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Topical Review

Pancreatitis in Cats

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A B S T R A C T

Pancreatitis was considered a rare disease in the cat until a couple of decades ago when several retrospective studies of severe acute pancreatitis were published. It was apparent that few of the diagnostic tests of value in the dog were helpful in cats. With increasing clinical suspicion, availability of abdominal ultrasonography, and introduction of pancreas-specific blood tests of increasing utility, it is now accepted that acute pancreatitis is probably almost as common in cats as it is in dogs, although the etiology(s) remain more obscure. Pancreatitis in cats often co-exists with inflammatory bowel disease, less commonly with cholangitis, and sometimes with both. Additionally, pancreatitis may trigger hepatic lipidosis, while other diseases, such as diabetes mellitus, may be complicated by pancreatitis. Therapy is similar to that used in dogs, with added emphasis on early nutritional support to prevent hepatic lipidosis. Less is known about chronic pancreatitis than the acute form, but chronic pancreatitis is more common in cats than it is in dogs and may respond positively to treatment with corticosteroids.

Introduction

Pancreatitis in cats is increasingly recognized as being far from rare. This is largely because of increased availability and utilization of abdominal ultrasonography (and growing veterinary expertise) as well as recognition of the limitations of classical diagnostic tests and development of newer, more sensitive and specific tests. Acute and chronic forms of pancreatitis are recognized in cats, and can be very similar to what is described in dogs.[1,2] It is likely that there is much common pathophysiology in the development of the disease in each species. Common nomenclature can be used to histopathologically define the categories of disease (even though that may not be clinically useful). There is an emerging clinical impression, however, that there are also important differences between pancreatitis in the two species. Predisposing causes such as hyperlipidemia and dietary indiscretion or ingestion of a high fat meal/diet that are thought to be important in dogs seem to be unimportant in cats. Chronic disease is suspected to be more common than acute disease in cats. Clinical signs of chronic pancreatitis are often vague and nonspecific such that an index of suspicion for the disease is not readily triggered. Furthermore, pancreatitis in cats commonly occurs concurrently with other disease(s), especially chronic inflammatory small intestinal disease, to a lesser extent hepatic lipidosis and cholangitis, and perhaps disease of other organs too. Pancreatitis may cause cats with diabetes mellitus to become ill and present with ketoacidosis. In cats with multiple diseases, it can be difficult to identify which disorder is most clinically important; all may need attention to treat the patient most effectively. Fortunately, awareness of feline pancreatitis is increasing, and clinical tools to evaluate the pancreas are more accessible than ever. Although much is still not known about feline pancreatitis, there have been major advances over the last few years.[3-6]

Etiology

Several studies have described and categorized the histopathological features of the pancreas in cats clinically ill with pancreatitis.[7-10] The 3 best characterized forms are acute necrotizing pancreatitis, acute suppurative pancreatitis, and chronic nonsuppurative pancreatitis. The chronic form of pancreatitis, defined histologically by mononuclear cell infiltration and variable amounts of fibrosis, is generally thought to be associated with less fulminant clinical signs that extend over a longer time period than is typical for either acute form, often with a waxing and waning course. One study of 63 cats found that history, physical examination, laboratory, or imaging findings failed to distinguish acute necrotizing pancreatitis from chronic pancreatitis.[9] However, this study was based on case identification from necropsy examinations with pancreatitis being suspected ante mortem in only 30% of the cats.

The underlying cause of most cases of feline pancreatitis remains unknown.[4,6] Histologic pancreatitis has been noted in infections with certain parasites, including *Toxoplasma gondii*, pancreatic (*Eurytrema procyonis*), and hepatic (*Amphimerus pseudofelineus*) flukes, and also with some viruses (coronavirus, parvovirus, herpesvirus, and calicivirus), but these are certainly not common causes of feline pancreatitis, and the pancreatitis is usually a minor component of the overall disease picture in affected cats.[11] Tumors of the pancreas may be associated with pancreatitis, perhaps because of local ischemia, duct obstruction, or release of inflammatory cytokines, but again the pancreatitis is usually not a major part of the overall clinical picture. Tighter associations with acute pancreatitis have been described for trauma (road traffic accidents or falling from high buildings), pancreatic ischemia secondary to hypotension or abdominal surgical procedures, and overstimulation of pancreatic secretion secondary to hy-

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percalcemia or intoxication with organophosphate cholinesterase inhibitors (topical fenthion).[4,6] Although other drug-associated pancreatitis has not been reported in cats, a rare idiosyncratic adverse drug reaction should always be considered. It should be noted that early concerns that corticosteroids could cause pancreatitis have now been largely dismissed and steroids are no longer included in lists of drugs suspected of being associated with pancreatitis.[1]

Concurrent disease is identified in the majority of cats with pancreatitis. One study identified concurrent pathology in 92% of the 63 cats, including all of the cats with chronic pancreatitis.[9] Common comorbidities reported were hepatic lipidosis, diabetes mellitus, and lymphocytic cholangitis. This study may have over-represented the frequency of comorbidities, however, as cats were identified from necropsy records, and pancreatitis was the primary clinical concern in a minority of cases.

