Feline Pancreatitis

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Profile

Definition
- Feline pancreatitis is increasingly diagnosed in clinical practice, likely secondary to increased diagnostic sensitivity.
- Feline pancreatitis can be acute (neutrophilic), chronic (lymphocytic, plasma-cytic, lymphoplasmacytic with fibrosis), or acute-on-chronic (neutrophilic and lymphocytic).
- Recurrent episodes require long-term medical management.

Systems
- Effects range from mild GI signs (eg, hyporexia, anorexia) to severe systemic inflammatory response syndrome (SIRS) that may lead to multiorgan dysfunction.

Incidence & Prevalence
- No definitive incidence of feline pancreatitis is known, but recent necropsy evidence suggests it is underdiagnosed.

Signalment
- Pancreatitis can affect any breed of cat at any age.
- It is most common in middle-aged to older patients.

Causes
- As with canine pancreatitis, the underlying cause is poorly understood.
- Affected cats may be overweight or have a history of pancreatitis (acute or chronic).
- Trauma (eg, fall from a height) has been associated with pancreatitis.
- Dietary indiscretion is a widely accepted inciting cause of canine pancreatitis, but it is a less common cause in cats.
- Certain toxins (eg, zinc, castor beans), infections (eg, *Toxoplasma gondii*), medications (see Drugs Associated with Feline Pancreatitis, page 20), and history of pancreatic ischemia with hypotension and/or surgical manipulation have been associated with feline pancreatitis.
- Feline pancreatitis is most commonly idiopathic in origin.

Risk Factors
- Inflammatory bowel disease, cholangitis, hepatitis (infectious or inflammatory), endocrine disease (eg, diabetes mellitus), and severe systemic illness are common comorbidities.

Feline pancreatitis produces a diverse array of effects ranging from mild GI signs (eg, hyporexia, anorexia) to severe systemic inflammatory response syndrome (SIRS) that may lead to multiorgan dysfunction.
Pathophysiology

- Results from activation of the potent pancreatic enzymes and local and systemic consequences of the ensuing inflammation (regardless of underlying cause).
- Intrapancreatic digestive enzyme activation is prevented by several mechanisms, including storage as zymogens in an inactive form, separation in the endoplasmic reticulum, and the requirement of trypsin for activation.
  - Trypsin is maintained as the inactive trypsinogen until enterokinase activation; enterokinase is secreted by enterocytes in the GI lining.
- Local and systemic inhibitors of activation include pancreatic secretory trypsin inhibitor and circulating enzyme inhibitor proteins.
- Proteolytic enzyme activation and neutrophil recruitment result in production of inflammatory mediators (eg, oxygen free radicals, phospholipase), which disrupt cellular membranes and exacerbate the cycle.
- Local inflammation can result in increased capillary permeability and pancreatic edema, necrosis, and hemorrhage.
- Respiratory difficulty (ie, acute respiratory distress syndrome), focal or diffuse peritonitis, renal injury, hepatic injury, and disseminated intravascular coagulation (DIC) may occur in severely affected patients.

Clinical Signs

- Presenting signs are typically vague and nonspecific.
  - Anorexia and lethargy are most common.
- Unlike dogs, cats have a lower incidence of specific signs (eg, vomiting, abdominal pain).
- Fever may occur, dehydration is common, and icterus may be present in cases with functional biliary obstruction, concurrent hepatic lipidosis, or severe cholangiohepatitis (Figure 1).

Laboratory Findings

- Routine laboratory tests have poor sensitivity and specificity for pancreatitis.
- Hematologic abnormalities supportive of pancreatitis may include an inflammatory leukogram (± left shift) and thrombocytopenia.
  - CBC is often normal.
- Mild to moderate increases in liver enzyme activity and bilirubin concentration may indicate hepatic lipidosis and/or cholangiohepatitis.
- Electrolyte and acid-base abnormalities are typically secondary to decreased fluid intake or fluid losses from vomiting and diarrhea.
  - Dehydration can result in azotemia; systemic protein loss and decreased albumin production can result in hypoalbuminemia.
- Serum amylase and lipase are neither specific nor sensitive.
- Small studies have demonstrated that commercial laboratory and bedside SNAP feline-specific pancreatic lipase (fPLI) tests are more specific (79.7%) and sensitive (79.4%) than other chemistry analyses (ie, trypsin-like immunoreactivity [TLI], amylase, lipase).2

Yellow discoloration of oral mucous membranes of a cat with pancreatitis. This may occur from functional biliary obstruction, secondary hepatic lipidosis, or concurrent cholangiohepatitis.

