritized over strict adherence to the hydrolyzed protein diet.

Carbohydrate—Carbohydrates are less of a pancreatic stimulant when compared to fat and protein.[23,27] Some reduction may be necessary if glucose resistance or overt diabetes mellitus (DM) is present.

Fiber—Diets high in viscous fiber should be avoided in vomiting or regurgitating cases due to their effects on slowing gastric emptying. Prebiotic fibers could be considered to help restore intestinal dysbiosis. Further information on dietary fiber, in general, is available in a recent review article.[28]

Considerations for chronic disease—Chronic pancreatitis (CP) may be differentiated from acute disease in that it is considered a progressive and irreversible process. Nutritional management of CP does not differ significantly from that of acute pancreatitis. It is the authors' opinion that many dogs with acute disease do not require a long-term low-fat diet; however, dogs that are prone to repeated acute bouts of pancreatitis, or CP, may benefit from a long-term low-fat diet. It is also possible, albeit uncommon, to develop DM or exocrine pancreatic insufficiency (EPI) secondary to CP. Further management of these disorders can be found below.

Pancreatitis with comorbid disease

The nutritional management of pancreatitis and concurrent disease may differ from those with pancreatitis alone. A summary of nutritional strategies for the patient with comorbid disease is provided in **Supplementary Material S2**.

Hypertriglyceridemia—Hypertriglyceridemia is an indication to reduce dietary fat. Pancreatic lipases hydrolyze triglycerides into free fatty acids, which when produced in excess can be toxic to pancreatic acinar cells.[29] The addition of triglycerides to a perfused canine pancreas has led to structural changes, suggestive of pancreatic injury.[30] While mild hypertriglyceridemia has been proposed to occur in dogs with pancreatitis, moderate to severe increases in serum triglyceride concentrations do not occur secondary to pancreatitis alone.[31] Dogs with a prior history of pancreatitis are more likely to have increased serum triglyceride concentrations (at pancreatic quiescence) than those without a history of pancreatitis, suggesting that moderate to severe hypertriglyceridemia is a cause rather than consequence of pancreatitis.[32] Therefore, recognizing and instigating dietary management of hypertriglyceridemia

by decreasing dietary fat is paramount. The authors recommend measurement of serum triglycerides in dogs and believe that serum triglyceride concentrations > 600 mg/dL warrant therapeutic management. One study[33] showed that a commercial low-fat diet was effective in reducing serum triglyceride concentrations and correcting lipoprotein profiles in hypertriglyceridemic Miniature Schnauzers. Feeding a therapeutic low-fat diet or selecting a diet with 50% reduction in dietary fat is a reasonable first step. If fasting triglycerides do not return to within normal range, then further decreasing dietary fat is indicated; this may entail feeding an ultralow-fat home-prepared diet formulated by a board-certified veterinary nutritionist. Alternatively, fenofibrate may be added to the treatment protocol.[34]

Studies[35,36] in humans and mice with hypertriglyceridemia have described benefits of using omega-3 fatty acids. One clinical study[37] aimed to evaluate the efficacy of omega-3 fatty acids in Schnauzers with primary hyperlipidemia by feeding one group a low-fat diet and the other a moderate-fat diet, with both groups receiving omega-3 fatty acids for 90 days. Both groups demonstrated decreased plasma cholesterol and triglyceride concentrations; however, the effect was greater in dogs fed the low-fat diet with omega-3 fatty acids. Unfortunately, it was not possible to tease out the individual effects of omega-3 fatty acids. The recommended[38] dose for the treatment of dogs with hyperlipidemia is 120 mg of eicosapentaenoic acid and docosahexaenoic acid/$kg^{0.75}$. In healthy dogs, using medium-chain triglycerides, compared to long-chain saturated or unsaturated fatty acids, decreased postprandial triglycerides.[39]

Obesity—Obesity has been associated with canine pancreatitis and is likely due to the inflammatory effects of adipose tissue.[40] The effect of obesity on feline pancreatitis is unknown. There is a wide range of dietary fat concentrations among therapeutic weight loss diets. Therefore, selecting a lower-fat diet may be considered, especially if dietary fat was suspected to be a predisposing factor in the pathogenesis of acute pancreatitis or if the animal has concurrent hypertriglyceridemia.

