CURRENT CONCEPTS IN DIAGNOSIS & THERAPY

Feline Acute Pancreatitis

FELINE FRIENDLY ARTICLE

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Pancreatitis appears to be a common disease in cats,[1] yet it remains frustratingly difficult to establish a clinical diagnosis with certainty. Clinicians must rely on a combination of compatible clinical findings, serum feline pancreatic lipase (fPL) measurement, and ultrasonographic changes in the pancreas to make an antemortem diagnosis, yet each of these 3 components has limitations.

PROFILE

Acute Versus Chronic Pancreatitis

Acute pancreatitis is characterized by neutrophilic inflammation, with variable amounts of pancreatic acinar cell and peripancreatic fat necrosis (**Figure 1**).[1] Evidence is mounting that *chronic pancreatitis* (see **In Brief: Diagnosis & Treatment of Feline Chronic Pancreatitis**, page 28) is more common than the acute form, but sonographic and other clinical findings overlap considerably between the 2 forms of disease.[1-3]

Diagnostic Challenges

Use of histopathology as the gold standard for diagnosis has recently been questioned because of the potential for histologic ambiguity.[3,4] A seminal paper exploring the prevalence and distribution of feline pancreatic pathologic abnormalities reported that 45% of cats that were apparently healthy at time of death had histologic evidence of pancreatitis.[1] The 41 cats in this group included cats with no history of disease that died of trauma, and cats from clinical studies that did not undergo any treatment (control animals). Conversely, multifocal distribution of inflammatory lesions was common in this study, raising the concern that lesions could be missed on biopsy or even necropsy.

Prevalence

Such considerations help explain the wide range in the reported prevalence of feline pancreatitis, from 0.6% to 67%.[3] The prevalence of clinically relevant pancreatitis undoubtedly lies somewhere in between, with acute and chronic pancreatitis suggested to represent opposite points on a disease continuum.[2]

ACUTE PANCREATITIS

Risk Factors

No age, sex, or breed predisposition has been recognized in cats with acute pancreatitis, and no relationship has been established with body condition score.[3-5]

- Cats over a wide age range, from kittens to geriatric cats, are affected; cats older than 7 years predominate.
- In most cases, an underlying cause or instigating event cannot be determined, leading to classification as *idiopathic*.[3]
- Abdominal trauma, sometimes from *high-rise syndrome*, is an uncommon cause that is readily identified from the history.[6]

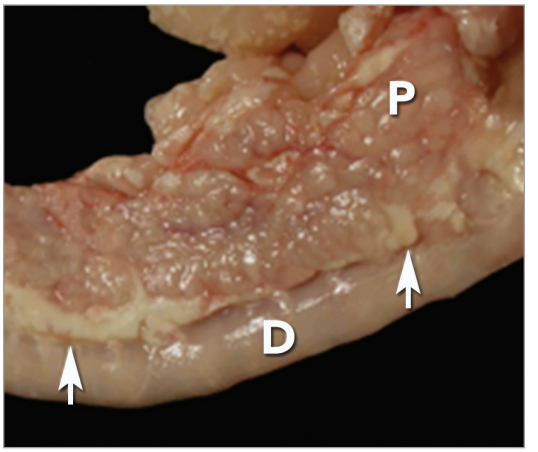

FIGURE 1. **Duodenum (D) and duodenal limb of the pancreas (P) in a cat with acute pancreatitis and necrosis; well-demarcated areas of necrosis are present at the periphery of the pancreas in the peripancreatic adipose tissue (arrows).**
Courtesy Dr. Arno Wuenschmann, Minnesota Veterinary Diagnostic Laboratory

- The pancreas is sensitive to hypotension and ischemia; every effort must be taken to avoid hypotensive episodes under anesthesia.

Comorbidities

In cats with acute pancreatitis, the frequency of concurrent diseases is as high as 83% (**Table 1**).[2]

- Pancreatitis complicates the management of some diabetic cats and may induce, for example, diabetic ketoacidosis.[7]
- Anorexia attributable to pancreatitis can be the precipitating cause of hepatic lipidosis.[8]
- The role of intercurrent inflammation in the biliary tract or intestine (also called triaditis) in the pathogenesis of pancreatitis is still uncertain.

