

Inflammatory Bowel Disease and Pancreatitis in Cats

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INFLAMMATORY BOWEL DISEASE

Inflammatory Bowel Diseases (IBDs) are a diverse group of intestinal disorders, which have been grouped together based on their histopathological commonalities. This is somewhat unfortunate, in that the tendency is to see “IBD” as a diagnostic endpoint, with a sole treatment protocol, rather than as a description of a pathophysiologic response. The term “IBD” should ideally be restricted to those forms in which all identifiable etiologies have been ruled out, and with good conscience, one can term the condition “idiopathic” in etiology.

“The normal intestine is usually in a steady state of physiologic inflammation, representing a dynamic balance between factors that activate the host immune system (e.g., luminal microbes, dietary antigens, endogenous inflammatory stimuli) and host defenses that maintain the integrity of the mucosa and down-regulate inflammation.” (Crawford JM.

The Gastrointestinal Tract. In: Cotran RS, Kumar V, Robbins SL, eds. *Robbins Pathologic Basis of Disease*. Philadelphia: WB Saunders Co, 1994; 755-829.) Inflammatory bowel disease seems to represent a loss of this balance.

As in humans with Crohn’s Disease, IBD primarily localized in the small bowel responds differently than disease primarily localized in the large bowel, even when the histologic lesions are similar. Unlike people, however, cats seem to respond more readily to dietary therapy. It is important to distinguish between a food hypersensitivity or food intolerance and IBD, which is, by definition, a term given to a group of disorders of unknown etiology. That is not to say, however, that IBD may not benefit from dietary therapy because the gut is naïve relative to the novel antigens. Current thought is focused on defects in the mucosal immune system as the probable cause, which initiates or perpetuates the pathophysiology underlying IBD.

There are several lines of reasoning supporting an autoimmune mechanism in IBD. These include the following:

- Lymphocytic, eosinophilic, and plasma cell infiltration occurs in tissues involved in hypersensitivity reactions.
- Resolution of disease occurs with immunosuppressive therapy. (This may, however, be an epiphenomenon reflective of the multiple effects of the drugs used.)
- Other diseases known or suspected to have an immunological basis occur concurrently in people with IBD. Similarly, in cats with IBD, nephritis, cholangiohepatitis and pancreatitis with lymphocytic-plasmacytic infiltrates are recognized commonly.
- Finally, disorders of the immune system are often multisystemic.

For normal intestinal immunity, an intact mucosal barrier is required. Because antibodies to enteric antigens are found in some people with IBD, a defect in mucosal permeability is suspected. It is not clear whether increased gut permeability occurs as a cause or a result of inflammation.

All of these disorders have inflammatory cells infiltrating the mucosa and lamina propria of the intestinal tract, sometimes, regionally, other times diffusely generalized. The infiltrate most commonly consists of lymphocytes and plasma cells, but can also be of a neutrophilic, eosinophilic or granulomatous character. The infiltrate is described by the predominant cell type present. From a pathologist’s perspective, a problem with describing the severity of the lesions is that, like describing cell populations in tracheal wash specimens, there is controversy and difference of opinions in how many cells of each type are normal in cat gut.

The result of these responses is a bowel that has regional or diffuse sections of rubbery walls thickened with edema, inflammation, and fibrosis. It is critical to verify by full thickness biopsy, whether there is evidence of intestinal lymphosarcoma as this may look similar to lymphocytic-plasmacytic IBD in the superficial luminal layers of the bowel. Chronic inflammation of the bowel may be self-perpetuating and IBD may progress to small cell lymphoma.

History and Clinical Findings

Clinical signs vary with location of the inflammatory process: duodenal and gastric lesions usually present as vomiting and weight loss while small intestinal or colonic lesions present as diarrhea +/- weight loss (if small intestinal). However, some colonic IBD may cause vomiting as well. There are also cats in whom the inflammatory process extends beyond the gastrointestinal tract and affects the liver (+/- the gallbladder) and pancreas. This is fondly termed “triaditis” (see below). These cats may present with signs attributable to these organs, which may or may not include vomiting and diarrhea.