Chronic enteropathy—Chronic enteropathy in dogs and cats is subclassified based on treatment response, with food-responsive enteropathy comprising approximately two-thirds of cases.[41] Dietary management of these cases involves trial and error to determine the most effective strategy.[42] The specific nutrient profiles of the different therapeutic diets available for CE vary, and attention to the profiles may help guide specific diet choices in an individual patient.

Beyond ingredients, fat and fiber concentrations may be prioritized in certain cases. Several studies[43–47] have demonstrated the effectiveness of fiber-enriched diets in animals with large intestinal signs, and anecdote suggests similar use in small intestinal disease. Low-fat diets may be beneficial for some cases of CE, for example lymphangiectasia or in cases where there is reduced fat digestion and absorption in the small intestine, resulting in increased passage of undigested fat into the colon and thus secretory diarrhea.[48] As reduced dietary fat is an overlapping nutritional strategy for acute pancreatitis and some CE cases, managing a dog with these concurrent conditions involves prioritizing dietary fat when selecting the specific diet used for CE. Dietary fat does not seem to affect the outcome of cats with chronic diarrhea, and therefore dietary fat may be less of a concern in cats with CE; however, it is important to note that a final diagnosis was not reached in these cats.[49]

Feline triaditis—Feline triaditis describes concurrent pancreatitis, cholangitis, and CE. The reported prevalence in ill referral cats is 17% to 39%.[50–52] While the underlying pathology of triaditis is poorly understood, overall, review articles suggest that the presence of inflammatory disease in the small intestine may be a common precipitating factor.[53] Cats with triaditis can present with vague nonspecific clinical signs, with 63% to 97% presenting with anorexia.[51,54,55] If anorexia is < 3 to 5 days, then nursing management, appetite stimulants, and addressing nausea and pain may help to increase appetite. For those cases where these interventions do not help or anorexia is prolonged, assisted EN should be started. For in-hospital feeding, the authors prefer to feed commercial gastrointestinal diets due to their higher digestibility; then, once the cat is home, a slow transition is made to a commercial hydrolyzed protein diet to address the CE.[53] The delayed introduction of the hydrolyzed protein diet is to prevent food aversion. If a cat does not accept the hydrolyzed protein diet, a slower transition over 2 to 3 weeks may help to improve acceptance, or this strategy can be abandoned to prioritize nutritional intake. Serum cobalamin should be assessed and supplemented if low, and serum potassium concentrations should also be maintained within normal concentrations to help with maintaining or improving appetite.

Feline EPI—Chronic pancreatitis is assumed to be a common cause of feline EPI, although pancreatic acinar atrophy is also reported.[56] Exocrine pancreatic insufficiency leads to a failure of intraluminal digestion and severe nutrient malassimilation. In addition, the lack of other pancreatic secretory products, such as gastrointestinal trophic factors, bicarbonate, intrinsic factor to help with cobalamin absorption, and antimicrobial factors, can result in impaired intestinal function and further nutrient malassimilation. The decrease or absence of gastrointestinal trophic factors and concurrent small intestinal bacterial overgrowth may also result in impaired mucosal enzyme activity, leading to decreased absorption of amino acids, fatty acids, and sugars.[57] Even though oral pancreatic enzyme supplementation is the cornerstone of treatment, EN also appears to be important in the management of EPI.

The most common clinical sign in feline EPI is weight loss.[58] Daily caloric intake should be adjusted to help achieve ideal BCS. Diets with high digestibility are prioritized to help with nutrient digestion and absorption, especially as studies[59,60] have demonstrated the

benefit of such diets in dogs. Caloric density should be maintained, typically by avoiding excessive fat restriction. Higher-fiber diets are avoided, as dietary fiber has been shown to hinder pancreatic enzyme activity in vitro.[61] That said, if soft stool persists, even with enzyme supplementation, increasing dietary fiber may be considered to improve stool quality. The effect of dietary fat in feline EPI is unknown, and cats present less commonly with diarrhea compared to dogs.[58] When possible, cats with EPI should be fed multiple small meals per day in an attempt to maximize digestion and prevent intestinal overload and resultant osmotic diarrhea.[57]

