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February 2011

## Canine Pancreatitis

**Pancreatitis** continues to be a poorly understood disease process in canine patients. Both acute and chronic forms exist, and within this differentiation, both mild and severe changes have been recognized. Traditionally, canine patients were thought to be afflicted more commonly by acute pancreatitis, however recent studies based on pathology samples obtained at necropsy in patients with pancreatitis suggest that this may not be the case, and that ***chronic pancreatitis may be up to twice as common as acute pancreatitis.***

**Signs** vary from none in subclinical patients to multi-organ involvement in more severe forms. In general, ***anorexia, vomiting and weakness*** are seen in greater than ¾ of patients with severe pancreatitis, followed by ***abdominal pain, dehydration and diarrhea*** (seen in ½ to 1/3 of patients). Rare patients display neurologic signs, termed pancreatic encephalopathy. Localized effects, such as vomiting and abdominal pain are thought to be secondary to trypsin activation, leading to pancreatic edema and necrosis of the peripancreatic fat. Systemic effects are now thought to be secondary to other inflammatory mediators that can lead to hypotension, pulmonary edema and DIC.

**Diagnosis** needs to be based on a multifactorial approach, including clinical signs, labwork, ***ultrasonography and cytology or histopathology***. The ***minimum database*** of CBC, chemistries, thyroid and urinalysis should be completed to evaluate for the presence of other underlying systemic disease. However, ***simple elevations in amylase and lipase alone are not diagnostic of pancreatitis, as both of these enzymes are only approximately 50% specific for the pancreas under the best of circumstances.*** Elevation in either amylase or lipase alone is not indicative of pancreatitis. Advances have been made in delineating the origin of pancreatic lipase, and the Spec ***cPL*** (Iddex and GI Lab at Texas A & M) is extremely specific and has greater than 80% sensitivity in detecting elevations in lipase secondary to pancreatitis and is now the standard laboratory test for acute pancreatitis in the dog and cat. This test is also available as a bedside snap test through Iddex (Snap CPL ®) but may have more false positives than the Spec CPL.

**Ultrasonographic examination** is essential not only to evaluate the pancreatic tissue, but to interrogate other organ systems as well. In **acute pancreatitis**, an ill-defined, hypoechoic mass effect is generally detectable in the region of the gastric and/ or duodenal pancreatic limbs, surrounded by hyperechoic peripancreatic fat. In **chronic pancreatitis**, the pancreas may display thickening, mixed to hyperechogenicity or the presence of cystic structures (pseudocysts). Often animals will require **light sedation** to allow a complete ultrasonographic examination of the pancreatic region due to pain, and owners should be prepared for this possibility. In addition, this allows for **sampling** of the pancreas via fine needle aspirate or guided biopsy if suspicious tissue is located or the case has been nonresponsive to medical therapy. Gastric or intestinal gas shadow and 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 often CANNOT initially be 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, airdales). 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 (improvement versus progressive mass formation to aid in the ultimate diagnosis of neoplasia).

The following is a **suggested protocol for hospitalized moderate/severe cases**:

- 1) **NPO** approximately 48 hrs then introduce small amounts of water providing there is no vomiting and the dog is no longer painful. When there is at least 12 hours of absence of vomiting/diarrhea, then small amounts of a fat restricted diet such as W/D, R/D or Royal Canin LF can be introduced. If relapses occur, then discontinue oral feeding in favor of parenteral nutrition (PPN), or consider a jejunostomy tube (dogs) or a PEG tube (cats). The possibility of underlying neoplasia and the need for an **us-guided biopsy** should also be considered if not yet performed. Note that recent information suggests that even shorter periods of NPO are ideal, and trickle feeding of a low fat diet via nasogastric tube is a better alternative as enterocyte nutrition is maintained.
- 2) **Fluid therapy** at maintenance + ½-1 (attention to cardiac status when treating with aggressive fluids if there is concurrent DCM/Mitral insufficiency) with potassium supplementation (20-40mequ/liter) and B complex. Oncotic pressure enhancement with Hetastarch (Hespan) therapy may be considered (5-10ml/# added to LRS every 48 hrs) reducing total fluids to 1.2-1.5 of maintenance. **Dopamine** cri 5ug/kg/min has been shown to exacerbate severe hemorrhagic pancreatitis by enhancing perfusion to the parenchyma. It must be

initiated in the first 12 hours of therapy to be of benefit. **Selenium** 0.3mg/kg sodium selenite added to IV fluids has been used as a beneficial antioxidant.