Most commonly, cats are presented with a history of chronic, intermittent problems, which may have been going on for weeks to years and may or may not be progressive. There is no breed or sex predilection and, although it can be diagnosed in any age cat from several months of age upward, there are more who are middle aged. Vomiting occurs acutely, may occur over a period of days and then cease till the next episode, or may occur daily. It is unrelated to eating and usually is clear froth or fluid or bile, but may include undigested/semi-digested food contents. The cat may seem otherwise completely well, or be lethargic and inappetent.

Diarrhea, if present, is of a chronic and unresponsive nature, and weight loss may be dramatic. The character of the stool varies widely, and may even be steatorrheic. If malabsorption is involved, the cat may have a voracious appetite, otherwise the history is unremarkable. Large intestinal lesions predispose to tenesmus, increased frequency of defecation +/- urgency, blood, mucous, and a change in or loss of litterbox habits. It is helpful to characterize existing diarrhea as small or large bowel in origin.

Other differentials for chronic diarrhea, weight loss, voracious appetite in cats include hyperthyroidism, lymphoma, exocrine pancreatic insufficiency and occasionally the cholangitis/cholangiohepatitis complex. Bacterial overgrowth, giardiasis and other parasites, adenocarcinoma, histoplasmosis, FIP, FeLV, and FIV are additional considerations.

Occasionally cats will be asymptomatic other than weight loss. Palpably thickened intestinal loops may be present and enlargement of the mesenteric lymph node may be noted. Mild dehydration may be evident.

Diagnostics

A baseline CBC, biochemical profile, T4, lipase, FeLV/FIV, urinalysis, fecal exam for parasites, rectal cytology (if large intestinal diarrhea) and fecal culture and sensitivity are rational first choices.

Hematologically and biochemically there are often minimal changes: a stress leukogram is common, +/- eosinophilia, a mild non regenerative anemia, +/- hyperglycemia, mild hypoalbuminemia, hypo or hyperglobulinemia, hypokalemia, +/- mildly elevated liver enzymes.

Second tier diagnostic tests rely on indirect visualization. Radiographic and ultrasonographic findings may be unre-

markable and non-specific; nevertheless, they may be helpful from the standpoint of determining the extent of the apparent disease process and in making appropriate recommendations towards direct visualization and biopsy via endoscopy vs. exploratory laparotomy. Ultrasound will often show regional thickening of intestinal walls, which retain normal architectural layers.

Can these changes progress to more severe disease? There is evidence of biopsy proven lymphocytic-plasmacytic enteritis (LPE), relapsing as diffuse intestinal lymphoma after years of good response to therapy. It is possible that severe lymphocytic-plasmacytic IBD may be a pre cancerous lesion. It is also noteworthy, however, that partial thickness endoscopic biopsies may miss the telltale neoplastic lymphoblasts because they are found in the muscularis and deeper layers. This risk can be minimized by harvesting many good quality tissue samples from each section of the gastrointestinal tract being evaluated.

Note:

The presence of increased inflammatory cells in an intestinal biopsy does not necessarily confirm a presumptive diagnosis of IBD. Biopsy criteria *MUST* include increased proprial lymphocytes and plasma cells, however there must also be alterations in the mucosal structure, such as villous atrophy/fusion, cryptal separation with edema, infiltrate and fibrosis. One must eliminate the other known and detectable causes of chronic inflammation first. Therefore, parasitic infestations and retroviral infections should be tested for. **A dietary trial with a limited antigen diet should be undertaken for a minimum of 6–8 weeks** and the possibilities of bacterial overgrowth or lymphoma should be considered.

If exploratory laparotomy is pursued, biopsy the stomach, duodenum, jejunum (several sites) and ileum (+/- colon). Get a definitive diagnosis wherever you can. Biopsy the liver and pancreas. Place one piece in culture medium in case a suppurative condition is found. It is helpful to differentiate between lymphocytic vs. suppurative cholangitis or cholangiohepatitis in determining an appropriate treatment protocol.

Evaluate for any and all allergens to check for or eliminate them if possible.