Role of Bacteria

In one study, culture-independent methods to identify bacteria in sections of the pancreas from cats with pancreatitis detected bacteria in 35% of cases.[9] This report renewed speculation about the role of bacteria in the pathogenesis of acute pancreatitis, and the potential role that the common insertion of the pancreatic duct and common bile duct into the duodenal papilla may play in facilitating reflux of enteric bacteria into the "common channel" in cats. Awareness of triaditis may affect the diagnostic evaluation of individual patients.

DIAGNOSTIC EVALUATION

Many cats with pancreatitis have vague, nonspecific clinical signs, which make diagnosis challenging.[5] Clinical signs related to common comorbidities, such as anorexia, lethargy, and vomiting, may overlap with, or initially mask, the signs associated with pancreatic disease.

Early publications on the clinical characteristics of acute pancreatitis required necropsy as an inclusion criterion, presumably skewing the spectrum of severity of the reported cases.[5,8,10,11] Cats with chronic pancreatitis were excluded from these reports.

Clinical Findings

Table 1 lists common clinical findings in cats from necropsy-based reports and a recent series of 89 cats with acute pancreatitis studied by the authors.[12]

- Note the lower prevalence of most clinical findings in the cats diagnosed clinically rather than from necropsy records.
- In our evaluation of affected cats, 17% exhibited no signs aside from lethargy and 62% were anorexic.
- Vomiting occurs inconsistently (35%–52% of cats).
- Abdominal pain is detected in a minority of cases even when the index of suspicion of pancreatitis is high.
- About ¼ of cats with pancreatitis have a palpable abdominal mass that may be misdiagnosed as a lesion of another intra-abdominal structure.

Laboratory Analyses

Hematologic abnormalities in cats with acute pancreatitis are nonspecific; findings may include nonregenerative anemia, hemoconcentration, leukocytosis, or leukopenia.

Serum biochemical profile results vary (**Table 1**). In our acute pancreatitis case series, 33% of cats had no abnormalities in their chemistry results at presentation.[12]

Serum cholesterol concentrations may be high in up to 72% of cases. Some cases of acute pancreatitis are associated with severe clinical

High-rise syndrome

describes the phenomenon of cats falling from higher than 2 stories (23–30 feet); it also refers to injuries sustained during a fall.

TABLE 1.
Clinical Data from 95 Cats with Acute Pancreatitis (1976–1998; 59% Mortality Rate) & 89 Cats Diagnosed with Acute Pancreatitis (2004–2011; 16% Mortality Rate)

PARAMETER	HISTORICAL DATA*	CATS WITH PANCREATITIS†	SURVIVING CATS WITH PANCREATITIS†
Number of Cats	**95**	**89**	**75**
ALP elevation	50%	23%	18%
ALT elevation	68%	41%	36%
Apparent abdominal pain	25%	30%	32%
Cholangitis	NA	12%	11%
Concurrent disease diagnosed	NA	69%	68%
Dehydration	92%	37%	42%
Diabetic ketoacidosis	NA	8%	5%
Diabetes mellitus	NA	11%	12%
Fever	7%‡	26%	11%
GGT elevation	NA	21%	18%
Hepatic lipidosis	NA	20%	19%
Hyperbilirubinemia	64%	45%	53%
Icterus	64%	6%	6%
Vomiting	35%–52%	35%	36%

ALP = alkaline phosphatase; ALT = alanine aminotransferase; GGT = gamma glutamyl transferase; NA = not available

* *Summarized from 4 published case series; a total of 56 cats had acute pancreatitis diagnosed at necropsy and 3 by pancreatic biopsy*[5,8,10,11]
† *Data obtained from reference*[12]
‡ *68% of cats were hypothermic*