Diagnosis

- Diagnosis can be extremely challenging.
- A high index of suspicion is important.
  - Clinical suspicion, imaging, and pathology can help diagnosis.
- Routine diagnostic testing can rule in or out other common acute abdominal disease:
  - Gastroenteritis, hemorrhagic gastroenteritis (rare in cats), toxin ingestion, hepatobiliary disease, primary infiltrative GI disease, renal failure, lower urinary tract disease, liver failure, organ torsion, obstruction
- Examination of pancreatic biopsy and histopathologic examination of tissue is the gold standard for diagnosis.
  - Most patients do not require surgical intervention.
Crystalloid fluid supplementation is the primary therapeutic modality to correct perfusion deficits and dehydration; targeted supplementation should continue for ongoing losses and maintenance.

Patients with SIRS resulting in increased capillary permeability and protein loss may require colloid supplementation (e.g., hydroxyethyl starches) to maintain intravascular oncotic pressure.

Because of the diversity of presentations, the author recommends monitoring based on Kirby’s Rule of 20 (i.e., a set of clinical considerations to be evaluated daily). Hospitalization is indicated for severe cases and for patients that fail outpatient therapy.

**Imaging**

- Abdominal radiographs may show a focal loss of serosal detail in the right upper quadrant (i.e., ground glass appearance) and a wide angle between the stomach antrum and duodenum.
- These findings are often nonspecific.
- Ultrasonography may disclose an enlarged hypoechoic pancreas or one of mixed echogenicity (Figure 2), variable biliary distention or functional obstruction, and small amounts of free abdominal fluid consistent with focal peritonitis.
- Ultrasonography has a sensitivity of 73%–85% (or more) and a specificity of 24%–67%.
- This depends on equipment and sonographer experience.
- Ultrasonography may often be normal.
- The author frequently uses ultrasonography to help rule out other causes of hyporexia.

- Advanced imaging (e.g., CT) may be sensitive but can be limited by cost.
- MRI with cholangiopancreatography and endosonography has been used to diagnose pancreatitis in cats and may be useful for those with equivocal ultrasound imaging.

**Treatment**

**Inpatient or Outpatient**

- Therapy is often based on treatment or elimination of underlying causes and supportive and symptomatic care.

**Medical**

- Crystalloid fluid supplementation is the primary therapeutic modality to correct perfusion deficits and dehydration; targeted supplementation should continue for ongoing losses and maintenance.
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**Table**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Function</th>
<th>Dose</th>
<th>Route</th>
<th>Frequency</th>
</tr>
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<tbody>
<tr>
<td>Chlorpromazine</td>
<td>Antiemetic/sedative</td>
<td>0.2–0.5 mg/kg</td>
<td>IV, IM, SC</td>
<td>q8h</td>
</tr>
<tr>
<td>Dolasetron</td>
<td>Antiemetic</td>
<td>0.5–1 mg/kg</td>
<td>IV, PO</td>
<td>q24h</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Analgesic</td>
<td>5–10 µg/kg/hr</td>
<td>IV</td>
<td>CRI</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>Analgesic</td>
<td>0.1–0.2 mg/kg</td>
<td>IV, IM, SC</td>
<td>q6–8h</td>
</tr>
<tr>
<td>Ketamine</td>
<td>Analgesic</td>
<td>2–10 µg/kg/min</td>
<td>IV</td>
<td>CRI</td>
</tr>
<tr>
<td>Maropitant</td>
<td>Antiemetic</td>
<td>1 mg/kg</td>
<td>SC, PO</td>
<td>q24h</td>
</tr>
<tr>
<td>Methadone</td>
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<td>Metoclopramide</td>
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<td>0.1–0.4 mg/kg</td>
<td>IM, SC</td>
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<td></td>
<td></td>
<td>1–2 mg/kg/day</td>
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<td>Ondansetron</td>
<td>Antiemetic</td>
<td>0.5–1 mg/kg</td>
<td>PO</td>
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*False negatives and positives occur; the author does not rely on fPLI tests for a definitive diagnosis.

Urinalysis, urine culture, thoracic radiography, coagulation testing, arterial blood gas analysis, and cytologic and clinico-pathologic evaluation of abdominal fluid may be warranted.

- These findings are often nonspecific.
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**Ultrasonogram of the right cranial quadrant of the abdomen in a cat with acute pancreatitis. Note the hypoechoic nature of the pancreas and the hyperechoic surrounding mesenteric tissues. These changes are not always present in feline pancreatitis.**

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**Drugs Commonly Used During Feline Pancreatitis Therapy**

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Antibiotics are rarely warranted. Antibiotic therapy may be indicated for patients that do not respond to initial supportive care or that respond and then deteriorate; attempts should be made to document a source of infection.