- Evaluate for food hypersensitivities and feed a unique antigen diet
- Deworm
- Treat for hairballs

Suppress/modulate the immune system using metronidazole (Flagyl™) to inhibit cell mediated immunity (CMI) and eliminate coexisting protozoal and anaerobic infection (10 mg/kg PO BID). If liver disease is present, reduce the metronidazole dose to 7.5 mg/kg PO BID. If dietary manipulation and metronidazole therapy are inadequate for controlling the clinical disease, addition of omega 3 fatty acids could be considered, in an attempt to interfere with arachadonic acid cascade thus reducing the inflammatory

response. However, as long as the diarrhea persists, they should be avoided, as unabsorbed fatty acids are hydroxylated by intestinal bacteria, stimulating colonic water secretion thus exacerbating diarrhea and fluid loss. Corticosteroids should be used to suppress the immune response. It is very important to start with a high dose before decreasing if they are to be used effectively! (2–4 mg/kg PO divided BID prednisone or dexamethasone 0.2 mg/kg PO BID)

Important:

Suppress the process, so that there are **NO SIGNS** for 2 weeks before decreasing the corticosteroid dose, gradually to the lowest alternate day or once a day dose that controls the signs completely.

Other therapies...

- Consider antibiotics for bacterial overgrowth (tylosin [Tylan™] 10–20 mg/kg PO BID).
- Vitamin K may be indicated where severe fat malabsorption predisposes to Vit K responsive coagulopathy.
- Serum cobalamin and folate may be indicated due to intestinal malabsorption or decreased production (Folate is given orally (0.5–1.0 mg q24h X 1 month); cobalamin must be given parenterally (125–250 mcg/week SC or IM once a week X 4–6 weeks).
- Fiber-enriched diets or supplements may be helpful in chronic large bowel diarrhea to absorb excess intestinal fluid and neutralizing toxins.
- Chlorambucil (Leukeran™) 0.25–0.33 mg/kg PO q 3 days [2 mg PO q3d]
- Cytotoxic (azathioprine [Immuran™] 0.3 mg/kg PO eod) MONITOR CBC. Note also, that beneficial effects may lag 2–3 weeks behind initiation of azathioprine therapy and may be needed for 3 to 9 months.
- Cyclophosphamide (Cytosan™) 50 mg/m² orally for 4 successive days each week. Note: MONITOR CBC before next dose
- Colonic antiinflammatory (sulfasalazine [Azulfidine™], a 5-ASA-sulfa agent 0.2–0.3 mg/kg PO q 24–48 hours) Note: observe for KCS and salicylate toxicity.

“TRIADITIS” VS. “IBD”

The term “triaditis” refers to a constellation of inflamed organs that are adjacent to each other, namely the small intestine, liver, and pancreas. Anatomically and pathologically, it is “logical” to understand why this may occur. Pathology in the distal common bile duct, either ascending from the duodenum or originating in the duct itself, (such as infection or cholelithiasis), can predispose to pancreatitis because of the functional relationship between the major pancreatic and common bile duct sphincters in the cat. Experimentally, it has been shown that when the major

pancreatic duct is perfused with bile acids, marked structural changes occur not only within the pancreatic duct, but also in the pancreas itself. (This is why feline pancreatic disease is a common cause of extrahepatic biliary obstruction.) In approximately 80% of cats, the accessory pancreatic duct is absent. The pancreatic duct enters the common bile duct before the latter opens into the duodenum at the major duodenal papilla (**Figure**).

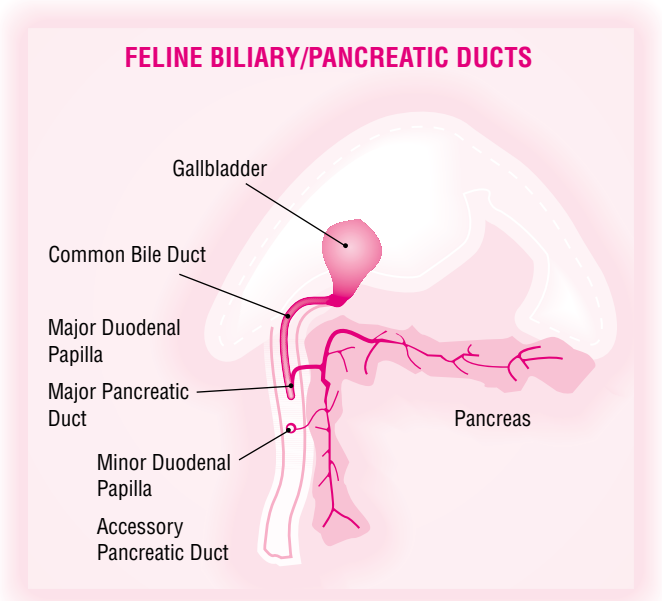


Figure. Normal anatomy of the common bile duct and pancreatic duct in the cat. (Adapted from Center SA, *Diseases of the gall bladder and biliary tree* in Guilford WG (ed). *Strombeck's Small Animal Gastroenterology*, Philadelphia, WB Saunders Co, 1996, p860.)