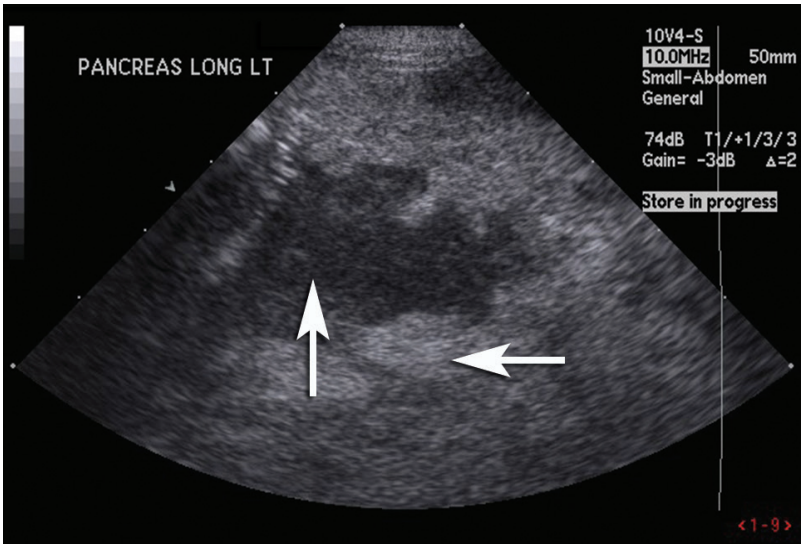

FIGURE 2. **Sagital sonographic image of the left limb of the pancreas in an 8-year-old neutered male Maine Coon cat. The cat presented with diabetic ketoacidosis (new diagnosis), evidence of abdominal pain, and a palpable midcranial abdominal mass. The pancreas is enlarged and hypoechoic** (up arrow) **with irregular margins. The mesentery adjacent to the pancreas** (horizontal arrow) **is hyperechoic (reactive). These changes are consistent with pancreatitis.** *Courtesy Dr. Kari Anderson, University of Minnesota Veterinary Medical Center*

Feline Pancreatic Lipase Assays

The **Spec fPL assay** (idexx.com) is a commercially available monoclonal enzyme-linked immunosorbent assay. A study presented in abstract form estimated the sensitivity and specificity of this test for diagnosing feline pancreatitis at 79% and 82%, respectively.[15]

Concentration results are considered:

- Diagnostic (positive) if **≥ 5.4 mcg/L**
- A gray zone if **> 3.5 mcg/L and < 5.4 mcg/L**
- Negative if **≤ 3.5 mcg/L**.

The **Snap fPL** (idexx.com) is a semiquantitative point-of-care test that can help rule out pancreatitis. A value of **> 3.5 mcg/L** is considered positive; therefore, a positive result must be confirmed by a Spec fPL assay.

With both tests, *positive results must be interpreted in light of other clinical information*, rather than considered an endpoint of diagnostic evaluation. After an episode of pancreatitis, the duration of fPL increase has not been reported.

Asymptomatic cats with persistently increased fPL concentrations may be encountered, especially if the fPL is included as a routine test in geriatric health panels. This may correlate with histologic evidence of pancreatitis reported in cats lacking clinical signs of disease.[1]

syndromes, such as shock, disseminated intravascular coagulation, and multiorgan failure, that influence some serum parameters, such as albumin, liver enzymes, and coagulation tests.

Plasma ionized calcium concentration may be low, and has been correlated with a poorer outcome.[11]

Serum amylase activity is of no clinical value in the clinical diagnosis of pancreatitis in cats; it actually decreases in experimental feline pancreatitis.[13] However, the serum activity of both amylase and lipase may increase whenever glomerular filtration rate is reduced.

Serum lipase activity is modestly increased early in experimentally induced disease, but is frequently normal in cats with spontaneous pancreatitis. A recent study found a high level of agreement between the 1,2-o-dilauryl-rac-glycero-3-glutaric acid-(6′-methylresorufin) ester lipase assay and feline pancreas-specific lipase assay, suggesting that the method used for lipase measurement may influence sensitivity and specificity.[14]

Serum pancreatic lipase (Spec fPL, idexx.com) is the serum test that provides the most useful information to support, or exclude, a diagnosis of pancreatitis (see **Feline Pancreatic Lipase Assays**).

Abdominal Radiography

Exclusion of other causes of vague gastrointestinal signs, such as partial intestinal obstruction, is a major rationale for survey abdominal radiography in cats with clinical signs compatible with pancreatitis. Thoracic radiographs may detect pleural fluid or pulmonary edema, both of which have been associated with acute pancreatitis and other complications, such as pneumonia.

Abdominal Ultrasonography

Abdominal ultrasonography is a *key diagnostic test* in cats suspected of having pancreatitis; **Table 2** lists the most important ultrasound findings.

