A Case-based Approach to the Feline Patient with Vomiting and Increased Liver Enzymes (Part 2: Inflammatory Liver Disease and Pancreatitis)

Joseph Taboada, DVM, Dipl. ACVIM (Small Animal Internal Medicine)
School of Veterinary Medicine, Louisiana State University
Baton Rouge, Louisiana

Pancreatitis is a common inflammatory disease that has historically probably been over diagnosed in dogs and under diagnosed in cats. It can be acute, recurring, or chronic. Acute necrotizing pancreatitis is most common in dogs. It is a pathophysiologic process in which intrapancreatic enzymes are activated resulting in increases in capillary permeability, initiating of vasoactive amines, and direct tissue damage. Vascular injury and tissue necrosis within the pancreas often extends locally to the stomach, duodenum, colon, and liver. Systemic inflammatory response syndrome (SIRS) is a common sequel. Acute necrotizing pancreatitis is seen less commonly than chronic pancreatitis in cats. Chronic pancreatitis is associated with inflammation and fibrosis. Pancreatic stellate cells appear to be important in the pathophysiology of chronic pancreatitis. Stimulated by oxidative stress and cytokines involved in the inflammatory process, activated stellate cells migrate to the periacinar areas to deposit collagen and fibronectin. The fibrosis contributes to obstruction of pancreatic ductules which in turn contributes to inflammation. Fibrosis appears to be important to the pathophysiology of disease in cats. The pathologic characteristics of feline pancreatitis appear to be similar to those in people; especially when compared to that of dogs.

Signalment

Any age or breed of cat or dog may develop pancreatitis. In the dog middle-aged, obese females are overrepresented. In cats most cases are domestic short-haired cats but Siamese and Persian cats may be overrepresented.

Clinical signs

The typical presentation in dogs includes sudden onset of vomiting, anorexia, depression, and abdominal pain. There is often a history of recent ingestion of a fatty meal or
dietary indiscretion. Presenting clinical signs in cats include anorexia, lethargy, dehydration, hypothermia, and weight loss. Vomiting and abdominal pain are noted less frequently in cats than in dogs with pancreatitis. Diarrhea is occasionally noted. Dogs and cats with severe pancreatitis may develop ascites and dyspnea associated with pleural effusion. Icterus is a variable finding. An abdominal mass may be noted and was noted in as many as a third of cats in some studies. This is probably representative of only those cats with very severe pancreatitis. Hypotension and shock are severe complications seen commonly in acute necrotizing pancreatitis. While usually idiopathic, pancreatitis has been associated with hyperlipoproteinemia and hypertriglyceridemia, cholinesterase inhibitors such as organophosphates, trauma resulting in hypoperfusion, and drugs such as thiazide diuretics, furosemide, estrogens, azathioprine, L-asparaginase, sulfonamides, tetracycline, metronidazole, H2-receptor blockers, acetaminophen, procainamide, and nitrofurantoin in dogs. In cats toxoplasmosis, FIP, hepatic lipidosis, liver and pancreatic flukes, lymphosarcoma, trauma, fenthion toxicity, idiopathic chylomicronemia, and diabetes mellitus have all been implicated. Potential risk factors for pancreatitis in dogs, such as obesity, dietary indiscretion, a high fat meal, high fat diets, and pre-existing endocrine diseases do not appear to be risk factors in cats.

