Animal Sounds

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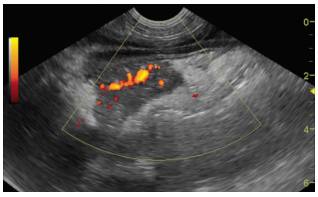
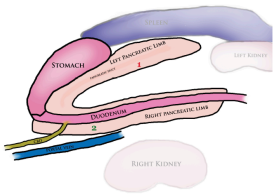
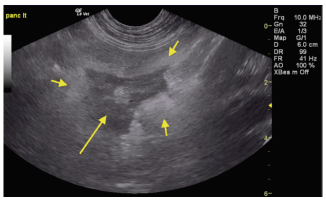
JUNE 2014: Canine Pancreatitis







Description: Pancreatitis continues to be a poorly understood disease process in canine patients. Both acute and chronic forms exist, and each form can entail a range of mild to severe changes. Traditionally, canine patients were thought to be afflicted more commonly by acute pancreatitis; however, recent studies based on pathology samples obtained at necropsy suggest that this may not be accurate. In fact, it may be the case that chronic pancreatitis is up to twice as common as acute pancreatitis.

Clinical Signs: Signs can vary from none in subclinical patients, to multi-organ involvement in more severe forms. Generally, anorexia, vomiting, and weakness are seen in more than three quarters of patients with severe pancreatitis; abdominal pain, dehydration, and diarrhea are seen in one-third to half of patients. Rare patients display neurologic signs, which are referred to as pancreatic encephalopathy. Localized pancreatic inflammation, which typically manifests as vomiting and abdominal pain, is thought to be secondary to trypsin activation; the latter leads to pancreatic edema and necrosis, as well as necrosis of the peripancreatic fat. Systemic effects are now thought to be secondary to other inflammatory mediators that may lead to hypotension, pulmonary edema, and DIC.

Diagnostics: Diagnosis should be based on a multifactorial approach that includes the evaluation of clinical signs, lab work, ultrasonography, and cytology or histopathology. At minimum, one should evaluate a CBC, the biochemical profile, and urinalysis to determine whether other underlying systemic diseases are present. Elevated levels of either amylase or lipase alone are not indicative of pancreatitis, as both of these enzymes are only approximately 50% specific for the pancreas under the best of circumstances. Advances have been made in identifying the origin of pancreatic lipase; the Spec cPL® (Idexx and GI Lab at Texas A&M) is extremely specific and has greater than 80% sensitivity in detecting elevations in lipase secondary to pancreatitis. The Spec cPL® and the Spec fPL® are now the standard laboratory tests for acute pancreatitis in dogs and cats, respectively. These tests are also available as bedside snap tests through Idexx (Snap cPL® and Snap fPL®), but may yield more false positives then the Spec cPL® / fPL®. There is improved specificity with the Spec cPL® test when a cutoff of 400 ug/L is utilized; however, both tests exhibit similar specificity when a cutoff of 200 ug/L is used, which is the lower limit of detection for the Snap cPL®. Not only is ultrasonographic examination essential for evaluating the pancreatic tissue, but it is also important for assessing other organ systems as well. In acute pancreatitis, an ill defined, hypoechoic mass effect surrounded by hyperechoic peri-pancreatic fat is usually detectable in the gastric and/or duodenal pancreatic limb regions. In chronic pancreatitis, the pancreas may become thickened, hyperechoic, or be of mixed echogenicity; it may also exhibit cystic structures (pseudocysts). Because it can be painful, complete ultrasonographic examination of the pancreatic region may require that animals undergo light sedation. Sedation additionally allows for sampling of the pancreas via fine needle aspiration (FNA) or ultrasound-guided biopsy if suspicious tissue has been identified or the case has hitherto been nonresponsive to medical therapy. Gastric or intestinal gas shadows and the position of the pancreas in deep chested breeds can interfere with full evaluation; however, other indications, such as intestinal stasis, duodenal corrugation, bile duct distension, and small amounts

of free fluid, may support a diagnosis of pancreatitis. Pancreatic neoplasia is not often initially differentiated by simple ultrasonographic examination; however, metastatic lesions in the liver or accompanying lymphadenopathy may be indicative, along with age (>10 years) and breed (poodles, spaniels, boxers). Again, aspirates or biopsies are recommended if neoplasia is suspected. The ultrasound can also be repeated, and the appearance of the pancreas can be monitored and assessed for change (one is evaluating improvement versus progressive mass formation to determine whether neoplasia is present).