TABLE 2.
Important Ultrasound Findings in Cats with Pancreatitis[14,16-17]

- Increased echogenicity of mesenteric fat immediately surrounding the pancreas*
- Increased pancreatic thickness (enlarged pancreas)
- Irregular pancreatic margins
- Peripancreatic free fluid
- Hypoechoic, hyperechoic, *or* mixed-echoic pancreas
- Mass effect in cranial abdomen
- Dilated common bile duct

** Abnormality with highest sensitivity, based on recent study[16]*

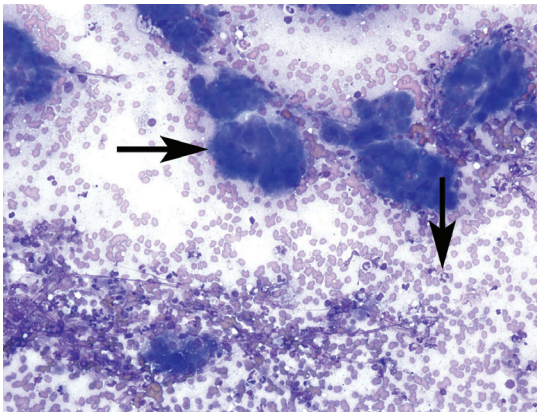

FIGURE 3. **Pancreatic aspirate from a cat with pancreatitis (200×, Wright's-Giemsa stain). There are multiple cohesive clusters of slightly hyperplastic pancreatic exocrine cells characterized by increased cytoplasmic basophilia (**horizontal arrow**). The background contains blood and increased numbers of neutrophils (**vertical arrow**) with occasional foamy macrophages.** *Courtesy of Dr. Leslie Sharkey, University of Minnesota Veterinary Medical Center*

The reported sensitivity of abdominal ultrasound for detecting feline pancreatitis varies widely (11%– 68%),[16] even when performed by board-certified radiologists. Therefore, some cats with biopsy-confirmed acute pancreatitis have no detectable sonographic abnormalities. However, the sensitivity of ultrasonography increases with increasing severity of pancreatitis.[18]

Abnormal sonographic findings are highly specific for pancreatitis—a cat with compatible clinical signs and visible changes in the pancreas is very likely to be correctly diagnosed with pancreatitis (**Figure 2**).

Fine-Needle Aspiration

Ultrasound-guided fine-needle aspirates of the pancreas and/or peripancreatic tissue may assist in the diagnosis of pancreatitis (**Figure 3**), and may also be helpful when nodular changes are present.[4,12]

INITIAL THERAPY

The initial medical management of cats with acute pancreatitis must not be delayed until a diagnosis is confirmed. In experimental studies, a major factor in the progression of mild pancreatitis to severe pancreatitis is disturbed pancreatic microcirculation.

Early IV fluid therapy with a balanced, isotonic replacement crystalloid (eg, lactated Ringer's solution, 0.9% saline, Plasma-Lyte 156, Normosol-R), supplemented with potassium and glucose as necessary, is recommended. This emphasis on early fluid resuscitation is consistent with human treatment guidelines for acute pancreatitis and *is of critical importance*.[19]

Potassium supplementation (up to 20–30 mEq potassium chloride/L of fluids) is necessary to replace losses and address reduced intake, and should be monitored by serial measurement of serum potassium levels. The level of supplementation may need to be reduced in patients with mild clinical signs, or increased in patients with concurrent diabetic ketoacidosis.

Calcium gluconate (50–150 mg/kg IV as a slow bolus) may be required for symptomatic hypocalcemia (tremors, seizure activity), a possible complication of acute pancreatitis, and serum ionized calcium concentrations should be monitored regularly during calcium therapy. Begin with a portion of this dose, and discontinue if ionized calcium normalizes. Continuous low-dose IV infusions of calcium gluconate (5–10 mg/kg/H IV) are required by some cats.

Insulin therapy is initiated in diabetic patients.[5]

Colloids, such as hydroxyethyl starch, are useful when hypoproteinemia is present, and may have antithrombotic effects that help maintain microcirculation. However, use of synthetic colloids in companion animals is increasingly being debated due to adverse effects on renal function noted in human patients.[20]

Plasma transfusion theoretically provides a source of circulating protease inhibitors, but numerous human studies fail to support its use, and 1 retrospective canine pancreatitis study failed to demonstrate a benefit.

Learn More

Read **Treatment Guidelines for Acute Pancreatitis in Cats** on page 31 for in-depth information on therapeutic approaches, medications, and dosages.

MEDICAL THERAPY

Antiemetics

Nausea and vomiting may be severe in patients with acute pancreatitis.