- 3) **Buprenorphine or fentanyl are good choices for pain management (meperidine** (5-10 mg/kg every 2-4 hrs IM/SQ) is an alternative as is the IV mixture of ketamine, morphine and lidocaine)
- 4) **Antiemetics** are very important to minimize vomiting and to allow for enteral nutrition. This is typically achieved with **Cerenia** (maropitant) 1 mg/kg SC SID for up to 5 days, **Zofran** (ondansetron) 0.11-0.22 mg/kg IV/IM/SC or **Anzemet** (dolasetron) at 0.2-0.6 mg/kg IV SID. More traditional anti-emetics include **Metoclopramide** (0.2-0.5 mg/kg SC TID or 0.01-0.02 mg/kg/hr CRI) or **Chlorpromazine** (watch excessive sedation with butrophanol/meperidine) (0.25-0.5 mg/kg IM tid/qid) .
- 5) **Plasma** (fresh frozen) therapy may be considered to supplement alpha2 macroglobulins which bind released proteases from the pancreas (which cause autodigestion of the pancreas and resultant inflammation). This will also aid at reducing protease induced cardiovascular compromise and DIC and will enhance oncotic pressures. Plasma will help treat hypoalbuminemia but 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 is stabilized. Begin transfusion at a slow rate of ¼ maintenance for 15-20 min, then increase rate to 0.5 maintenance for ½ hour then give the rest over 4 hours. Pretreatment with diphenhydramine can be given at 0.5 mg/kg IM.
- 6) **Antibiotic therapy** is controversial yet essential if fever is present or abscessation suspected on ultrasound. **Enrofloxacin** (Baytril) has been found to penetrate the pancreas well at 2.5 mg/kg bid IV. This drug should be diluted into the IV fluids or given as a half strength dilution with physiologic sodium chloride. Ampicillin (20 mg/kg IV, SC, PO TID) is a useful broad spectrum antibiotic.
- 7) **Peritoneal lavage** may prove beneficial if marked accumulation of abdominal fluid occurs. Abdominocentesis samples should be analyzed with C/S prior to initiating the procedure.
- 8) **Hypertriglyceridemia** can be treated with dietary fat restriction (W/D or Royal Canin LF) which already is a mainstay of therapy as well as treating underlying endocrinopathies. Addition of an Omega 3 fatty acid

supplement has been shown to further aid in decreasing overall triglyceride levels (marine fish oils 50-150 mg/kg PO SID once the dog is eating again). If triglyceride reduction is not ideal in 4 weeks (target <400mg/dl) then **Niacin** (100mg/day/dog) or **Gemfibrozil** (200mg/day/dog; 10mg/kg bid for cats) may be tried with attention paid toward side effects of erythema, pruritis, abdominal pain, G.I. symptoms, and altered liver function.

- 9) **Cortisone** can be considered in non-suppurative cases of lymphocytic plasmacytic pancreatitis (mainly cats) or as a one time attempt to liberate bile duct obstruction from pancreatic inflammation (author prefers one injection of dexamethazone 0.25 mg/kg and monitor bilirubin over the next 48 hrs).
- 10) **Pancreazyme** supplementation has been used anecdotally in the therapy of chronic pancreatitis with presumed negative feedback of release of pancreatic enzymes.

The preceding was obtained from sources primarily from, but not exclusive to, ACVIM 2001-2009.

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*Note: A representative case workup of canine pancreatitis with ultrasound guided fine needle aspirate and core biopsy may be found as the February 2011 case of the month on [www.SonoPath.com](http://www.SonoPath.com).*



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