GASTROINTESTINAL (GI) LYMPHOMA

Lymphoma is the most common form of GI neoplasia in cats. Alimentary lymphoma has, in the past, been given a poor/grave prognosis. Dr. Keith Richter presented important findings emphasizing the need to distinguish between lymphocytic and lymphoblastic lymphomas. In a study of 67 cats, Dr. Richter found, through extensively biopsying stomach and small intestine endoscopically as well as surgically, that 90% of lymphocytic lymphoma involved the small intestine and might have been missed with endoscopy alone. For *lymphoblastic* lymphoma, approximately 50% of the cases involved small bowel only; the remainder involved stomach or stomach and small bowel. On histology, 25% of the 67 cases had *lymphoblastic*, 75% had *lymphocytic* lymphoma.

It is important to differentiate between the two types of lymphoma because they are treated very differently and because they have a different prognosis. *Lymphocytic* GI lymphoma is readily treated by the client in the home envi-

ronment with prednisolone (2–4 mg/kg PO divided BID) and clorambucil (2 mg PO q3days). The median disease free interval was 20.5 months (range 5.8–49 months). Rescue was achieved with cyclophosphamide. Cats with lymphoblastic lymphoma, on the other hand, responded poorly to chemotherapy using either CVP (cyclophosphamide, vincristine, prednisolone) or ACOPA (CVP + doxorubicin and L-asparaginase). Of interest also was his observation that cats with lymphoblastic lymphoma were more likely to have recurrences of abdominal masses.

PANCREATITIS

For years, feline pancreatitis has been assumed to be a similar disease to that in dogs. Currently, as with so many other disorders, it is recognized that this group of disorders is different in the cat. Remembering that the term “pancreatitis” implies nothing more than inflammation of that organ, it is not surprising that each species may have a variety of etiologies.

The incidence of pancreatitis is higher than previously believed. In fact, in a German retrospective study, the prevalence of pathologically significant lesions in dogs was found to be 1.5% and in cats, 1.3% of the specimens submitted. Indeed, there are papers reporting the incidence as high as 2.9 and 3.5% of necropsied cats.

Drs. Jörg Steiner and David Williams classify feline pancreatitis as acute or as chronic. Acute pancreatitis is a short term, completely reversible and without fibrosis on biopsy evaluation. Chronic pancreatitis is a long-term inflammation of the pancreas associated with irreversible histopathological changes, primarily fibrosis. Most of what we see in cats is the latter which is unfortunate in that it isn't curable; however, it can generally be controlled and is less fatal than severe necrotizing pancreatitis. Diabetes mellitus may be a result of chronic pancreatitis in some individuals.

Both acute and chronic pancreatitis can be mild or severe, but most commonly acute cases tend to be more severe, and chronic cases mild. Mild pancreatitis generally results in minimal clinical signs, minimal necrosis, and low mortality.

In severe pancreatitis, (necrotizing, hemorrhagic) extensive pancreatic necrosis and multiple organ involvement +/- organ failure are seen. Fortunately, because in cats this form is rare, severe multi-system complications are uncommon. The prognosis for severe pancreatitis is poor.

Pancreatic complications may or may not be present including fluid accumulation around the pancreas, infection of necrotic areas, pseudocysts, and abscesses.

It is conceivable that we will have histopathological classification schemes in the not too distant future (eg, focal suppurative, diffuse fibrosing, lymphocytic/plasmacytic, eosinophilic pancreatitis, etc.). This should help in designing appropriate therapeutic protocols for our patients. In order to achieve this goal, pancreatic biopsies are required for histopathological evaluation in our patients.