- The potent antiemetic **maropitant**, an NK_1 receptor antagonist, is useful for controlling emesis (and probably nausea) and providing visceral analgesia.[21]
- An alternative antiemetic is a 5-HT_3 antagonist (**ondansetron** or **dolasetron**), which may be combined with maropitant in severe cases.
- The dopaminergic antagonist **metoclopramide** may help enhance motility in the upper gastrointestinal tract. It acts as a weak peripherally acting antiemetic in dogs, but this effect is questionable in cats.
- The histamine-2 receptor antagonist **ranitidine** may be selected for dual acid inhibition and prokinetic effects. Correction of hypokalemia also helps improve gastrointestinal motility.

Gastroprotectants

Gastric acid suppression is commonly incorporated into therapy for feline acute pancreatitis. The rationale includes protecting:

- The esophagus from exposure to gastric acid during episodes of vomiting
- Against gastric ulceration, to which patients with pancreatitis may be predisposed due to hypovolemia and local peritonitis.

Higher gastric pH may decrease exocrine pancreatic stimulation but remains undocumented as a treatment for pancreatitis.

When gastric acid suppression is desired, a proton-pump inhibitor (**pantoprazole**) may be preferred over a histamine-2 receptor antagonist; an experimental study in rats demonstrated that pantoprazole reduced inflammatory changes and leakage of pancreatic acinar cells.[22]

When a histamine-2 receptor antagonist is used, famotidine is believed to be most effective for suppression of gastric acid production.

Analgesics

Pain management is a critical aspect of treating acute pancreatitis, and can be easily overlooked because cats may not exhibit easily recognized signs of pain. Analgesia can be provided using **opioids**, such as buprenorphine or fentanyl, delivered by IV or SC injection, sublingual route, or transdermal patch.

Convincing evidence suggests that the antiemetic **maropitant** also provides visceral analgesia.[21] Tramadol is usually avoided in cats because it can cause severe dysphoria.

Antibiotics

Acute pancreatitis is thought to begin as a sterile process, and reports of bacterial complications, such as pancreatic abscessation, are uncommon. Broad-spectrum antibiotics may be warranted in cats with complete blood count findings suggestive of sepsis but, otherwise, are *not* routinely used.

However, a recent study in cats using culture-independent methods[9] suggested that bacterial infection may warrant greater consideration. Coliforms are the principal pathogens, and are also seen in bile cultures from cats with cholangitis.[23]

Glucocorticoids

Historical reluctance to use corticosteroids for treating pancreatitis has been based on concern that these agents could lead to pancreatitis; however, no evidence supports this assumption in cats.

Corticosteroids exert broad anti-inflammatory effects, and may have a role in increasing production of pancreatitis-associated protein, which helps protect against inflammation. They may also address critical illness-related corticosteroid insufficiency, a relative adrenal insufficiency that could occur in acute pancreatitis.

Steroid use in cats with acute pancreatitis is being reconsidered but remains unexamined. There is no existing data supporting the use of corticosteroids in feline pancreatitis, and care must be exercised when considering their use in cats with diabetes. Judicious short-term corticosteroid administration may be considered in a cat with severe acute pancreatitis that is failing to respond to other therapies.

NUTRITIONAL THERAPY

In cats suspected of having acute pancreatitis, oral intake has historically been initially withheld for 24 to 48 H; then gradually re-introduced, as tolerated. The theory behind this rationale—which has come under close scrutiny in human and veterinary medicine—was to "rest" the pancreas by decreasing pancreatic stimulation.

Clinical and experimental data support the concept that nutritional management plays an important therapeutic role in recovery from acute pancreatitis. The current standard of care—which attempts to maintain enterocyte integrity, reduce risk for bacterial translocation, and attenuate the systemic inflammatory response—is to:[24,25]

- Administer **antiemetics** immediately upon presentation; then as required to control vomiting
- Begin **enteral feeding** as soon as possible
- Only implement **parenteral nutrition** in patients in which refractory vomiting precludes enteral support (rare). A small prospective controlled study in dogs with acute pancreatitis demonstrated that the group fed via esophagostomy tube had significantly fewer episodes of vomiting or regurgitation compared with the group fed parenterally.[26]

A nasoesophageal tube or esophagostomy tube may be used to provide nutritional support to cats with acute pancreatitis; this also helps treat or prevent concurrent hepatic lipidosis. A liquid diet must be fed through a nasoesophageal tube (see page 31); a variety of diets will pass through an esophagostomy tube. The volume of food fed is increased toward calculated resting energy requirement as tolerance permits.

