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**ACUTE PANCREATITIS: PREVENTION AND TREATMENT**

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**INTRODUCTION**

Pancreatitis is an inflammatory condition of the pancreas that occurs when proteolytic enzymes are activated and autodigestion of the pancreas occurs. A retrospective study revealed significant pancreatic pathologic lesions in 1.3% of 6504 feline necropsy cases and in 1.7% of canine necropsy examinations. 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. phenobarbital and potassium bromide therapy, azothioprin), toxins (zinc, cholinesterase-inhibitor insecticides, uremic toxins), hypercalcemia, pancreatic duct obstruction, trauma, ischemia/reperfusion injury, infection (Herpesvirus, Toxoplasma gondii, feline infectious peritonitis, liver flukes), and concurrent disease (e.g., hepatobiliary, hyperadrenocorticism, or diabetes mellitus).

**CLINICAL PRESENTATION**

Pancreatitis is most common in middle aged to older dogs. Hess et al reported that 43% of dogs with acute pancreatitis were overweight or obese. Miniature Schnauzers, Yorkshire Terriers, Shetland sheep dogs, and Silky Terriers, appear to have an increased risk compared to other breeds. Feline pancreatitis is an emerging diagnosis that has been reported in cats with hepatic lipidosis and inflammatory bowel disease. Siamese cats seem to have an increased risk.

Clinical signs vary from mild and/or subclinical to severe, necrotizing acute pancreatitis. Chronic or recurrent pancreatitis may ultimately result in exocrine pancreatic insufficiency and/or diabetes mellitus. Most patients that present with pancreatitis have a history of anorexia, depression, lethargy, vomiting and occasional diarrhea. There may be a recent episode of dietary indiscretion or drug administration. Clinical signs that were reported in 40 cats with necropsy confirmed pancreatitis included lethargy (100%), anorexia (97%), dehydration (92%), hypothermia (68%), vomiting (35%), and abdominal pain (25%). Therefore, unlike dogs, vomiting and abdominal pain are not consistent clinical signs in cats with pancreatitis.

Findings noted on physical examination are variable and include dehydration, fever and abdominal pain. Less commonly, the patient may be jaundiced, in respiratory distress with shock (tachycardia, prolonged capillary refill time, tacky mucous membranes, and hypothermia). The clinical signs of chronic pancreatitis tend to be waxing and waning anorexia, vomiting, and depression, with or without diarrhea.

Alterations noted on routine hematology and biochemistry panels are helpful in diagnosing pancreatitis, and are necessary to evaluate the severity of concurrent fluid, electrolyte, acid-base and organ dysfunction such as renal azotemia and hepatocellular damage. Changes that may be noted include leukocytosis, hemocooncentration, thrombocytopenia, increased liver enzymes, bilirubinemia, ionized hypocalcemia, hypokalemia, hypercholesterolemia, hypertriglyceridemia, azotemia, and hyperglycemia. Severe acute necrotizing pancreatitis may progress to disseminated intravascular coagulation with thrombocytopenia and prolongation of coagulation times.

Amylase and/or lipase concentrations may be elevated. Although supportive, neither of these enzymes can definitively diagnose pancreatitis. Serum trypsin-like-immunoreactivity (TLI) is much more specific and sensitive for the diagnosis of pancreatitis. TLI measures antibodies against circulating trypsin and trypsinogen. TLI values in the normal range do not rule out pancreatitis, and abnormally elevated concentrations are supported, but not diagnostic for pancreatitis. Pancreatic lipase immunoreactivity assays have been recently developed and may provide a more sensitive diagnostic test for pancreatitis.

Radiographic findings in patients with pancreatitis include loss of serosal detail, increased density in the right cranial quadrant of the abdomen, displacement of the stomach to the left and widening of the angle between the pyloric antrum and proximal duodenum. Abdominal ultrasound is particularly helpful in the diagnosis of pancreatitis. Ultrasonographic findings include an enlarged, hypechoic pancreas, cavitory lesions, dilated pancreatic duct, hypomotile duodenum, biliary dilatation and peritoneal fluid.

**MANAGEMENT**

The management of pancreatitis involves decreasing pancreatic autodigestion by decreasing pancreatic enzyme release, maintaining or restoring adequate tissue perfusion, correcting electrolyte and acid-base imbalances, and providing nutritional support to optimize pancreatic regeneration and repair.

Patients with pancreatitis are often dehydrated secondary to the anorexia, vomiting and diarrhea. An estimation of their initial fluid deficit should be made by assessing body weight, skin turgor, capillary refill time, heart rate, PCV, and TP. The initial fluid deficit should be corrected using balanced polyionic solutions or 0.9% NaCl over 4-6 hours. Further fluid administration should aim to achieve mild volume expansion (3-5%) to maintain pancreatic perfusion. Additional disturbances such as hypokalemia and hypoglycemia should be corrected where necessary. Plasma and colloidal therapy (dextran 70, hetastarch, Oxycobolin) may be indicated for patients with severe hypoproteinemia or shock. Broad spectrum antibiotic therapy may be indicated for patients with shock, fever or evidence of gastrointestinal barrier defects. Pain relief should be provided with injectable opioids or transdermal fentanyl patches.

A key requirement for the management of pancreatitis is to minimize pancreatic enzyme release. Agents that inhibit pancreatic secretion (glucagon, somatostatin) or the intracellular activation of proteases (gabexate mesylate) have been evaluated in experimental models, but can not yet be recommended for clinical application. Oral pancreatic enzyme administration to provide negative feedback inhibition on pancreatic enzyme secretion has been suggested by some authors, but has not yet been proven to be clinically effective. To date, the most effective mechanism to limit pancreatic enzyme release is nil per os therapy.

**NUTRITIONAL MANAGEMENT**

The nutritional goal of managing pancreatitis is to minimize pancreatic stimulation, 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 signs resolve. Water, followed by small “bland” highly digestible diets 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. Studies in dogs and cats have confirmed that CCK and gastrin are involved in triggering pancreatic enzyme secretion. 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 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, anecdotally feeding high fat diets - either commercial or table foods – has been associated with pancreatitis. However, what constitutes a restricted fat diet varies considerably among manufacturers. 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. It is also important to consider the energy content of the diet. Some diets that are low in fat will also be low energy diets, formulated for achieving weight loss in obese dogs. These diets are not appropriate for the critically ill patient that is recovering from pancreatitis.

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. In addition, most cats with pancreatitis often present with evidence of malnutrition such as body weight loss or loss of lean body mass. Therefore, nil per os will worsen the condition of the cat and further hinder recovery from the disease. 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 or feline 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. 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 gastrosomy 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.

Feeding the cat with pancreatitis is a significant challenge. There is a paucity of evidence available to guide an evidence based approach. Clearly, feeding the cat with pancreatitis is a high priority. However, it is less clear what effect dietary fat concentration has on the feline pancreas, and what the preferred method of feeding is. Anecdotal reports have suggested success with nasoesophageal, esophagostomy and gastrostomy tube feeding.

REFERENCES