Etiology

1. More than 90% of the cases of feline pancreatitis are idiopathic.
2. Anything causing ischemia to the organ. “The most pivotal determinant in the development and progression of pancreatitis is likely the maintenance of local blood flow. Ischemia favours progression to an auto-digestive state; impairment of the microcirculation results in retention of activated enzymes, depletion of anti-proteolytic proteins, and reduced removal of toxic products. Necrosis of the gland follows pancreatic ischemia, leading to a self-perpetuating cycle of damage.” (Center SA, Proceedings of AAEP 2000 Fall Meeting)
3. Traumatic pancreatitis has been reported in a few cats associated with motor vehicle accidents or high-rise syndrome.
4. Several infectious agents have been implicated including feline parvovirus, *Toxoplasma* organisms (of 45 pancreata examined in 100 cats infected with *Toxoplasma*, 38 had lesions), feline herpesvirus I, *Eurytrema procyonis* (a fluke), feline infectious peritonitis (FIP), and, rarely, *Amphimerus pseudofelineus*. Look for toxoplasmosis.
5. Feline pancreatitis was reported in 2 cats following topical fenthion administration (organophosphate intoxication is a common cause of pancreatitis in children in developing countries).
6. Experimentally, hypercalcemia induced by calcium gluconate IV; and pancreatic duct infusion of oleic acids or infected fluids have induced pancreatitis in experimental models but probably are not significant causes of spontaneous pancreatitis.
7. Drugs have been implicated as causing pancreatitis in humans and dogs but not yet in cats. Drugs associated with pancreatitis in humans include azathioprine, chlorothiazide, hydrochlorothiazide, estrogens, furosemide, tetracycline, sulfonamides, L-asparaginase, 6-mercaptopurine, methyl dopa, pentamidine, nitrofurantoin, dideoxyinosine, valproic acid, and procainamide. Bear these in mind when selecting medications for patients with suspected pancreatitis.
8. NOTE. There is no evidence for glucocorticoids causing acute pancreatitis in dogs or in cats!

Pathogenesis

It is believed that various noxious stimuli can cause the exocrine pancreas to decrease the secretion of pancreatic enzymes, followed by the formation of cytoplasmic vacuoles with the co-localization of proenzymes of digestive enzymes and lysosomal enzymes. Normally the lysosomal enzymes are strictly segregated from proenzymes to prevent premature activation of the proenzymes. A decreased pH along with the loss of segregation of the lysosomal enzymes and proenzymes cause abnormal intrapancreatic activation of trypsinogen which, when activated to trypsin, activates other proenzymes resulting in a local and systemic inflammatory response.

Clinical Findings

Pancreatitis should be included in a diagnostic rule-out list whenever there is a history of lethargy, anorexia, dehydration, hypothermia, vomiting, abdominal pain, abdominal mass effect, dyspnea, diarrhea and ataxia. One retrospective study reported that vomiting occurred in only 35% of cats with pancreatitis. Concurrent problems may include hepatic lipodosis, cholangitis/cholangiohepatitis, idiopathic inflammatory bowel disease, enteritis, diabetes mellitus, and vitamin K1 responsive coagulopathy. As such, the clinical findings on examination may be vague.

Statistically, 38% of cats diagnosed with hepatic lipodosis had concurrent acute pancreatitis and these patients were more likely to be cachectic and have coagulation abnormalities. This is very important, as these lipodotic cats have a worse prognosis.

Note:

The most common clinical problems in cats with pancreatitis are lethargy, anorexia, and dehydration.

Diagnostics

The classical signs of abdominal tenderness or mass effect in the right anterior quadrant, haziness in this region and displacement of abdominal viscera on abdominal radiographs and/or visualization of a (nodular) hyperechogenicity or peripancreatic fluid or a pancreatic abscess or mass on ultrasound examination support the presumptive diagnosis of pancreatitis. However, these findings are not common.

- 1) Radiographic findings may include reduced contrast in the cranial abdomen, localized dilatation of small intestinal loops, displacement of abdominal organs with the duodenum often moved dorsally and laterally, the stomach moved to the left, and the transverse colon caudally.
- 2) Ultrasonographic findings may include the following changes in the pancreas: swelling, increased echogenicity of the pancreas and peripancreatic fat, mass effects, and fluid accumulation around the pancreas.
- 3) Contrast-enhanced computed tomography (CT) is used in humans to diagnose and stage the severity of pancreatitis with its ability to detect and delineate areas of necrosis.

Practically speaking, ultrasound is the most sensitive, commonly available, non-invasive evaluative tool that we have at this time.