SURGICAL THERAPY

Exploratory laparotomy (or laparoscopy) to obtain pancreatic biopsy specimens in a cat suspected of having acute pancreatitis is not indicated, but pancreatic biopsy can be performed with relative safety if the abdomen is being explored for other reasons.[27]

Surgery is rarely needed to remove devitalized or infected tissue. Serum bilirubin may remain increased for weeks during apparent recovery from a bout of pancreatitis, but surgery is only occasionally required to relieve an obstruction of the common bile duct.[28]

Fluid accumulation within the pancreas usually resolves spontaneously.

fPL = feline pancreatic lipase

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In Brief: Diagnosis & Treatment of Feline Chronic Pancreatitis

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Treatment Guidelines

See **Treatment Guidelines for Acute Pancreatitis** on page 31 for dosages of mirtazapine, maropitant, and SAMe.

Chronic pancreatitis appears to be much more common in cats than acute pancreatitis (**Figure 1**).[1-4] Unfortunately, there is poor correlation between the usual clinical definition of chronic (time course) and histologic definition of chronic (fibrosis, lymphocytic inflammation, and acinar atrophy) (**Figure 2**). Adding to the complexity of feline chronic pancreatitis is evidence that it can be very mild or asymptomatic and has a high histologic prevalence in apparently healthy cats.[1]

DIAGNOSTIC EVALUATION

Histologic features of acute and chronic pancreatitis overlap somewhat, suggesting that they may represent different points on a disease spectrum.

In the Literature

One necropsy-based study of 63 cats could not identify clinical features helpful in distinguishing acute from chronic pancreatitis.[5]

- Notably, abdominal ultrasonography results were unremarkable in 50% of all cats; when pancreatic abnormalities were detected, findings did not differ between cats with acute and those with chronic pancreatitis.
- Concurrent disease was common in both groups of cats, but 100% of cats with chronic pancreatitis had one or more concurrent diseases (**Table**).

Ultrasonography Findings

Ultrasonographic findings in cats with histologically confirmed chronic pancreatitis overlap considerably with findings in cats with acute pancreatitis (**Table**).[5,6] Given the clinical and histologic overlap between these forms of the disease, this is not surprising.

TABLE.
Common Findings in Feline Chronic Pancreatitis

FREQUENT CONCURRENT DISEASES	ULTRASOUND FINDINGS
Diabetes mellitus Gastrointestinal disease Hepatobiliary disease Renal disease	Dilated common bile duct Enlarged pancreas Hyperechoic or mixed echogenicity pancreas Irregular pancreatic margins Peripancreatic free fluid

Fine-Needle Aspiration

Nodular changes may develop; fine-needle aspiration cytology may provide a useful minimally invasive method of investigation.[3,7]

THERAPEUTIC MEASURES

Nutritional Therapy

There is no evidence that a low-fat diet helps treat or prevent pancreatitis in cats. The authors' choice is to feed cats with a history of pancreatitis a diet high in antioxidants and provide S-adenosyl methionine (SAMe) as an antioxidant supplement. A highly digestible diet with a novel or hydrolyzed protein source may be of benefit if concurrent inflammatory bowel disease is present.

Medical Therapy

Anti-inflammatory doses of prednisolone (2.5–5 mg/cat Q 48–72 H) are increasingly being used

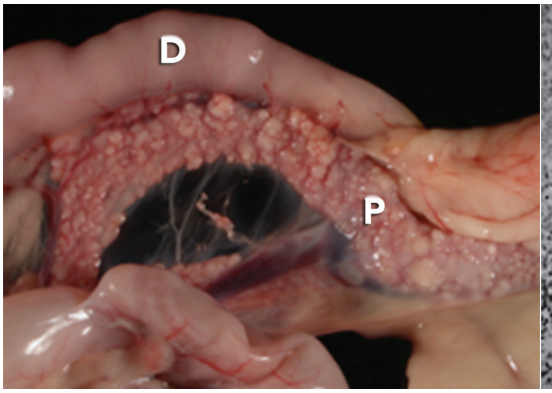

FIGURE 1. **Duodenum (D) and duodenal limb of the pancreas (P) in a cat with chronic pancreatitis and nodular regeneration of the exocrine pancreas; the entire parenchyma is composed of regenerative nodules.**