**Diagnosis**

Hematology and serum biochemistry findings are generally non-specific. A mild normocytic, normochromic non-regenerative anemia and leukogram findings consistent with a stress or mild inflammatory leukogram are typical in cats. In dogs a more significant inflammatory leukogram and hemoconcentration would be more likely. A severe neutrophilia with a left shift is usually only seen in severe acute necrotizing pancreatitis. Thrombocytopenia may be noted and is usually mild. Increased ALT, AST, GGT, and alkaline phosphatase activities are typically noted. Bilirubin may be mildly increased but this is not a consistent finding. Dogs are often azotemic but cats are less likely to be azotemic than dogs with pancreatitis. Hyperglycemia is common and may be associated with stress or with the development of diabetes mellitus. The relationship between pancreatitis and diabetes mellitus is well documented in dogs but not well described in cats. However, cats with diabetes mellitus caused by chronic pancreatic inflammation appear to be very sensitive to insulin administration. Anorexia may result in hypokalemia. Hypocalcemia is common but it is rarely severe enough to cause clinical signs. Hypocalcemia may indicate a poor prognosis in cats with pancreatitis. Occasionally hypercalcemia may be noted. Hypoalbuminemia may be noted, especially in dogs.
and cats with severe disease or concurrent liver disease. Serum cobalamin is low in a high percentage of cats with pancreatitis.

A lack of sensitive and specific markers of pancreatitis may make antemortem diagnosis of pancreatitis difficult, especially in cats. Serum amylase and lipase have long been used as screening tests for pancreatitis in dogs but are of little use in the diagnosis of pancreatitis in cats. Serum lipase is thought to be the better of the two enzymes for diagnosing pancreatitis in dogs because it is less likely to be increased in renal or other intraabdominal disease. That said, it is neither sensitive nor specific. Feline trypsin-like immunoreactivity (fTLI) has been used as a test for pancreatitis in cats but has not proven sensitive. Feline TLI appears to increase acutely but returns to normal very early in the disease course in most cats making it less than ideal as a diagnostic test. It has been suggested that serum concentration of fTLI > 100 µg/L is approximately 80-90% specific and 30-60% sensitive for feline pancreatitis. An abnormal result is therefore usually, but not always, associated with pancreatitis. Azotemia may increase fTLI and increases have been noted in cats with severe inflammatory bowel disease. An immunoassay for measuring pancreatic lipase [pancreatic lipase immunoreactivity (PLI)] has been developed by the Texas A&M Gastrointestinal Diagnostic Laboratory and licensed to Idexx Laboratories. It is now considered to be the most sensitive and specific test for diagnosing pancreatitis in dogs and cats. Studies in cats with experimental pancreatitis would indicate that fPL increases rapidly after the development of pancreatitis and stays increased for much longer than fTLI. In one published study (Forman et al., J Vet Inten Med 2004;18:807-815), fPL was found to be 80% sensitive for feline pancreatitis, but there was not a significant difference between cats with pancreatitis and healthy cats. The use of fPL is recommended in combination with abdominal ultrasound (see below). Commercial assays for measurement of cPLI (Spec cPL™) and fPL (Spec fPL™) are based on the original cPLI and fPLI technology. The Spec fPL™ has become available through IDEXX Laboratories.

**Diagnostic Imaging**

Radiographs are usually non-specific. Decreased serosal detail may be noted if ascites is present. Decreased detail in the upper right quadrant on the ventrodorsal view may be noted but is seen less commonly in cats than in dogs with pancreatitis. A mass effect may be noted in severe cases. Pleural effusion may be noted on thoracic radiographs. Ultrasound of the pancreas in pancreatitis may reveal a mixed or hypoechochogenic pattern, cavitary lesions, dilation of the pancreatic ducts, or evidence of peripancreatic edema and effusion. The pancreas may
appear normal on ultrasound in many cats with pancreatitis, but a recent study (Forman et al.) found 80% sensitivity and 88% specificity for ultrasound in diagnosing feline pancreatitis. Because of the similar sensitivity and higher specificity of abdominal ultrasound vs. fPLI found in this study, ultrasound could be considered the more useful diagnostic test. Further study is needed before firm recommendations can be made. Abdominal computed tomography (CT) has not been shown to be useful in diagnosing pancreatitis in cats.

