Acute Pancreatitis: Do we treat cats the same as dogs?

Professor Caroline Mansfield

College of Veterinary Medicine, Michigan State University E: mansfi77@msu.edu

Acute pancreatitis (AP) in cats remains a diagnostically and therapeutically challenging condition, often presenting with vague clinical signs and necessitating a management approach distinct from that used for dogs.

Pancreatitis in all species initially develops due to premature activation of inert digestive enzymes (zymogens) within the pancreatic tissue. Possible reasons for this premature activation include auto-activation of trypsinogen, activation by thrombin, apical block of zymogen granules (leading to co-localisation with lysosomal proteases), cathepsin B mediated activation and enterokinase refluxing through the pancreatic duct.

Once this activation has occurred, local and systemic inflammation ensures- independent on the degree of trypsin activation but related to ongoing stimulation of the NFκB pathway. The stimulation of this pathway and acinar cell necrosis leads to an influx of neutrophils, release of mediators that increase vascular permeability and creation of a worsening cycle of acinar cell death and systemic inflammation. The extent to which this occurs in each individual animal is highly variable, and not always proportional to the initial degree of pancreatic inflammation present. Hypoperfusion and thrombosis of the pancreatic microcirculation is another factor that can worsen systemic disease and localized pancreatic necrosis.

Clinical Presentation

Acute pancreatitis in cats is frequently idiopathic, although associations with hepatic lipidosis, inflammatory bowel disease, cholangitis, toxoplasmosis, toxicity and trauma have been identified. Unlike dogs, correlation with food intake (i.e. high fat, low protein) is not common. Additionally, where vomiting and abdominal pain are hallmark signs in dogs, cats often present with nonspecific signs such as lethargy, anorexia, and mild dehydration. Hypothermia, rather than fever, may be more prominent in feline AP.

General Management Principles

Management of feline AP is largely supportive, aimed at controlling pain, restoring fluid and electrolyte balance, and providing nutritional support. If identified, removal or treatment of inciting causes should be undertaken.

Fluid Therapy

Intravenous crystalloid fluids (e.g., Lactated Ringer's solution) are typically administered to correct dehydration and maintain perfusion. In cats, the use of isotonic crystalloids should be carefully monitored to prevent volume overload. Regular assessment of body weight, packed cell volume, total solids, and urine output is essential. In people, thoracic ultrasound is often used to monitor for signs of fluid overload, and so frequent T-FAST in cats requiring high rates of fluids may detect fluid overload before pulmonary oedema or pleural effusion develops.

If coagulopathies are present, plasma (fresh frozen) or whole blood may be given. Use outside of this scenario is not indicated currently. Synthetic colloids (e.g., hydroxyethyl starch) are less commonly

used in cats than dogs but may be beneficial if there is severe hypotension despite adequate crystalloid therapy; careful monitoring of central venous pressure is required for these cats.

If colloid and crystalloid therapy fails to maintain blood pressure, then vasopressor support is indicated. Dopamine may also be effective at increasing pancreatic circulation but may also induce vomiting in cats.

Analgesia

Pain management is a cornerstone of AP treatment in any species. Buprenorphine, a partial μ-opioid receptor agonist, is favoured for its safety and ease of administration via the buccal or subcutaneous routes. Full μ-agonists such as methadone or fentanyl (via CRI or transdermal patches) may be necessary for more severe pain.

Non-steroidal anti-inflammatory drugs should be avoided in feline AP due to the risk of gastrointestinal and renal complications, particularly in hypovolemic patients. Adjunctive analgesics such as gabapentin and maropitant (a neurokinin-1 receptor antagonist with antiemetic and potential visceral analgesic properties) may be beneficial.

Antiemetic, prokinetic and gastroprotection therapy

Maropitant and ondansetron are frequently used to manage nausea and vomiting, though vomiting is less prominent than in dogs. In cats, it is more likely that nausea contributes to inappetence, and so absence of vomiting does not mean that anti-emetics should be withheld.

If there is significant gastroparesis or ileus, prokinetic therapy should be initiated. Metoclopramide as a CRI is considered most effective in the hospital setting to stimulate upper GI motility. A theoretical disadvantage of this drug (stimulation of the sphincter of Oddi) has not been supported clinically to date, so there is no clear indication to avoid the drug. Other alternative medications include compounded cisapride or erythromycin.

