Feline Acute Pancreatitis

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Pancreatitis appears to be a common disease in cats, 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). 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.

Diagnostic Challenges

Use of histopathology as the gold standard for diagnosis has recently been questioned because of the potential for histologic ambiguity. 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. 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%. 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.

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.

- 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.
- Abdominal trauma, sometimes from high-rise syndrome, is an uncommon cause that is readily identified from the history.

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

<p>| TABLE 1. Clinical Data from 95 Cats with Acute Pancreatitis (1976–1998; 59% Mortality Rate) &amp; 89 Cats Diagnosed with Acute Pancreatitis (2004–2011; 16% Mortality Rate) |</p>
<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>HISTORICAL DATA*</th>
<th>CATS WITH PANCREATITIS†</th>
<th>SURVIVING CATS WITH PANCREATITIS‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Cats</td>
<td>95</td>
<td>89</td>
<td>75</td>
</tr>
<tr>
<td>ALP elevation</td>
<td>50%</td>
<td>23%</td>
<td>18%</td>
</tr>
<tr>
<td>ALT elevation</td>
<td>68%</td>
<td>41%</td>
<td>36%</td>
</tr>
<tr>
<td>Apparent abdominal pain</td>
<td>25%</td>
<td>30%</td>
<td>32%</td>
</tr>
<tr>
<td>Cholangitis</td>
<td>NA</td>
<td>12%</td>
<td>11%</td>
</tr>
<tr>
<td>Concurrent disease diagnosed</td>
<td>NA</td>
<td>69%</td>
<td>68%</td>
</tr>
<tr>
<td>Dehydration</td>
<td>92%</td>
<td>37%</td>
<td>42%</td>
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<tr>
<td>Diabetic ketoadiposis</td>
<td>NA</td>
<td>8%</td>
<td>5%</td>
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<tr>
<td>Diabetes mellitus</td>
<td>NA</td>
<td>11%</td>
<td>12%</td>
</tr>
<tr>
<td>Fever</td>
<td>7%†</td>
<td>26%</td>
<td>11%</td>
</tr>
<tr>
<td>GGT elevation</td>
<td>NA</td>
<td>21%</td>
<td>18%</td>
</tr>
<tr>
<td>Hepatic lipidosis</td>
<td>NA</td>
<td>20%</td>
<td>19%</td>
</tr>
<tr>
<td>Hyperbilirubinemia</td>
<td>64%</td>
<td>45%</td>
<td>53%</td>
</tr>
<tr>
<td>Icterus</td>
<td>64%</td>
<td>6%</td>
<td>6%</td>
</tr>
<tr>
<td>Vomiting</td>
<td>35%–52%</td>
<td>35%</td>
<td>36%</td>
</tr>
</tbody>
</table>

* Summarized from 4 published case series; a total of 56 cats had acute pancreatitis diagnosed at necropsy and 3 by pancreatic biopsy5,8,10,11
† Data obtained from reference12
‡ 68% of cats were hypothermic
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>
<thead>
<tr>
<th>Important Ultrasound Findings in Cats with Pancreatitis14,16-17</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Increased echogenicity of mesenteric fat immediately surrounding the pancreas*</td>
</tr>
<tr>
<td>• Increased pancreatic thickness (enlarged pancreas)</td>
</tr>
<tr>
<td>• Irregular pancreatic margins</td>
</tr>
<tr>
<td>• Peripancreatic free fluid</td>
</tr>
<tr>
<td>• Hypoechoic, hyperechoic, or mixed-echoic pancreas</td>
</tr>
<tr>
<td>• Mass effect in cranial abdomen</td>
</tr>
<tr>
<td>• Dilated common bile duct</td>
</tr>
</tbody>
</table>

*Abnormality with highest sensitivity, based on recent study16
The reported sensitivity of abdominal ultrasound for detecting feline pancreatitis varies widely (11%–68%), 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.

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.

**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.*

**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.

**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.

**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.

**MEDICAL THERAPY**

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

- The potent antiemetic maropitant, an NK₁ receptor antagonist, is useful for controlling emesis (and probably nausea) and providing visceral analgesia.

- An alternative antiemetic is a 5-HT₃ 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 methods9 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:

- 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.

\[ fPL = \text{feline pancreatic lipase} \]

References


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Sarah Crain, DVM, MS, Diplomate ACVIM, is currently in a PhD program at Cummings School of Veterinary Medicine at Tufts University, exploring the utility and mechanism of stem cell therapies for canine inflammatory diseases. Dr. Crain received her DVM at University of Minnesota, where she also completed a residency in small animal internal medicine following an internship at North Carolina State University. Dr. Crain’s primary professional interests are gastrointestinal disease and auto-immune/hematologic diseases.
In Brief:
Diagnosis & Treatment of Feline Chronic Pancreatitis

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).3,6 Given the clinical and histologic overlap between these forms of the disease, this is not surprising.