**Biopsy**

The gold standard in diagnosis of pancreatitis is histopathology. Findings in cases of acute pancreatitis include peri-pancreatic fat necrosis, and focal to multifocal pancreatic acinar cell necrosis and inflammation. The inflammation can be quite variable making evaluation of multiple biopsies from different sites critical. Chronic pancreatitis is more common in cats and is usually characterized by variable degrees of fibrosis and lymphocytic inflammation. Fibrosis appears to be more important than inflammation in chronic pancreatitis in cats. The pancreas may appear grossly normal so biopsy is warranted even in cases where the pancreas appears normal at laparotomy or laparoscopy. In a recent study the prevalence of pancreatitis based on histopathology was 67% in cases with GI and other disease and 45% in apparently healthy cats.

**Treatment (Canine)**

Depending on the severity of the pancreatitis, treatment of dogs can at times be both difficult and frustrating. In most cases of canine pancreatitis a specific underlying etiology cannot be determined but if it is, specific treatment for that cause should be initiated. Treating shock, rehydration and maintenance of normovolemia are the initial goals of therapy. Fluids such as 0.9% NaCl or lactated Ringers should initially be given. Potassium should be added to the fluids to maintain normal potassium, and glucose should be added if hypoglycemia is noted. Maintaining pancreatic microcirculation may be enhanced by plasma (20ml/kg IV) or colloid (10-20ml/kg/day IV) administration. Plasma transfusion is widely recommended in veterinary medicine but has not been critically evaluated in dogs and cats with pancreatitis. Studies in human patients with pancreatitis have not shown an advantage of plasma therapy. Dextran 70 and hetastarch may have antithrombotic effects that help maintain the microcirculation in addition to their colloidal effects. An external source of heat may be necessary to treat hypothermia, especially in smaller dogs with a larger surface area to weight ratio. Antibiotics should be directed against a bacterial infection if one is suspected. Even if a primary bacterial
infection is not suspected, a broad spectrum antibiotic should be given to minimize the effects of bacterial translocation. Abdominal pain is common so analgesics should be used as needed to keep the patient comfortable. When pain is present, parenteral administration of opioid agonists such as hydromorphone, morphine, and fentanyl provide relief to most patients with severe pain. A fentanyl patch can be applied as an effective means of delivering analgesia in a dog that is likely vomiting. An epidural catheter can be placed for epidural delivery of analgesic medications or local anesthetic can be administered into the caudal thoracic space or cranial peritoneal cavity. Corticosteroids are indicated in acute pancreatitis only if shock is present. There is some controversy as to whether steroids may have negative effects on patients with pancreatitis but there is little convincing evidence that steroids are either helpful or detrimental except in situations such as shock. Immune-mediated pancreatitis, especially in association with systemic lupus erythematosus, is increasingly recognized as an important cause of pancreatitis in people, and steroid therapy has been shown to reduce mortality significantly in these patients. Primary immune-mediated pancreatitis has not been described in dogs and cats, but it should be considered that a definitive cause is not found in most dogs and cats with pancreatitis. Conventional therapeutic approach to patients with pancreatitis would indicate that the patient should be fasted to allow the pancreas to “rest.” Fasting should result in a physiologic state in which less pancreatic enzyme is being produced and released which may result in reduced pancreatic damage during periods of pancreatic inflammation. The conventional approach is to fast the patient until the clinical signs associated with the pancreatitis have stopped. In some cases the clinical signs may linger for quite some time and nutritional support may become indicated. Dogs are metabolically suited for long fasts and one should not be too quick to start feeding in canine patients with pancreatitis. If nutritional support is deemed necessary, however, ideally, jejunostomy tube feeding or TPN should be considered to reduce pancreatic activity. It should be noted that even when jejunostomy feeding or TPN is used pancreatic activity will be increased over the basal fasted state.

The common practice of fasting dogs with pancreatitis is somewhat questionable. The underlying mechanism of pancreatic damage involves abnormally high levels of cholecystokinin, resulting in activation of digestive enzymes within the pancreatic parenchyma and suppression of pancreatic secretion. One could argue that feeding to restore more normal pancreatic secretion might be advantageous. In fact, large-scale studies in human pancreatitis have shown decreased morbidity and mortality when patients are not fasted. Some veterinary
internists recommend feeding of dogs with pancreatitis unless vomiting prevents it, and the practice of fasting these patients should probably be reconsidered.

