G – Gastroenterology

NUTRITIONAL MANAGEMENT OF CANINE PANCREATITIS

Denise Elliott BVSc (Hons) PhD Dipl ACVIM Dipl ACVN

Director of Scientific Communications
Royal Canin USA
500 Fountain Lakes BLVD,
Suite 100
St Charles, Missouri, 63301
USA
denise.elliott@royalcanin.us

Introduction

Pancreatitis is an inflammatory condition of the pancreas that occurs when proteolytic enzymes are activated and autodigestion of the pancreas occurs. The underlying cause of pancreatitis is often unclear, but a number of factors have been implicated, including obesity, the consumption of high fat diets, hyperlipidemia (either idiopathic or dietary), drugs (e.g. phenobarbitol and potassium bromide therapy, azothioprin), toxins (zinc, cholinesterase-inhibitor insecticides, uremic toxins), hypercalcemia, pancreatic duct obstruction, trauma, ischemia/reperfusion injury, and concurrent disease (e.g., hepatobiliary, hyperadrenocorticism, or diabetes mellitus).

Pancreatitis is most common in middle aged to older dogs. The clinical signs vary from mild and or subclinical to severe, necrotizing acute pancreatitis. Chronic or recurrent pancreatitis may ultimately result in exocrine pancreatitic insufficiency and/or diabetes mellitus. Most patients that present with pancreatitis have a history of anorexia, depression, lethargy, vomiting and occasional diarrhea. Vomiting and abdominal pain are the most consistent signs in dogs.

Traditional Management

The medical management of pancreatitis involves decreasing pancreatic autodigestion by decreasing pancreatic enzyme release, maintaining or restoring adequate tissue perfusion, and correcting electrolyte and acid-base imbalances. A key requirement for the management of pancreatitis is to minimize pancreatic enzyme release, and yet provide adequate nutritional support to the patients to minimize protein calorie malnutrition and optimize healing and recovery. To some, these two goals are diabolically apposed hence traditional therapy has focused on nil per os until the clinical sign of vomiting has resolve.

Once the vomiting has resolved, water, particularly in the form of ice cubes is reintroduced. If the patient is able to tolerate oral water, then small amounts of "bland" highly digestible diets, offered multiple times per day, are gradually reintroduced. Pancreatic enzyme secretion is triggered by several gastrointestinal hormones including gastrin, secretin, and cholecystokinin (CCK). CCK is the most potent stimulator of pancreatic secretions. The release of CCK is triggered by long chain fatty acids, amino acids, and hydrogen ions. Carbohydrates appear to have a weak to negligible effect on stimulating CCK release. Therefore, reintroducing a highly digestible carbohydrate source such as rice may be prudent when refeeding the patient. If tolerated, small amounts of high biological value protein such as low fat cottage cheese or boiled skinless chicken breast can be gradually introduced.

At all stages of refeeding, diets or ingredients that are high in fat should be avoided, since fat is the most potent stimulator of CCK secretion. In addition, feeding high fat diets - either commercial or table foods – has been anecdotally associated with pancreatitis. However, what constitutes a restricted fat diet varies considerably. Nutritionists consider a restricted fat diet to be one that contains less than 18% of the energy from fat. Using this recommendation, it is clear that many diets formulated for the management of gastrointestinal disease are not actually low fat diets, and would be inappropriate for the management of pancreatitis.

Enteral versus Parenteral Nutrition

Nil per os therapy can only be instituted for 1-3 days. Patients that have persistent vomiting or severe pancreatitis for longer than 3 days will require nutritional support. There are three modes of nutritional therapy that will minimize pancreatic secretions; partial parenteral nutrition, total parenteral nutrition and jejunostomy tube feeding.

Parenteral nutrition involves the administration

of essential nutrients by intravenous infusion. Parenteral nutrition should be used only when enteral feeding is not possible. Parenteral nutrition is complicated, more expensive and is associated with a high risk of infection and villous atrophy of the small intestine, which may increase the risk of bacterial translocation and sepsis. Total parenteral nutrition solutions are very hypertonic (>1500 mOsm/L) and must be administered into a large central vein to minimize the incidence of phlebitis and thrombosis. Partial parenteral solutions are generally formulated with an osmolality less than 600 mOsm/L and hence may be administered into a peripheral vein. However, because of the dilute nature of PPN, the total daily caloric intake can not be achieved. At best PPN solutions deliver only 50% of the daily illness energy requirement.