Several studies have reported an association between pancreatitis in cats and chronic idiopathic inflammatory disease of the small intestine (lymphocytic/plasmacytic enteritis) and biliary tract (cholangitis).[9,12-14] Sometimes, inflammation occurs in each of these three locations and the condition is referred to as "triaditis," although it is the authors' impression that hepatic involvement is far less common than concurrent involvement of the other 2 sites. In cats with cholangitis, however, a high proportion also has intestinal and pancreatic abnormalities. In 1 study, 50% of cats with cholangitis had concurrent pancreatic inflammation[12] and, in a second study, 65% of cats with cholangitis had histologic evidence of pancreatitis (acute or subacute in 4 of 34 cats, chronic-active in 6, chronic in 7, and unspecified in 5; pancreatic necrosis was present in 5 cats).[13] Interestingly, in the latter study, there was no difference in the biochemical variables (total bilirubin, alanine aminotransferase [ALT], alkaline phosphatase [ALP], aspartate aminotransferase, and gamma-glutamyl transpeptidase) and white blood cell count between cholangitis in cats with and without pancreatitis. Some reports suggest a bacterial pathogenesis in cats with cholangitis,[15] but the rate of positive bile cultures is low, even in untreated cats. Applying culture-independent methods to look for bacteria in tissue has provided further support for the role of enteric bacteria in pathogenesis. Using a florescence in situ hybridization assay, Twedt et al[16] and Simpson et al[17] observed intrahepatic bacteria in 33% of cats with cholangitis, and the highest bacterial numbers were associated with neutrophilic cholangitis rather than the lymphocytic form. Further clinical investigation of these associations with pancreatitis in cats is warranted, but awareness of these potential comorbidities should prompt their consideration in all feline patients with suspected or proven pancreatitis.

Acute pancreatitis is thought to begin as a sterile process and reports of positive microbiological cultures or bacterial complications, such as pancreatic abscessation, are very uncommon. As with cholangitis, recent studies in cats using culture-independent methods to evaluate tissue for the presence of bacteria suggest that greater consideration may need to be given to bacterial infection in feline pancreatitis.[17] Using florescence in situ hybridization, bacteria were identified in 35% (11 of 31) of pancreatic biopsy samples from cats with moderate to severe pancreatitis (especially acute necrotizing and acute suppurative pancreatitis) and in only 1 normal control cat. The common channel theory, whereby the pancreatic ducts and bile ducts join as a common duct before entering the duodenum, may partially explain the relationship of cholangitis and chronic pancreatitis, whereby bacterial infections in the biliary system could involve the pancreatic ducts as well.

Clinical Presentation

No significant age, breed, or sex predisposition has been recognized in cats with pancreatitis and no relationship has been established with body condition score. A wide age range (5 weeks to 20 years) has been reported, but most authors agree that cats older than 7 years are most commonly affected. Clinical signs associated with both acute and chronic pancreatitis in cats are notoriously nonspecific. Anorexia and lethargy are the primary reasons for presentation of cats with acute pancreatitis. "Textbook" signs of acute pancreatitis seen in dogs are usually absent in cats. For example, vomiting occurs in less than half of affected cats, abdominal pain is less often appreciated than in dogs, and hypothermia is reported more commonly than fever. Also in contrast to dogs, an anterior abdominal mass (usually comprising the pancreas and inflamed peripancreatic fat) can sometimes be palpated. This can be easily misdiagnosed as another intra-abdominal structure such as a lesion of the intestinal tract or mesenteric lymph nodes, and palpation of it may not elicit a response of apparent pain. Diarrhea, dehydration, icterus, pale mucous membranes, and dyspnea have also been reported. Some cases of acute pancreatitis are associated with severe clinical syndromes including shock, disseminated intravascular coagulation, and multi-organ failure. Progressive insidious weight loss, perhaps associated with decreased appetite, is more common in cats with chronic disease, although it may not be clear if this reflects pancreatitis per se or concurrent disease, as mentioned above. Finally, in patients with diabetes mellitus or those endstage patients developing exocrine pancreatic insufficiency, there may be a history of episodes of polyuria, polydipsia, or polyphagia.