**Treatment (Feline)**

Therapy for cats with pancreatitis is not well described or agreed upon. If a specific underlying etiology is noted it should be treated. Cats with acute necrotizing pancreatitis should be treated with fluids such as 0.9% NaCl or Lactated Ringers. Potassium should be added to the fluids to maintain normal potassium and glucose should be added if hypoglycemia is noted. Treating shock, rehydration and maintenance of normovolemia are the goals. Maintaining pancreatic microcirculation may be enhanced by plasma (20ml/kg IV) or colloid (10-20ml/kg/day IV) administration. Dextran 70 and hetastarch may have antithrombotic effects that help maintain the microcirculation. An external source of heat may be necessary to treat hypothermia. Antibiotics are indicated if bacterial infection or toxoplasmosis is suspected but are not necessary to prevent the bacterial translocation that appears to be common in dogs. Abdominal pain is not common so analgesics are not indicated in most cases. When pain is present, butorphanol (0.2–0.5 mg/kg every 4–6 hours) can be given. Buprenorphine (0.005-0.01mg/kg SQ q6-12hrs) or oxymorphone (0.05-0.1mg/kg cats IM, SQ q1-3hrs can also be used but may have a negative effect on respiration. Non-steroidal antiinflammatory agents should probably not be used. Corticosteroids are indicated in acute pancreatitis only if shock is present. Corticosteroids may be indicated in cats with chronic pancreatitis and should be used if concurrent inflammatory bowel disease or liver disease is present. Because cobalamin deficiency is common, supplemental parenteral cobalamin should be considered. Injectable cobalamin can be administered at a dose of 250 ug subcutaneously once a week for 6 weeks, then every 2 weeks for 6 doses, then monthly. Most generic cobalamin preparations contain 1 mg/ml (1000 ug/ml). Most multi-vitamin and B-complex injectable formulations contain significantly lower concentrations of cobalamin. This practice of fasting the patient to “rest” the pancreas is more controversial in the cat because hepatic lipidosis can be a concurrent problem or a sequela to fasting. Ideally, jejunostomy tube feeding or TPN should be considered but if the patient is not vomiting gastrostomy tube, esophagostomy tube, or nasoesophageal tube feeding are probably appropriately used. Some authors would suggest that treatment indications include use of antioxidants such as vitamin E, SAMe, or Silybin. Supportive studies are lacking but the possible association with inflammatory liver disease and the mechanisms of actions of such nutraceuticals make their use logical.
Feline Inflammatory Liver Disease

Cholangitis and cholangiohepatitis is a complex of related inflammatory hepatobiliary disorders. They accounted for approximately 26% of the liver diseases reported in cats in one large retrospective study (Gagne, et al. JAVMA, 1999; 214:513). This was second to hepatic lipidosis which accounted for approximately 50% of the cases. Inflammatory liver diseases are characterized by the predominant inflammatory cell infiltrate seen histopathologically. The inflammation is usually seen in the portal areas; and can be characterized as suppurative (neutrophilic), non-suppurative (lymphocytic/plasmacytic); sclerosing lymphocytic cholangitis, or biliary cirrhosis (fibrosis). There have been many terms used in the veterinary literature to describe inflammatory liver diseases prompting the World Small Animal Veterinary Association (WSAVA) Liver Standardization Group to suggest standardized criteria for diagnosis of liver diseases of dogs and cats. The standards define three main forms of cholangitis which are recognized to occur in feline patients: neutrophilic cholangitis, lymphocytic cholangitis, and chronic cholangitis associated with liver fluke infestation. Cholangitis is often associated with periportal necrosis. Neutrophilic cholangitis can be further subdivided into acute (also termed suppurative by some authors) in which neutrophils are seen and chronic in which a mixture of neutrophils and lymphocytes/plasma cells are seen. Lymphocytic cholangitis (formerly lymphocytic portal hepatitis) is the term that has become accepted to describe the histologic classification in which lymphocytes and/or plasma cells are noted to infiltrate the portal areas. This replaces the older term, “lymphocytic/plasmacytic cholangiohepatitis.” Lymphocytic cholangitis was more common than neutrophilic cholangitis; being seen in 61% of the cats with inflammatory liver disease in the study by Gagne, et al. Although other studies have noted that chronic neutrophilic cholangitis may be more common. Whether these classifications represent different stages in the progression of one disease or are separate etiologic entities is not known. Nor is the underlying etiology of inflammatory liver disease in cats. Bacterial, allergic, and immune mechanisms have all been speculated to be involved. Bacterial cholangitis may either initiate the inflammatory process or perpetuate it early in the disease course. Immune mechanisms probably also play a role especially in chronic neutrophilic cholangitis and lymphocytic cholangitis. Cats with inflammatory hepatobiliary disease, especially those with suppurative disease, may also have pancreatitis and inflammatory bowel disease. The relationship between these three inflammatory conditions is not well worked out but it has been speculated that the underlying initiator of the inflammatory process may affect the liver, the
pancreas, and the small intestine concurrently. The term, “triaditis” has been coined to describe those situations in which inflammation of the liver, pancreas, and small intestine are seen to occur concurrently. While not a very accurate description, the term seems to have stuck.