Biochemically and hematologically, changes are most commonly mild and nonspecific. There may be a mild, non-regenerative anemia in chronic pancreatitis or a severe

anemia terminally in acute, necrotizing pancreatitis. An inflammatory or stress leukon may be present, and in the case of a pancreatic abscess or a suppurative pancreatitis, a left shift may be seen.

Concurrent elevations of sap and alt are not uncommon and reflect inflammatory or lipidotic involvement of the liver. Nonspecific changes, such as hyperglycemia (stress or concurrent diabetes), hypocalcemia, hypokalemia (inappetance), hypercholesterolemia, azotemia (prerenal and/or renal), and hyperbilirubinemia have all been reported.

The lack of sensitivity and specificity of amylase and lipase is a source of frustration in diagnosing feline pancreatitis. Elevations in serum amylase may occur not only with pancreatitis, but more commonly from other gastrointestinal diseases, as well as from decreased renal clearance of this enzyme. Additionally, a normal serum lipase cannot be depended on to rule-out pancreatitis.

Trypsin-like immunoreactivity (TLI) has been shown to be diagnostic for severe acute pancreatitis. However, it does *not* detect the more common, chronic and milder forms of pancreatitis. Trypsinogen and trypsin are pancreas-specific in origin, and both are detected by the TLI assay. Serum TLI is very specific but *not* sensitive. Even though published normals are 17–48 micrograms/dl values under 150–200 are equivocal (GI Laboratory at Texas A&M University). TLI seems most reliable in identifying acute pancreatitis. Later in the course of disease it may not be elevated either because the sick pancreas has leaked all of the enzymes that it had made and is not capable of producing more (after several days of inflammation) or the pancreatic blood flow has decreased following the worst phase of the inflammatory response.

Recently, feline pancreatic lipase immunoreactivity has been validated by the GI Laboratory at Texas A&M University. In an abstract presented at the ACVIM Forum 2003, Dr. MA Forman showed that “feline PLI and abdominal ultrasound have good sensitivity and specificity, whereas helical CT does not appear to be a useful screening tool for the diagnosis of feline pancreatitis.”

The same group looked at serum and urinary markers for feline pancreatitis and found that: “both serum fTLI and trypsinogen activation peptide (TAP) concentrations were significantly higher in cats with pancreatitis than in clinically healthy cats...(whereas) urine TAP and urinary TAP/creatinine ratio do not appear to be clinically useful for diagnosing feline pancreatitis.”

Ultimately, surgical biopsy is required to make a definitive diagnosis. Whilst dogma was that biopsying the pancreas is a pathophysiologically dangerous undertaking, this does not appear to be the case in the cat. The author routinely biopsies pancreatae in all of her exploratory patients.

The procedure is simple: gently isolate the pancreas from the surrounding viscera and pack it off with a few gauze swabs prior to selecting either a gross lesion or routine selection of

both poles for biopsy using fine iris scissors. Submit a small piece in culture medium as well as formalin preserved samples, in case the lesion is reported as suppurative. Biopsies may also be collected via laparoscopy.

Therapy

Therapy for pancreatitis is best planned with knowledge of the type of pancreatitis present. Fluid therapy, pain relief, and nutrition are the cornerstones in supportive care. The goals of fluid therapy are to sustain blood and plasma volume, normalize blood pressure, and to correct acid-base and electrolyte disorders. Concurrent problems (such as lipidosis or enteritis) should be addressed as well. A noteworthy difference between the dog and cat is the recommendation to feed, rather than fast, those patients suspected of (or confirmed as) having pancreatitis unless they are vomiting. Even with the vomiting cat, designing a nutritionally supportive protocol is of great importance due to this species' predisposition for developing lipidosis. It is important not to fast cats for longer than 48 hours. Utilize anti-emetics as necessary. In the rare case where vomiting cannot be controlled even with ondansetron, total parenteral nutrition or jejunostomy tube feeding may be advisable for 7–10 days. Discussion of tube feeding (nasogastric, esophageal, gastrostomy, jejunostomy) is beyond the scope of this article.

If concurrent liver pathology is present, drug doses, including anti-emetic agents, should be reduced to take the impaired hepatic metabolism into consideration. Anti-emetics commonly used in the cat include metaclopramide (Reglan™) and chlorpromazine (Largactil™). Each of these drugs also has its own, inherent side effects, such as the central nervous system (CNS) sedation or frenzied behavior or disorientation of Reglan™ in the cat or the hypotensive effect of the Largactil™. Other antiemetics should be considered (**Table**). Zofran™ while costly, is very beneficial in the intractably vomiting patient.