Cobalamin deficiency has been associated with EPI in 77% of cases.[58] Cobalamin supplementation also improves response to treatment.[58] Therefore, supplementation should be initiated as soon as possible in these cases. Supplementation with folate should be considered if low concentrations are documented. While hypofolatemia occurs uncommonly (5% of cases), supplementation should be considered in these cases, as folate deficiency has been shown to inhibit pancreatic exocrine function in rats.[58,62]

Cats with EPI may be deficient in the fat-soluble vitamins A, D, E, and K due to fat malassimilation.[63] If a coagulopathy is documented in a cat with EPI, subcutaneous vitamin K therapy should be initiated. Empirical supplementation of vitamin A and D should be avoided, as excess levels may be harmful.[57] Vitamin E can be dosed orally at 10 IU/kg once a day.[57]

Chronic kidney disease—Optimal nutritional management of chronic kidney disease (CKD) has been shown to slow disease progression and improve survival times.[64,65] Nutritional management is reviewed elsewhere.[66] It can be challenging to manage dogs with CP and CKD since many renal diets are high in fat. In these instances, it may be necessary to feed lower-fat diets that are still appropriately reduced in phosphorus (for CKD) and protein (for proteinuria). This can be achieved by selecting a low- to moderate-fat renal diet or by choosing a diet that may not be marketed for kidney disease but otherwise meets the desired nutrient profile. Selected diets are listed in **Supplementary Material S3**. Similarly, for cats with concurrent CKD and pancreatitis, feeding a lower-phosphorus hydrolyzed protein diet should be prioritized whenever possible.

Diabetes mellitus—A subset of dogs and cats with CP develop DM. A review of nutritional management of DM in cats and dogs can be found elsewhere.[67] In dogs and cats with concurrent pancreatitis and DM, overall feeding strategies will greatly depend on BCS to choose a diet with an appropriate caloric density. For dogs, the presence or absence of moderate to severe hypertriglyceridemia is the next major factor to consider when selecting a diet. For cats with concurrent pancreatitis and DM, feeding a lower-carbohydrate, limited-ingredient diet should be considered. If the clinical signs of pancreatitis are minimal, there may be less priority given to a limited-ingredient diet. Selected diets are listed in **Supplementary Material S3**.

Management of the Hospitalized Dog or Cat

Overview and oral food intake

The authors support the use of early EN in acute presentations of pancreatitis due to high tolerance in both dogs and cats and a faster return to voluntary food intake and reduced rates of gastrointestinal upset in dogs with pancreatitis.[68–70] The duration of inappetence prior to hospitalization should be considered when determining nutrition requirements.

To promote oral food intake, it is important to recognize and treat nausea. Consideration of factors related to the diet, nursing, and environment may help to improve oral food intake. For example, the use of preferred ingredients, textures, and flavors of food; warming the food to increase acceptance; handfeeding; moistening food; and ensuring a stress-free environment should be considered to help increase intake. The authors do not recommend assisted oral feeding due to difficulty meeting daily caloric requirements as well as the risk of aspiration and food aversion.

If animals are not meeting their energy needs within 3 to 5 days, assisted EN should be initiated. On day 1, feeding is often reinstated at 25% of resting energy requirement (RER; 70 X body $weight_{kg}^{0.75}$) and slowly titrated up to full RER over a few days based on tolerance. In the hospital, feeding should not exceed 100% of RER in order to prevent complications associated with overfeeding.[71] For EN, feedings should be spaced out and initially provide no more than 5 to 10 mL/kg of body weight.[72] If pain persists despite analgesia, reducing the rate of feeding, adjusting frequency, reducing fat content, or potentially using supplemental parenteral nutrition (PN) should be considered.