**Diagnosis**

The clinical findings seen in cats with inflammatory liver disease are similar to those seen with hepatic lipidosis and other liver diseases. Vomiting, anorexia, lethargy, and weight loss are typical. Fever is occasionally seen. Diarrhea while not usual is more common than in cats with hepatic lipidosis and may represent the subset of cats with concurrent inflammatory bowel disease. Affected cats are rarely obese. Cats with neutrophilic cholangitis tend to be younger and are more likely to be severely systemically ill when compared to those cats with lymphocytic cholangitis. Any age cat can be affected. Males predominate in populations of cats with neutrophilic cholangitis as compared to those with lymphocytic cholangitis. Suppurative disease often has an acute course while disease characterized by lymphocytic/plasmacytic inflammation may be more chronic. In evaluating liver enzymes, alkaline phosphatase tends not to be as elevated as in cats with hepatic lipidosis and transaminase activities tend to be higher. It is important to note that liver enzymes can be normal, even in cats with significant hepatobiliary inflammation. Neutrophil counts, transaminase activities, and total bilirubin concentrations tend to be higher in cats with neutrophilic cholangitis when compared to cats with lymphocytic cholangitis. All liver enzymes may be normal early in the course of disease, however. Diagnosis is usually dependent on biopsy as FNA is often normal or reveals non-specific changes. Biopsy for both histopathology and culture should be performed if inflammatory liver disease is suspected. The advent of readily available ultrasonography has resulted in Tru-cut needle biopsy becoming the most popular method of obtaining tissue for histopathology. The diagnostic accuracy of Tru-cut obtained biopsies has been questioned (Cole, et al. JAVMA, 2002; 220:1483-90). In the study by Cole, et al. liver biopsies obtained from dogs and cats by tru-cut techniques were compared to wedge biopsies. Paired 18 g Tru-cut needle biopsies commonly yielded a different diagnosis than wedge biopsy. If it is assumed that the wedge biopsy is the “gold standard” then the 18 g Tru-cut biopsies were highly inaccurate. Larger samples obtained with a 14 g needle may be more accurate. Laparoscopically obtained samples should be considered when feasible. Prior to biopsy, coagulation parameters should be evaluated. PIVKA may be the most sensitive indicator of potential bleeding tendencies. Vitamin K1 (0.5-1.5 mg/kg SQ given within 24 hours of biopsy may decrease the risk of bleeding.
Treatment