It has been suggested that bland, low-fat, high-carbohydrate diets are most suitable; however, this author is not aware of any research done supporting this recommendation. Cats, being obligate carnivores, do not normally utilize carbohydrates well, therefore feeding a balanced, non protein-restricted diet is warranted. Restriction of fat is not indicated.

Modification of gastric acidity is advised; the gastric pH can be checked by measuring pH of vomitus or by gastric suctioning via a nasogastric (or other) tube. An H2 blocker, such as famotidine (0.5 mg/kg IV q12h) or a proton pump inhibitor, such as omeprazole (0.5–1.0 mg/kg PO q24h) may be used.

While pancreatic enzymes are generally only used in feline exocrine pancreatic insufficiency, David Williams has mentioned their use in human pancreatitis patients to reduce pain through feedback to the pancreas inhibiting further enzyme release (and leakage). Whether this is of benefit in cats (or dogs) is not known.

Analgesia is of critical importance in the comfort of the patient, but also in the progression of the disease/inflammation through the negative physiological effects of pain. **Pain causes disease and prevents healing.** Even if obvious abdominal pain isn't present, a test dose of 0.1–0.2 mg/kg oxymorphone IV, to see if the patient improves over the approximately 6-hour effective period, should be offered. If improvement is seen, then constant rate infusion of a narcotic may be considered or a transdermal fentanyl patch (Duragesic™) for continuous relief. Torbugesic™ is not as effective for visceral pain as the mu opioid agonists are. (A thorough discussion of analgesia is beyond the scope of this paper.)

Antibiotics are only indicated if the diagnosis of a suppurative pancreatitis has been made. In this case, antimicrobial selection is best made with the knowledge of a sensitivity spectrum. Generally gram negative and anaerobic organisms are implicated. Note that a suppurative pattern may be seen on histology in a *sterile* pancreatitis caused by enzyme damage.

Corticosteroids are indicated if a lymphocytic/plasmacytic form is reported or in an acute shock presentation. Other anti-inflammatories are not currently recommended; nor have any benefits been seen with the use of antacids, anticholinergics, GI hormones (somatostatin, glucagon), or calcitonin. Dopamine has been useful in acute experimental feline pancreatitis.

Fresh frozen plasma may be considered in cats with severe pancreatitis to replace plasma proteases, albumin and alpha 2 macroglobulins. Selenium was shown to be useful in dogs, however, to date; no study has been done to assess the role of selenium in therapy of pancreatitis in cats.

Table.

SELECT ANTI-EMETICS FOR USE IN THE CAT

| Generic Name | Product™ | Dose (feline) |
|----------------------------------|----------------------|---|
| Chlorpromazine | Thorazine, Largactil | 0.5 mg/kg q8h IM |
| Prochlorpromazine | Compazine | 0.1 mg/kg q6h IM |
| Diphenhydramine | Benadryl | 2.0-4.0 mg/kg q8h PO 2.0 mg/kg q8h IM |
| Dimenhydrinate | Dramamine | 8.0 mg/kg q8h PO |
| Prochlorpromazine + Isopropamide | Darbazine | 0.5-0.8-mg/kg q12h IM, SQ |
| Metoclopramide | Reglan | 1-2 mg/kg constant rate infusion IV over 24 hours |
| Ondansetron | Zofran | 0.1-0.15 mg/kg slow push IV q6-12 hours prn |
| Dolasetron | Anzemet | 0.6 mg/kg IV q24h |

Complications of ACUTE pancreatitis that may arise include DIC, thromboembolism, cardiac arrhythmia, sepsis, acute tubular necrosis, pulmonary edema, and pleural effusion. It has been suggested that a low dose of dopamine (5 mcg/kg/min) diminishes the severity of the disease. To prevent bacterial translocation, cover these patients with broad-spectrum antibiotics.

Prognosis

The prognosis for cats with pancreatitis depends on the type of pancreatitis as well as the degree of duration and severity. Many cats have chronic, low-grade smoldering pancreatitis and live long lives, but will have a better quality of life if a definitive diagnosis is made resulting in appropriate therapy.

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