**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

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**TABLE.**
Common Findings in Feline Chronic Pancreatitis

<table>
<thead>
<tr>
<th>FREQUENT CONCURRENT DISEASES</th>
<th>ULTRASOUND FINDINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>Dilated common bile duct</td>
</tr>
<tr>
<td>Gastrointestinal disease</td>
<td>Enlarged pancreas</td>
</tr>
<tr>
<td>Hepatobiliary disease</td>
<td>Hyperechoic or mixed echogenicity pancreas</td>
</tr>
<tr>
<td>Renal disease</td>
<td>Irregular pancreatic margins</td>
</tr>
<tr>
<td></td>
<td>Peripancreatic free fluid</td>
</tr>
</tbody>
</table>

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**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.
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

**References**

## Treatment Guidelines for Acute Pancreatitis in Cats

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Sarah Crain, DVM, MS, Diplomate ACVIM, Tufts University

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

<table>
<thead>
<tr>
<th>TREATMENT</th>
<th>DOSE</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. FLUID THERAPY</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| 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 |
| Ondansetron | 0.5–1 mg/kg IV Q 12 H or 24 H | Provides visceral analgesia |
| 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 | 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 |
| Amoxicillin | 20–30 mg/kg IV Q 8 H  
62.5 mg/cat PO Q 12 H | Sulbactam may cause diarrhea/appetence |
| Ampicillin/amoxicillin with sulbactam | 5 mg/kg IV or PO Q 24 H | Use in combination with metronidazole |
| Enrofloxacin | 2.5–5 mg/kg PO Q 24 H | Use in combination with metronidazole |
| Marbofloxacin | 10–15 mg/kg IV or PO Q 12 H | Not suitable for use alone  
Best in combination with aerobic/gram-positive targeting therapy |
### 7. NUTRITIONAL THERAPY

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nutrition</td>
<td>Via nasoesophageal tube as:</td>
<td>Achieve RER over 3–5 days:</td>
</tr>
<tr>
<td>Clinicare Canine/</td>
<td>1. Continuous infusion</td>
<td>Body weight (kg)(^{0.75} \times 70)</td>
</tr>
<tr>
<td>Feline Liquid Diet or</td>
<td>2. Multiple small bolus</td>
<td>Reduced-fat diet not indicated in cats</td>
</tr>
<tr>
<td>Vivonex (1 kcal/mL)</td>
<td>feedings</td>
<td></td>
</tr>
<tr>
<td>Mirtazapine (appetite</td>
<td>1.88–3.75 mg/cat PO Q 2–3</td>
<td>Also has antiemetic effect</td>
</tr>
<tr>
<td>stimulant)</td>
<td>days</td>
<td>Main side effect is increased affection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sedation is dose-related</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Do not administer with tramadol</td>
</tr>
</tbody>
</table>

### 8. OTHER THERAPIES

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular insulin</td>
<td>Based on blood glucose</td>
<td>For patients with diabetic ketoacidosis or inappetent diabetic patients</td>
</tr>
<tr>
<td></td>
<td>monitoring:</td>
<td>Strict monitoring of blood glucose required</td>
</tr>
<tr>
<td></td>
<td>1. 0.1–0.2 U/kg SC or IM</td>
<td>Pancreatitis may destabilize a previously controlled diabetic patient</td>
</tr>
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<td></td>
<td>Q 4 H</td>
<td></td>
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<tr>
<td></td>
<td>2. 1.1 U/kg per day IV CRI,</td>
<td></td>
</tr>
<tr>
<td></td>
<td>with or without dextrose</td>
<td></td>
</tr>
<tr>
<td></td>
<td>supplementation</td>
<td></td>
</tr>
<tr>
<td>S-adenosyl methionine</td>
<td>Cats &lt; 5 kg: 90 mg PO Q 24 H</td>
<td>Give intact tablet on an empty stomach; may be useful with concurrent liver disease</td>
</tr>
<tr>
<td>(SAMe)</td>
<td>Cats &gt; 5 kg: 180 mg or 225</td>
<td></td>
</tr>
<tr>
<td></td>
<td>mg PO Q 24 H</td>
<td></td>
</tr>
</tbody>
</table>

### 9. VITAMIN THERAPY

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyanocobalamin</td>
<td>250 mcg/cat SC weekly for 6</td>
<td>Empirical use or based on serum concentration</td>
</tr>
<tr>
<td>(Vitamin B(_{12})</td>
<td>weeks, then 30 days;</td>
<td>May be useful in cats with concurrent IBD or hepatic lipidosis</td>
</tr>
<tr>
<td></td>
<td>retest after 30 days</td>
<td></td>
</tr>
<tr>
<td>B vitamin complex</td>
<td>1–2 mL/L of IV fluids</td>
<td>May be useful in cats with prolonged anorexia</td>
</tr>
<tr>
<td>Vitamin K(_{1})</td>
<td>0.5–1.5 mg/kg SC Q 12 H</td>
<td>May be useful in hyperbilirubinemic patients (ie, concurrent hepatic lipidosis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use 25-gauge needle; avoid IV use due to risk of anaphylaxis</td>
</tr>
</tbody>
</table>

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