Correct placement must be confirmed via radiography.

The author recommends analgesia for abdominal pain to improve comfort, mobility, and ventilatory capacity.

- Opioid analgesics (e.g., fentanyl, methadone, buprenorphine) are useful in cats (see Drugs Commonly Used During Feline Pancreatitis Therapy, previous page).
- Low-dose ketamine and lidocaine infusions, epidural injection, or abdominal lavage may be useful.
- NSAIDs are not recommended in cats with pancreatitis.

Cats may benefit from antiemetic therapy.

- Maropitant, dolasetron, and ondansetron are efficacious.

Patients that still vomit should have GI obstruction ruled out and a nasogastric (NG) tube placed (Figure 3).

- Ongoing vomiting can be a contraindication for NG tube placement.
- The author often uses NG tubes (easy to place, inexpensive) with feline pancreatitis.
- Frequent aspiration of NG tubes limits gastric distention and may improve gastric myotonic contractions, decrease risk for aspiration pneumonia, improve patient comfort and nausea, and allow for enteral nutrition.
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Drugs Associated with Feline Pancreatitis

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<th>Furosemide</th>
<th>Penicillin</th>
<th>Tetracyclines</th>
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<tbody>
<tr>
<td>Amiodarone</td>
<td>Gabapentin</td>
<td>Prazosin</td>
<td>Thiazide diuretics</td>
</tr>
<tr>
<td>Amlodipine</td>
<td>L-asparaginase</td>
<td>Procaimamide</td>
<td>Trimethoprim–sulfamethoxazole</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>Macrolides</td>
<td>Propofol</td>
<td>Vincristine</td>
</tr>
<tr>
<td>Aspirin</td>
<td>Methimazole</td>
<td>Proton pump inhibitors</td>
<td></td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>Metronidazole</td>
<td>Ranitidine</td>
<td></td>
</tr>
<tr>
<td>Doxorubicin</td>
<td>Mirtazapine</td>
<td>Steroids</td>
<td></td>
</tr>
<tr>
<td>Fluoroquinolones</td>
<td>NSAIDs</td>
<td>Tacrolimus</td>
<td></td>
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NG = nasogastric, SIRS = systemic inflammatory response syndrome
Nutritional

- Nutritional supplementation is essential.

- Little evidence supports outdated NPO therapies in pancreatitis patients.
  - This approach may be detrimental, as villous atrophy may occur shortly after discontinuation of oral alimentation.

- NG tubes allow gastric suctioning and liquid diet supplementation.
  - NG tubes are typically well tolerated as a temporary feeding method.
  - Trickle feeding bypasses the cephalic, gastric, and duodenal phases of pancreatic secretion.

- Alternative feeding tubes include nasoesophageal or esophagostomy tubes, which do not allow gastric suctioning, and jejunal or gastrosomy tubes, which require endoscopy or surgery.
  - The author does not routinely use syringe or forced oral nutrition.

- Total parenteral nutrition is limited by requirements for strict aseptic placement and delivery and does not provide direct nutrition to the intestinal tract.

Surgical

- Surgical treatment is rarely pursued unless pancreatic lavage, debridement, drainage, or partial pancreatectomy is indicated (ie, clinical deterioration despite therapy; pancreatic abscessation).

- Although rare, choledochal stenting of extrahepatic biliary obstruction may be required.

**In General**

Relative Cost

- Management varies; treatment and expense often depend on severity of signs.
- Supportive care is the mainstay of therapy; previous recommendations of withholding food and water are no longer recommended.
- Outpatient therapy (eg, SC fluids, antiemetics, pain medications): $100
- Hospitalization and supportive care (eg, IV fluids, analgesics, nutritional therapy): $251–$500
- Intensive medical and surgical intervention (eg, complications with SIRS): $501–$1000

### Cost Key

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$</td>
<td>up to $100</td>
</tr>
<tr>
<td>$</td>
<td>$101–$250</td>
</tr>
<tr>
<td>$$$</td>
<td>$251–$500</td>
</tr>
<tr>
<td>$$$$$</td>
<td>$501–$1000</td>
</tr>
<tr>
<td>$$$$$$</td>
<td>more than $1000</td>
</tr>
</tbody>
</table>

Prognosis

- Most prognoses of acute and chronic feline pancreatitis are favorable.
  - Cases involving severe complications or cost-prohibitive treatments may result in death or humane euthanasia.

**See Aids & Resources, back page, for references & suggested reading.**