



DIETARY MANAGEMENT OF FELINE PANCREATITIS

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Pancreatitis is the most common condition of the feline exocrine pancreas. Although diseases of the exocrine pancreas have been thought to occur much less commonly in cats than in humans or dogs, a retrospective study revealed significant pancreatic pathologic lesions in 1.3% of 6504 feline necropsy cases compared with 1.7% of canine necropsy examinations. In addition, a recent report involving 47 cats with pancreatitis documented a high incidence (59%) of concurrent fatty change in their livers. The lack of sensitive and specific markers for feline pancreatic diseases, as well as the low index of suspicion for pancreatic disorders in the cat, have contributed to the relatively infrequent antemortal diagnosis of pancreatitis in cats.

ETIOLOGY

The etiology of feline pancreatitis is unknown. Some cases have been associated with severe abdominal trauma, infectious diseases (herpesvirus 1, feline infectious peritonitis, *Toxoplasma gondii*), cholangitis, cholangiohepatitis, and organophosphate or drug intoxication. Chronic pancreatitis has also been associated with pancreatic neoplasia and liver fluke infestation. Regardless of the underlying etiology, intraacinar activation of trypsin is believed to play an essential part in pancreatitis. Once activated, further activation of all zymogens, in particular proelastase and phospholipase occur, amplifying pancreatic damage. The association of feline hepatic lipidosis and pancreatitis has been well documented. Pancreatitis is present in approximately 40% of cats with hepatic lipidosis (Akol et al.) and usually warrants a poorer prognosis when present. It is difficult to predict which disease occurs initially. Speculation is also increasing about the association between feline inflammatory bowel disease and pancreatitis.

DIAGNOSIS

The clinical presentation of cats with pancreatitis is vague and nonspecific. In a retrospective study by Hill and Van Winkle of 40 cats with necropsy-confirmed pancreatitis, clinical signs reported included:

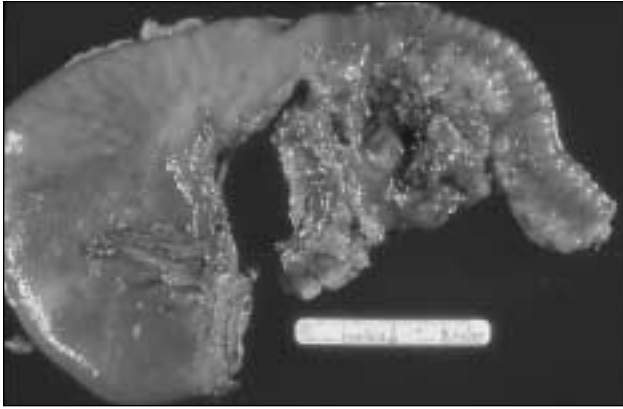


Figure 1. Chronic severe pancreatitis in a cat.

- Lethargy—100% of cases
- Anorexia—97% of cases
- Dehydration—92% of cases
- Hypothermia—68% of cases
- Vomiting—35% of cases
- Abdominal pain—25% of cases
- Palpable abdominal mass—23% of cases
- Dyspnea—20% of cases
- Ataxia—15% of cases
- Diarrhea—15% of cases

In contrast, vomiting and abdominal pain are the most consistent clinical signs in dogs and in humans suffering from pancreatitis.

Hematologic abnormalities are uncommon and nonspecific. Leukocytosis is a relatively common finding in acute pancreatitis. The patient may have a left shift or have toxic white cells if the disease is severe. Other hematologic changes reflect fluid loss and hemoconcentration. **Biochemical abnormalities** include mild elevations in alanine transferase, alkaline phosphatase, and bilirubin and probably reflect secondary hepatic changes. Azotemia is frequently observed secondary to dehydration in most cases.

Radiographs are often subtle and subjective. Decreased contrast in the anterior abdomen, dilated and gas-filled small intestines, and transposition of the duodenum, stomach, and colon are commonly reported. Abdominal **ultrasound** may reveal a hypoechoic pancreas surrounded by hyperechoic mesentery, with or without dilated bile ducts.

The measurement of **serum lipase and amylase** activities is of *low value* in the diagnosis of pancreatitis in cats, with serum concentrations appearing quite variable. Feline **trypsin-like immunoreactivity** (TLI) measures antibodies against circulating trypsin and trypsinogen. TLI is cleared by the kid-

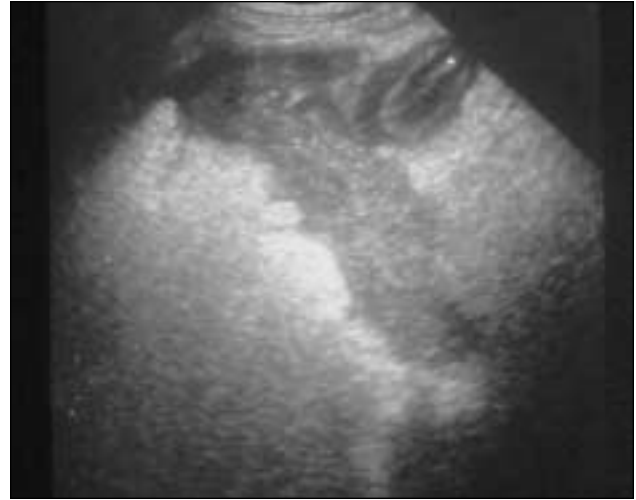


Figure 2. Abdominal ultrasound demonstrating the hypoechoic pancreas surrounded by the hyperechoic mesentery in a cat with severe pancreatitis. A cross-section of the duodenum is visualized adjacent to the pancreas.

ney; therefore elevations can occur with renal dysfunction. TLI values in the normal reference range do not rule out pancreatitis, and abnormally elevated TLI concentrations are not diagnostic for pancreatitis.