In addition to the supportive and nutritional support used to manage cats with hepatic lipidosis, antibiotics should be used when treating cats with inflammatory liver disease. For patients with suspected loss of hepatic function, it is ideal to select drugs that rely on the kidneys for elimination rather than hepatic biotransformation. In the case of antibiotic therapy, the \( \beta \)-lactam antibiotics (penicillin, ampicillin, cephalosporins) are the best choice. (Hepatic reactions observed in people caused by amoxicillin-clavulanate were associated with a specific leukocyte antigen and have not been reported in animals.) The fluoroquinolones (enrofloxacin, marbofloxacin, orbifloxacin, difloxacin) have had a good safety record and increased risk of toxicity in animals with hepatic disease has not been documented. Although some of these drugs are metabolized, the clearance is low and probably not affected unless there is substantial loss of hepatic function. These drugs are also cleared by the kidneys. Fluoroquinolones have been known to cause central nervous system (CNS) problems in susceptible individuals. This is most likely caused by penetration across the blood-brain-barrier (BBB) and inhibition of the action of the GABA neurotransmitter. Problems observed have been seizures, excitement, and disorientation. Animals with seizure disorders caused by hepatic encephalopathy may be more prone to CNS problems caused by fluoroquinolones. If a complication is observed after prescribing a fluoroquinolone drug, a switch to a safer drug is appropriate. Macrolides (erythromycin, azithromycin, and similar drugs) are sometimes used for infections in animals with hepatic disease. There are no specific problems identified in patients with hepatic disease, but these antibiotics are often associated with gastrointestinal problems in animals (diarrhea and vomiting). Therefore, when prescribing these drugs, veterinarians should be careful not to mistake a drug-related problem for an underlying disease, or complicate an already-existing problem. Metronidazole and related drugs (tinidazole, ronidazole) are sometimes used in patients with hepatic disease because of the anaerobic spectrum. They have been safe drugs when prescribed according to standard dose recommendations, but when doses have been exceeded, problems may arise. The most serious problem caused by metronidazole has been attributed to CNS toxicity and include seizures, ataxia, nystagmus, tremors, and rigidity. These signs have been attributed to inferring with the inhibitory neurotransmitter GABA. Because animals with hepatic disease also may be prone to CNS disorders that also share these clinical signs, veterinarians should understand the risks of metronidazole, and become familiar with the signs associated with toxicity. When using other
antimicrobials, veterinarians should be aware of the common adverse effects that may occur if a
drug accumulates because of a deficiency in metabolism. Drugs to avoid, if possible, include:
trimethoprim-sulfonamides, tetracyclines, rifampin, nitrofurantoin, and chloramphenicol.

Immunosuppressive agents should be added to the treatment regime in cats with
lymphocytic disease and in cats with neutrophilic disease that fail to respond to antibiotics alone
or antibiotics and nutraceuticals. Prednisolone (2-4 mg/kg/day initially then slowly tapered to 1
mg/kg QOD) is used most commonly. Other immunosuppressives that may be used in cats
responding poorly to glucocorticoids include chlorambucil (1.5 to 4 mg/M² twice a week to every
other day; approximately 1 mg < 7 lb cat, 2 mg > 7 lb cat) [probably a safer alternative to
azathioprine in the cat], azathioprine (0.3 mg/kg q24-72 hrs) [Note that cats are much more
sensitive to the myelosuppressive effects of azathioprine than dogs], methotrexate (0.4 mg
divided into 3 doses and given over 24 hours and repeated every 7-10 days has been
advocated as a pulse therapy but has not been extensively studied). Ursodeoxycholic acid
(Actigall®) 10-15 mg/kg PO SID is a safe treatment alternative that can be used in cats with
either supplicative or non-suppurative disease. The drug appears to have multiple actions
including shifting the bile acid pool to a less toxic hydrophilic population, a choleretic effect,
reducing expression of Class 2 major histocompatibility complex, and an antiinflammatory
effect. Vitamin E (aqueous alpha tocopherol, 10-100 IU/kg/day) has been advocated for its
antioxidant effects. SAMe (90-180 mg PO SID; Denosyl® NutraMax) is a precursor of
 glutathione. Glutathione is an important antioxidant that has been shown to be reduced in dogs
and cats with liver disease. The nutriceutical SAMe may help replace glutathione. It also may
have hepatoprotective effects in preventing programed cell death (apoptosis) that occurs during
inflammatory liver disease. Milk thistle (silymarin) is a nutriceutical that is widely used for its
hepatoprotective effects. It may be of benefit as an antioxidant, as an antifibrotic agent, or as
an aid in hepatic regeneration. Many studies have evaluated its use in people and show mixed
results. Studies in dogs and cats are lacking. Anecdotial evidence would suggest it may be
useful at a dose of 50-200 mg/kg PO SID. NutraMax markets a product for cats, Marin®, that is
a combination of silybin and vitamin E. Silybin is one of the active ingredients in milk thistle. In
Marin® it is complexed with phosphatidylcholine to increase the bioavailability. The amount of
vitamin E in the tablets is lower than is generally recommended in treating liver disease. A
combination of silybin and SAMe is available as the nutriceutical Denamarin®.