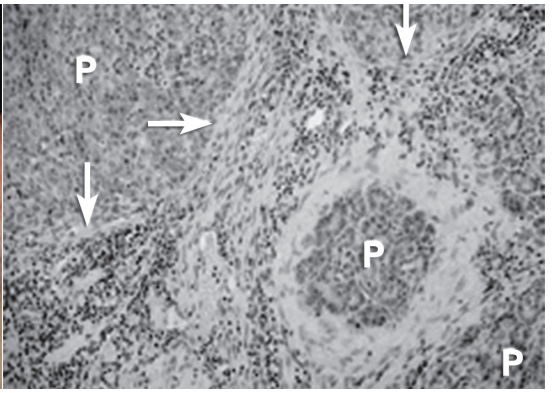

FIGURE 2. **Histomicrograph of the pancreas (P) with chronic fibrosing lymphoplasmacytic pancreatitis and nodular regeneration; the parenchyma is dissected by strands of fibrous tissue infiltrated by lymphocytes and plasma cells (arrows). Hematoxylin and eosin stain, 20× magnification.**

Figures courtesy Dr. Arno Wuenschmann, Minnesota Veterinary Diagnostic Laboratory

in cats with presumed chronic (or intermittent relapsing) pancreatitis, along with:

- Mirtazapine on an ongoing basis as needed for appetite stimulation (mirtazapine appears to have an antiemetic effect as well)
- Maropitant at the first indication of relapse (declining appetite, apparent nausea, or vomiting).

Coexisting conditions, such as cholangitis and inflammatory bowel disease, are common in cats with pancreatitis and often must be managed concurrently. No evidence suggests that steroid use is problematic in cats with chronic pancreatitis.

Exocrine Pancreatic Insufficiency

Exocrine pancreatic insufficiency is more common in cats than previously believed, and most cases are due to chronic pancreatitis.[8]

Measurement of **serum trypsin-like immunoreactivity (TLI)** is recommended in cats with weight loss, loose stools, and/or polyphagia. Some cats have greasy soiling of the hair in the perianal region, and most cats with exocrine pancreatic insufficiency have a severely decreased **serum cobalamin concentration**, which can lead to various gastrointestinal and systemic complications and to treatment failure.

Pancreatic enzyme supplements are used in some humans with chronic pancreatitis with normal exocrine pancreatic function because they are associated with decreased frequency and intensity of episodes of abdominal pain.[2,3] This has not been investigated in cats, but there are anecdotal reports that this therapy improves appetite and gastrointestinal signs in cats with chronic pancreatitis.[2]

SAMe = S-adenosyl methionine; TLI = trypsin-like immunoreactivity

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Treatment Guidelines for Acute Pancreatitis in Cats

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Read **Feline Acute Pancreatitis: Current Concepts in Diagnosis & Therapy** on page 22 for further details.