Methods of assisted EN

Nasogastric (NG) or nasoesophageal (NE) tubes are frequently utilized in the initial management of pancreatitis, as they can be placed with sedation and do not require general anesthesia. Complication rates are similar between NG and NE tubes. Nasogastric tubes allow for quantification of gastric residual volume, which may be of benefit in animals with pancreatitis, although this remains controversial and is affected by several factors. A study[69] involving 55 cats with suspected acute pancreatitis fed via an NG tube showed that this was well tolerated, with an overall survival rate of 91%. In that study, vomiting was uncommon (13%), and all cats that vomited had a history of vomiting prior to initiation of feeding. The study also documented a relatively low mechanical complication rate (13%), and low rate (9%) of hypersalivation following tube placement or feeding. One disadvantage of NE and NG tubes is their smaller lumen, which often precludes the use of slurried diets. A range of suitable commercial veterinary liquid diets are available **(Supplementary Material S4)**. Caution is advised when using liquid diets intended for human consumption, as they have a lower

amino acid content and adult formulas may be devoid of essential amino acids such as arginine, which may be detrimental to cats.[73]

An esophagostomy tube can be placed if assisted feeding is anticipated to be needed for longer than 7 days and the animal is stable for general anesthesia. Additionally, an esophagostomy tube may allow for feeding of blenderized dry diets, which may be more energy dense and less costly than commercial liquid diets. Early EN with a commercial low-fat diet delivered proximal to the pylorus via esophagostomy tube was shown to be well tolerated in dogs with severe acute pancreatitis and resulted in fewer complications compared to PN.[68] For those dogs and cats with esophageal disease, percutaneous or surgically placed gastrostomy tubes can be considered. The placement of a jejunostomy tube allows avoidance of the stomach, duodenum, and pancreas, and a study[74] in experimental models of pancreatitis in dogs showed this does not stimulate pancreatic secretions. Two retrospective studies[75,76] in naturally occurring acute pancreatitis, 1 in dogs and 1 in cats, have described the application of these tubes in cases undergoing surgical management. In the canine study,[75] of the 30 dogs that had a jejunostomy tube placed, cellulitis associated with the jejunostomy site was noted in 6, and 1 suffered a major complication of septic peritonitis that was directly related to the tube. Although minimally invasive techniques for placement of nasojejunal tubes using fluoroscopy or endoscopy have been described in dogs, these are not widely used. Interestingly, even though delivering nutrients distal to the duodenum may allow for prevention of pancreatic enzyme secretion and reduction of inflammation, a meta-analysis[77] in humans showed no difference in incidence of mortality, infectious or digestive complications; achievement of energy balance; or length of hospital stay between NG and nasojejunal tube feeding. As NG and esophagostomy feeding tubes have been shown to be well tolerated in cats and dogs with acute pancreatitis, respectively, jejunostomy or nasojejunal tube feedings are not commonly used.[68,69]

Parenteral nutrition

For those animals with intractable vomiting, dull mentation, or coagulopathy, an enteral feeding tube may not be possible, prompting consideration of PN. One study,[78] which evaluated the risk of PN in pancreatitis, among other diseases, reported a mortality rate of 31% for dogs and 19% for cats.

Transitioning From the In-Hospital to At-Home Diet

The amount of RER fed via assisted EN should be slowly decreased as the patient's oral food intake increases. Once an animal is receiving > 50% RER orally and the animal is stable on oral medications, it is a candidate for discharge. To be able to accurately determine food intake, the amount of food should be weighed prior to feeding a patient, and the leftover food should be reweighed after the patient has finished eating. The difference between these values represents the weight of food ingested by the animal and can be used to accurately calculate the percentage of RER ingested. After hospital discharge, owners should be counseled to avoid the risks associated with pancreatitis or its recurrence, including abrupt food change, ingestion of unusual food items, trash, and table scraps, and should address obesity.[40]

Conclusions and Future Outlook

This review article proposes the use of low-fat gastrointestinal diets and hydrolyzed protein diets during the initial management of pancreatitis in dogs and cats, respectively. If a cat does not readily accept the recommended diet, then it may need to be abandoned to prioritize nutritional intake. These recommendations may need to be edited on a case-by-case basis, particularly if comorbid disease is present. While these recommendations are based on the strongest available evidence, there is a lack of available research regarding the nutritional management of naturally occurring pancreatitis in both dogs and cats. The authors hope that this review article will stimulate future prospective studies. These guidelines should continue to be adapted over time as new data become available to clinicians and new dietary formulations come into existence.

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Supplementary Materials

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