Concurrent pancreatitis, inflammatory bowel disease, and liver disease in cats
Clinicians have noted an apparent relationship between inflammatory bowel disease, pancreatitis, and liver disease in cats. Damage to intestinal mucosal epithelial integrity occurs during inflammation such as that seen in IBD which may permit inflammatory mediators, endotoxins, and microbial components access to the portal circulation. The concentration of these inflammatory mediators may exceed the capacity of hepatic macrophages to remove and degrade them resulting in deposition of immune complexes in the liver, complement system activation, and eventually hepatocellular inflammation and necrosis. The anatomic association between the common bile duct and the pancreatic duct may allow for ascending bacterial infections to affect both organs. While this association is often referred to, there is little objective evidence in the literature that documents it well. The relationship was confirmed in a study designed to determine whether cats with inflammatory hepatic disease had concurrent IBD, pancreatitis and interstitial nephritis. [Weiss DJ, et al. JAVMA 209:1114; 1996] In Weiss's study, the prevalence of IBD (83%) and pancreatitis (50%) was significantly greater for cats with cholangiohepatitis (suppurative cholangitis) compared to cats without inflammatory liver disease. However, the relationship was not apparent in cats with lymphocytic portal hepatitis (lymphocytic cholangitis). In studies of cats with pancreatitis, an association between hepatobiliary disease and pancreatitis has been observed. [Akol KG, et al. JVIM 15:327; 2001], [Hill RC, et al. JVIM 7:25; 1993], [Clark JEC, et al. JFMS 13:570; 2011] There appears to be a detrimental effect on prognosis for cats with both pancreatitis and hepatic lipidosis and pancreatitis and inflammatory liver disease. An association with inflammatory bowel disease was implied in Clark’s study. In a study evaluating cats with subnormal serum concentrations of cobalamin, 22 of 49 cats had simultaneous presence of inflammatory disease of the intestines, pancreas, or hepatobiliary system. [Simpson KW, et al. JVIM 15:26; 2001] While none of these studies conclusively makes an association, the evidence is compelling that inflammatory disease can at times affect multiple gastrointestinal organs. Based on the limited data available it would appear that cats with pancreatitis that die are more likely to have multiple organ systems affected.

Treatment of cats with concurrent inflammatory bowel disease and pancreatitis is generally based on management of the inflammatory bowel disease (or inflammatory liver disease). The effect of corticosteroids on cats with pancreatitis is not known but there is no evidence that steroids would have a detrimental effect in cases that are not of primary bacterial origin. In chronic pancreatitis, the anti-inflammatory effect of the corticosteroids may be of benefit for similar reason to the benefit achieved when treating inflammatory bowel disease or
inflammatory liver disease. Steroids have long been considered a cause of pancreatitis in dogs, but a clear cause and effect relationship has not been established. It’s possible that steroid-induced pancreatitis has been over-diagnosed in the past. Studies have shown that steroids are associated with increases in serum amylase and lipase independent of any detectable pancreatic pathology. Nutritional support probably plays a more important role in the management of cats with concurrent inflammatory bowel disease and pancreatitis because of the potential association with hepatic lipidosis. Fasting cats with concurrent disease is not recommended.