Famotidine and pantoprazole are sometimes administered, though evidence for a clinical befit in AP from acid suppression is lacking. Their use should be limited to cats with suspected concurrent gastrointestinal ulceration (haematemesis or melena).

Antibiotic Use

Unlike in dogs, where bacterial translocation and secondary infection are rare but possible, the role of antibiotics in feline AP remains controversial. Infected local tissue/abscessation is not considered a likely sequalae in cats. However, concurrent disease may have a bacterial component (e.g. cholecystitis) or there may be ascending bacteria through the common bile/pancreatic duct. Direct involvement of bacteria in the progression of pancreatitis in cats is not proven. Therefore, currently routine antibiotic administration is not recommended unless there is clinical evidence of infection (e.g., sepsis, neutrophilia with left shift, positive culture, or imaging evidence of abscessation). Broad-spectrum antibiotics such as ampicillin or amoxicillin-clavulanate may be appropriate in suspected cases of secondary bacterial involvement.

Nutritional Intervention

Nutritional support is critical in cats with AP and to a higher degree than for dogs, particularly due to cats' propensity for hepatic lipidosis during periods of anorexia. Early enteral nutrition is advocated once vomiting is controlled and the patient is stable. Nasooesophageal (NO), nasogastric (NG), or

oesophagostomy (O) tubes can facilitate feeding. Selection of which method by which to feed is highly dependent on each individual case presentation.

Contrary to traditional dogma of pancreatic "rest," early feeding does not exacerbate disease and may improve outcomes. A highly digestible, moderate-fat diet is typically recommended, and again availability more often determines the food choice. To date, supplementation with glutamate or other nutritional compounds has not been shown to have any clinical benefit.

Management of Co-Morbidities

Feline AP often co-exists with inflammatory liver and/or intestinal disease. In these cases, a comprehensive treatment approach involving hepatoprotectants (e.g., SAMe, ursodiol), cobalamin supplementation, and dietary modification is warranted. Chronic kidney disease, diabetes mellitus, and hepatic lipidosis frequently complicate feline AP, necessitating individualised fluid, insulin, and nutritional strategies. Respiratory complications can occur in cats with AP, but to a lower degree than with dogs or people, and no specific treatment is currently recommended to prevent this.

Emerging Therapies: Panoquell and Beyond

Panoquell-CA1 (fuzapladib sodium), a novel leukocyte function-associated antigen-1 (LFA-1) inhibitor, is approved in Japan and the United States for the treatment of acute pancreatitis in dogs. It exerts its effects by modulating neutrophil migration and inflammation. Although not yet approved for use in cats, its mechanism of action suggests potential cross-species benefit. Preliminary research is needed to assess safety, dosing, and efficacy in cats.

Monitoring and Prognosis

Frequent reassessment of hydration status, electrolyte balance (particularly potassium and calcium), and clinical response is necessary. Serial fPLI measurements can support monitoring, although correlation with clinical improvement may vary.

Prognosis in feline AP is variable and depends on the severity of the episode, response to therapy, and presence of comorbidities. Cats with mild to moderate disease that receive early nutritional and supportive care often recover well, whereas those with multiorgan involvement or delayed intervention may have a guarded prognosis.

Conclusion

Managing acute pancreatitis in cats requires an appreciation of their unique physiological and clinical profile. Tailored analgesic strategies, cautious fluid therapy, and early nutritional support are central pillars of care. While extrapolation from canine protocols provides a foundation, species-specific evidence and approaches remain essential. Emerging therapies such as Panoquell warrant investigation in feline populations. Lastly, addressing concurrent diseases holistically enhances outcomes and supports long-term recovery.

References

- Forman MA et al 2021 ACVIM consensus statement on pancreatitis in cats. *J Vet Intern Med*. 2021;35(6):703-725
- Xenoulis PG, Fracassi F. Feline Comorbidities: Clinical perspective on diabetes mellitus and pancreatitis. *Journal of Feline Medicine and Surgery*. 2022;24(7):651-661. doi:10.1177/1098612X221106355

- Steiner JM et al. Fuzapladib in a randomized controlled multicenter masked study in dogs with presumptive acute onset pancreatitis. *J Vet Intern Med*. 2023;376):2084-2092.