Treatment: The following is the suggested protocol for hospitalized moderate/severe cases:
NPO for approximately 48 hours or until vomiting ceases, then introduce small amounts of water provided there has been no vomiting and the dog is no longer in pain. When it has been at least 12 hours since there has been any vomiting and/or diarrhea, introduce small amounts of a fat restricted diet. Therapeutic diets such as Hill's i/d low fat®, Hill's w/d®, or Royal Canin LF are appropriate choices. If relapses occur, discontinue oral feeding and pursue parenteral nutrition (PPN), or consider a jejunostomy tube (dogs) or a PEG or esophagostomy tube (cats). The possibility of underlying neoplasia and the need for an ultrasound-guided biopsy should also be considered if it has not yet been performed. Note: Recent data suggests that even shorter periods of NPO are ideal, and trickle feeding of a low fat diet via a nasogastric tube is a better alternative while enterocyte nutrition is being maintained.
Fluid therapy at 1.5 to 2 times maintenance fluid rate with potassium supplementation (20-40 mEq/L based on serum potassium levels) and vitamin B complex. (Note: Attention must be paid to cardiac status when treating with aggressive fluids if there is concurrent cardiovascular disease.) Oncotic pressure enhancement with Hetastarch may be considered as well. Dopamine constant rate infusion (CRI) at 5 ug/kg/min has been shown to enhance perfusion to the pancreatic parenchyma; however, it must be initiated in the first 12 hours of therapy to be of benefit. Selenium 0.3 mg/kg is sometimes added to IV fluids and is a beneficial antioxidant.

Buprenorphine and fentanyl are good choices for pain management. Meperidine (5-10 mg/kg every 2-4 hours IM or SQ) is a potential alternative, as is an IV mixture of ketamine, morphine, and lidocaine.

Antiemetics are very important for minimizing vomiting and thus allowing for enteral nutrition. As such, maropitant citrate (Cerenia) should be administered at 1 mg/kg subcutaneously once daily for up to 5 days; odansetron (Zofran) at 0.11-0.22 mg/kg IV, IM, or SQ; or dolasetron (Anzemet) at 0.2-0.6 mg/kg IV once daily. More traditional antiemetics include metoclopramide (0.2-0.5 mg/kg SC TID or 0.01-0.02 mg/kg/hr CRI) or chlorpromazine (0.25-0.5 mg/kg IM TID/QID). Monitor for excessive sedation if chlorpromazine is administered in conjunction with butorphanol/meperidine.

Fresh frozen plasma administration may be considered to supplement alpha 2 macroglobulins, which bind released proteases from the pancreas (these cause autodigestion of the pancreas and result in inflammation). The plasma will help reduce protease-induced cardiovascular compromise and DIC, and enhance oncotic pressures. It will also help treat hypoalbuminemia; however, large volumes and repeat transfusions are often needed to raise albumin levels. Fresh frozen plasma can be administered at 10 ml/kg/24 hours as needed until the patient has stabilized. Begin transfusion at a slow rate of one-quarter maintenance for 15-20 minutes, then increase the rate to one half maintenance for half an hour and give the rest over 4 hours. Pretreatment with diphenhydramine can be administered at 0.5-1 mg/kg IM.

Antibiotic therapy is controversial yet essential if fever is present or abscessation is suspected based on the ultrasound. Enrofloxacin (Baytril) has been found to penetrate the pancreas well at 2.5 mg/kg bid IV or IM. This drug should be diluted in the IV fluids or given at half-strength dilution with physiologic sodium chloride. Ampicillin (20 mg/kg IV, SC, or PO TID) is a useful broad spectrum antibiotic.
Peritoneal lavage may prove beneficial if marked accumulation of abdominal fluid occurs. Abdominocentesis samples should be analyzed with culture and sensitivity prior to initiating the procedure.

Hypertriglyceridemia is treated with dietary fat restriction—a therapeutic mainstay for pancreatitis. Treating underlying endocrinopathies is also important if they are contributing to hyperlipidemia (ex. hypothyroidism). The addition of an omega-3 fatty acid supplement has been shown to aid further in decreasing overall triglyceride levels (marine fish oils 1000 mg/4.54 kg PO once daily or 120 mg EPA/DHA/kg0.75). If triglyceride reduction has not been optimized within 4 weeks (target <400mg/dl), then oral niacin (100 mg/day/dog) or gemfibrizol (200 mg/day/dog;

10mg/kg BID for cats) may be tried. It is important to monitor side effects such as erythema, pruritis, abdominal pain, GI symptoms, and altered liver function.

Prednisone can be considered in non-suppurative cases of lymphocytic plasmacytic pancreatitis (mainly cats) or as a one-time attempt to eliminate bile duct obstruction due to pancreatic inflammation (give one injection of dexamethasone 0.25 mg/kg and monitor bilirubin over the subsequent 48 hours).

Pancreazyme supplementation has been used anecdotally in the treatment of chronic pancreatitis. Improvement in clinical signs is presumably due to the negative feedback of Pancreazyme on the release of endogenous pancreatic enzymes.

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