The current “gold standard” for diagnosing pancreatitis is pancreatic **biopsy** for histologic evaluation. Peripancreatic fat necrosis is a typical finding in cats with pancreatitis, with variable amounts of acinar cell necrosis and inflammation. Chronic pancreatitis is characterized by interstitial fibrosis with acinar atrophy and lymphocyte infiltrates.

TREATMENT

The clinical picture of pancreatitis in cats differs markedly from that in dogs. Most cats diagnosed with pancreatitis have a more chronic and indolent form of the disease, with vomiting or diarrhea being relatively uncommon presenting complaints. Because of these dissimilarities, therapeutic recommendations for the cat are quite different from those for the dog. Many cats are anorectic, and fasting the cat for an additional 3 to 5 days to “rest” the pancreas will be of little to no clinical benefit. In addition, there is little clinical evidence to support excessive dietary fat restriction in cats with pancreatitis.

At the Veterinary Medical Teaching Hospital at the University of California, Davis, cats with pancreatitis that are anorectic or have lost significant body weight undergo gastrostomy tube placement for enteral feeding. Despite the dogma recommending complete “pancreatic rest” in patients with pancreatitis, we have not observed any clinical deterioration associat-



Figure 3. Necropsy specimen from a cat with severe hepatic lipidosis. The liver is pale, yellow, friable, greasy, and enlarged.



Figure 4. Severe icterus in a cat with pancreatitis causing extrahepatic bile duct obstruction.

ed with feeding these patients intragastrically. Gastrostomy tube placement is avoided if the cat is vomiting intractably or has moderate ascites present. Jejunostomy tube feeding or total parenteral nutrition (TPN) can be used in cats that are vomiting despite the administration of antiemetic therapy. Surgical placement of jejunostomy tubes is preferred over percutaneous endoscopic placement.

Most cats with chronic pancreatitis can be fed a commercial complete and balanced canned diet formulated for maintenance of the animal. It is unnecessary to feed human liquid formulas and liquid veterinary products that frequently contain large amounts of fat and can contribute to delayed gastric emptying. In addition, most human liquid enteral formulas are too low in protein, contain no taurine, and are deficient in arginine.

The foundation of treatment for cats with severe acute necrotizing pancreatitis is similar to that in the dog with acute pancreatitis. These cats have a more acute history of anorexia, vomiting, and weight loss, and many will be icteric due to extrahepatic bile duct obstruction. Maintenance of **fluid and electrolyte balance** is of paramount importance. Most of these cats will not tolerate intragastric feeding, and jejunostomy tube feeding or TPN should be administered. **Antibiotic administration** is controversial but may be used prophylactically, particularly if the patient is febrile or toxic changes are exhibited on the hemogram. Enrofloxacin (5 mg/kg IV q12h) and cefotaxime (25 to 50 mg/kg IV q8h) penetrate well into the pancreas. **Antiemetic therapy** is indicated if the vomiting is persistent or severe. Phenothiazine derived

antiemetics such as chlorpromazine work well, although prokinetic drugs such as metoclopramide as a continuous infusion (1 to 2 mg/kg/24 hours) may also be helpful. Gastric mucosal protection with an **H₂ blocker** is recommended in all patients with acute pancreatitis. Whole blood or plasma transfusion is administered to correct for severe hypoalbuminemia, provide a source of coagulation factors, antithrombin III, and protease inhibitors. Colloid support to enhance pancreatic perfusion can be supplied with hydroxyl starch or high molecular weight dextran.

PROGNOSIS

Survival rates are unknown because antemortem diagnosis has been difficult. However, cats with concurrent acute pancreatitis and hepatic lipidosis have a much poorer prognosis compared to cats with hepatic lipidosis alone. A recent study (Akol et al.) determined the survival rate of cats with concurrent pancreatitis and hepatic lipidosis to be 20%, compared to a survival rate of 50% in cats with hepatic lipidosis alone.

SUGGESTED READINGS

- Akol KG, Washabau RJ, et al: Acute pancreatitis in cats with hepatic lipidosis. *J Vet Intern Med* 7:205–209, 1993.
- Hill RC, Van Winkle TJ: Acute necrotizing pancreatitis and acute suppurative pancreatitis in the cat. A retrospective study of 40 cases (1976-1989). *J Vet Intern Med* 7:25–33, 1993.
- Parent C, Washabau RJ, Williams D, et al: Serum trypsin-like immunoreactivity, amylase and lipase in the diagnosis of feline acute pancreatitis. *J Vet Intern Med* 9:194, 1995 (abstract).
- Simpson K, Shiroma J, Biller D, et al: Ante-mortem diagnosis of pancreatitis in four cats. *J Small Anim Pract* 35:93–99, 1994.
- Steiner JM, Medinger TL, Williams DA: Development and validation of a radioimmunoassay for feline trypsin-like immunoreactivity. *Am J Vet Res* 57:1417–1420, 1996.