TREATMENT	DOSE	COMMENTS
1. FLUID THERAPY		
Balanced isotonic replacement crystalloid	Maintenance at 40–60 mL/kg Q 24 H Additional replacement of ongoing losses may be required	Rehydrate according to speed of losses, monitor weight and urine production, and account for cardiovascular disease
Potassium chloride	20–30 mEq/L to start; adjust depending on: • Serum potassium values • Fluid rate	As indicated by potassium deficit: • Replace total body losses resulting from vomiting or anorexia, or if managing diabetes • Reduce in less symptomatic patients
Calcium gluconate	50–150 mg/kg IV bolus (if symptomatic for hypocalcemia) 5—10 mg/kg/H IV CRI (if needed)	With serial monitoring, discontinue when patient normalizes
2. ANTIEMETIC THERAPY		
Maropitant	1 mg/kg SC, PO, *or* IV Q 24 H	Refrigerate to reduce pain associated with SC injection IV use is extralabel Provides visceral analgesia
Ondansetron	0.5–1 mg/kg IV Q 12 H *or* 24 H	Administer IV injection slowly
Dolasetron	0.5–1 mg/kg PO *or* SC Q 12 H *or* 24 H	
3. PROKINETIC THERAPY		
Metoclopramide	0.2–0.4 mg/kg SC *or* PO Q 6–8 H 1–2 mg/kg IV/day CRI Q 24 H	Watch for drug interactions May induce neurologic signs Ineffective antiemetic in cats
4. GASTROPROTECTANT THERAPY		
Pantoprazole	1 mg/kg IV Q 24 H	
Omeprazole	0.7–1 mg/kg PO Q 24 H	May reduce absorption of other medications
Famotidine	0.5–1 mg/kg IV *or* PO Q 12 H *or* 24 H	Administer IV injection slowly to avoid hypotension Reports of intravascular hemolysis when IV used in cats
Ranitidine	1–2 mg/kg IV *or* PO Q 12 H	Mild prokinetic effect Administer IV injection slowly to avoid hypotension
5. ANALGESIC THERAPY		
Buprenorphine	0.005–0.02 mg/kg SC, IV, *or* sublingual Q 6–8 H	Adverse effects uncommon Can produce sedation
Fentanyl	Loading dose of 1–4 mcg/kg; then 1–4 mcg/kg/H IV CRI per H	Do *not* combine with buprenorphine or butorphanol
Fentanyl patch	12.5 mcg per H patch *or* 25 mcg per H patch	Patch lasts 3–4 days once applied Effect noted in 6–12 H May cover half of 25-mcg patch if needed
Butorphanol	0.1–0.2 mg/kg per H IV CRI, after loading dose of 0.1–0.2 mg/kg IV 0.2–0.4 mg/kg SC, IM, *or* IV	May not provide sufficient analgesia when used alone; often combined with ketamine (loading dose, 0.1 mg/kg IV; IV CRI, 0.4 mg/kg/H) Intermittent administration only may provide analgesia for 1 H or less
6. ANTIBIOTIC THERAPY		
Ampicillin **Amoxicillin**	10–20 mg/kg IV *or* SC Q 8 H 10–20 mg/kg PO Q 12 H	Useful in combination with metronidazole Ampicillin has poor bioavailability when administered orally
Ampicillin/amoxicillin with sulbactam	20–30 mg/kg IV Q 8 H 62.5 mg/cat PO Q 12 H	Sulbactam may cause diarrhea/inappetence
Enrofloxacin	5 mg/kg IV *or* PO Q 24 H	Useful in combination with metronidazole
Marbofloxacin	2.5–5 mg/kg PO Q 24 H	Useful in combination with metronidazole
Metronidazole	10–15 mg/kg IV *or* PO Q 12 H	Not suitable for use alone Best in combination with aerobic/gram-positive targeting therapy

TREATMENT	DOSE	COMMENTS
7. NUTRITIONAL THERAPY		
Nutrition Clinicare Canine/ Feline Liquid Diet *or* Vivonex (1 kcal/mL)	Via nasoesophageal tube as: • Continuous infusion • Multiple small bolus feedings	Achieve RER over 3–5 days: *Body weight (kg)$^{0.75}$ × 70* Reduced-fat diet not indicated in cats
Mirtazapine (appetite stimulant)	1.88–3.75 mg/cat PO Q 2–3 days	Also has antiemetic effect Main side effect is increased affection Sedation is dose-related Do *not* administer with tramadol
8. OTHER THERAPIES		
Regular insulin	Based on blood glucose monitoring: 0.1–0.2 U/kg SC *or* IM Q 4 H 1.1 U/kg per day IV CRI, with or without dextrose supplementation	For patients with diabetic ketoacidosis or inappetent diabetic patients Strict monitoring of blood glucose required Pancreatitis may destabilize a previously controlled diabetic patient
S-adenosyl methionine (SAMe)	Cats < 5 kg: 90 mg PO Q 24 H Cats > 5 kg: 180 mg *or* 225 mg PO Q 24 H	Give intact tablet on an empty stomach; may be useful with concurrent liver disease
9. VITAMIN THERAPY		
Cyanocobalamin (Vitamin B_{12})	250 mcg/cat SC weekly for 6 weeks, then 30 days later; retest after 30 days	Empirical use or based on serum concentration May be useful in cats with concurrent IBD or hepatic lipidosis
B vitamin complex	1–2 mL/L of IV fluids	May be useful in cats with prolonged anorexia
Vitamin K_1	0.5–1.5 mg/kg SC Q 12 H	May be useful in hyperbilirubinemic patients (ie, concurrent hepatic lipidosis) Use 25-gauge needle; avoid IV use due to risk of anaphylaxis

CRI = constant-rate infusion; IBD = inflammatory bowel disease; RER = resting energy requirement