Parenteral nutrition solutions are generally formulated with 3-6 grams of protein per 100 kcal, with the energy provided by a ratio of fat (intralipid) to dextrose. There is no evidence to date to suggest that high lipid parenteral nutrition solutions are detrimental in the management of canine pancreatitis. In general, fat-soluble vitamins and trace elements do not need to be added if parenteral nutrition is conducted for less than 1-2 weeks. Vitamin K should not be added to the parenteral nutrient solution, but should be administered subcutaneously once weekly.

The nutrient-rich parenteral solutions provide an ideal growth media for bacteria. To minimize complications with infections, the solutions must be prepared and administered under sterile conditions through a dedicated catheter. Parenteral solutions should always be mixed in the following manner – dextrose, amino acids, and lipid, and refrigerated until use. Parenteral nutrition solutions should be administered for a maximum of 2 days before discarding. It has been recommended to cover the solution with a bag or aluminum foil to protect the amino acids and lipids from light degradation.

Enteral feeding is considered more physiologically sound than intravenous feeding, as it maintains the health of the gastrointestinal tract, and prevents bacterial translocation. In addition, recent studies in humans suggest that enteral feeding is superior to parenteral feeding with lower morbidity and shorter hospitalization. Studies in dogs have clearly demonstrated that jejunal feeding does not exacerbate acute pancreatitis.

Jejunal feeding requires the placement of a feeding tube into the jejunum. This is most commonly achieved via surgical placement. However, there are newer techniques described whereby the jejunum tube is placed transpylorically via a gastrostomy tube. Feeding through a jejunostomy tube must be by a continuous infusion pump due to the narrow diameter of the tube and the volume necessary to meet energy demands. Theoretically, low fat, highly digestible elemental liquid diets would be the first choice for feeding the pancreatic patient with a jejunostomy tube. However, veterinary diets with these specifications are not available. Such diets are available for humans, but care must be given to ensure that these diets provide adequate protein, taurine, and arachidonic acid for feline patients.

Nutritional Management Post-hospitalization

Significant risk factors for the development of pancreatitis in dogs include obesity, hyperlipidemia and dietary indiscretion. Therefore, it is necessary to provide nutritional counsel to the client to reinforce the necessity to remain on a fat-restricted diet, and to avoid high fat foods, including human foods. Hess et al reported that 43% of dogs with acute pancreatitis were overweight or obese. Therefore, prevention of obesity by maintaining optimal body condition should help to reduce the likelihood of pancreatitis. For those patients that are overweight or obese, a weight management plan incorporating nutrition, exercise and behavior modification should be implemented.

References

Chan DL, Freeman LM, Labato MA et al. Retrospective evaluation of partial parenteral nutrition in dogs and cats. J Vet Intern Med 2002; 16: 440-5

Hess RS, Saunders HM, Van Winkle TJ, et al. Clinical, clinicopathologic, radiographic, and ultrasonographic abnormalities in dogs with fatal acute pancreatitis: 70 cases (1986-1995). J Am Vet Med Assoc 1998; 213: 665-670.

Hess R, Kass P, Shofer F, et al. Evaluation of risk factors for fatal acute pancreatitis in dogs. J Am Vet Med Assoc 1999; 214(1): 46-51.

Kalfarentzos F, Kehagias J, Mead N et al. Enteral nutrition is superior to parenteral nutrition in severe acute pancreatitis: results of a randomized prospective trial. Br J Surg 1997; 84: 1665-9.

Qin HL, Su ZD, Gao Q et al. Early intrajejunal nutrition: bacterial translocation and gut barrier function of severe acute pancreatitis in dogs. Hepatobiliary Pancreat Dis Int 2002: 1: 150-4.

Qin HL, Su ZD, Hu LD, et al. Parenteral versus early intrajejunal nutrition:effect on pancreatic natural course, entero-hormones release and its efficacy on dogs with acute pancreatitis. World J Gastroenterol 2003; 9: 2270-3.