Diagnosis

Results of routine tests (complete blood counts, serum biochemical profiles, and urinalysis) are highly variable in feline pancreatitis, and generally not contributory to the diagnosis. Leukocytosis, leukopenia, hemoconcentration, and anemia have all been reported. Although less common than leukocytosis, leukopenia is associated with a worse prognosis.[7,18,19] Increased serum activities of liver enzymes (ALT, ALP, aminotransferase, and gamma-glutamyl transpeptidase) and increased total bilirubin are commonly reported, and sometimes reflect concurrent lipidosis or cholangitis. In 1 study, serum liver enzyme activities were significantly higher in cats with chronic pancreatitis than in cats with acute necrotizing pancreatitis, probably because of the higher prevalence of concurrent hepatobiliary disease in cats with chronic pancreatitis.[9]

Somewhat surprisingly, the most commonly reported biochemical abnormality in cats with pancreatitis is hypercholesterolemia, with 72% of cases exhibiting this abnormality.[7,9,18-21] Because there are few clinically encountered rules for hypercholesterolemia in cats, finding this abnormality in a cat with vague signs of illness may prompt further evaluation of the pancreas. Hypocalcemia reportedly occurs more commonly in cats with pancreatitis (45%-65%),[7,9,18,21] than in dogs (3%-5%). Hypocalcemia was reported with equal frequency in cats with acute and chronic pancreatitis[9] and reductions in ionized or total calcium were a poor prognostic finding.[21] Other abnormalities encountered include pre-renal (or less commonly renal) azotemia, hypoalbuminemia, hyperglycemia, hypoglycemia, and hypokalemia.

Despite an early experimental study of feline pancreatitis that showed transient 2 to 6-fold increases in serum lipase activity (but not that of amylase), traditional assays for serum amylase and lipase activities have been reported to be of no value in the diagnosis of spontaneous feline acute pancreatitis because activities of both enzymes are frequently normal in cats with spontaneous pancreatitis.[22,23] Increased amylase and lipase activities are both associated with chronic malabsorption in cats with chronic intestinal disease, and both have been noted to increase when the glomerular filtration rate is reduced. Therefore, neither serum lipase nor amylase activity is of clinical value in the diagnosis of pancreatitis in cats.

More recently, specific immunoassays for feline trypsin-like im-

munoreactivity (fTLI) and feline pancreatic lipase (fPL) have been developed.[18,19,23-28] Although the former was established to diagnose exocrine pancreatic insufficiency in cats, serum values do increase in cats with pancreatitis, although these increases are short-lived and so the test lacks sensitivity for diagnostic purposes. Nonetheless, in the absence of azotemia, increased serum fTLI values are highly suggestive of pancreatitis. Like fTLI, fPL is made only by pancreatic acinar cells, and immunoassays for fPL do not cross react with lipases of nonpancreatic origin. Unlike fTLI, fPL is not increased in patients with pre-renal azotemia,[28] and increases in pancreatitis seem to be sustained for several days, even in mild or moderate transient disease (Fig. 1).

Controversy exists as to the sensitivity and specificity of the newer tests for the diagnosis of pancreatitis. Part of this confusion results from the lack of agreement regarding a gold standard against which diagnostic methods should be evaluated; gross and histologic evaluation of the pancreas as well as clinical signs, and a clinical diagnosis of pancreatitis have all been used, and each has its pros and cons. Some cases of pancreatitis are not apparent on gross inspection of the pancreas, and histologic changes can be patchy and biopsy samples may not be representative. Conversely, histologic abnormalities may not be associated with clinical signs. Furthermore, concurrent disease in the small intestine or liver may be responsible for some clinical signs and not pancreatic pathology, per se. It seems unlikely that there will be general agreement on a gold standard in the short term, and so when numbers for test sensitivity and specificity are quoted they should be carefully interpreted in the context of the above comments. It is accepted, however, that fPL is pancreas-specific in its origin, and is a large molecule (almost as large as albumin) that is not cleared from the blood by renal filtration, and therefore serum fPL is unaffected by azotemia.[28] Increases in serum fPL reflect increased release from the exocrine pancreas. Serum fPL is certainly a more sensitive test for pancreatitis than either fTLI or abdominal ultrasonography. Pancreatitis can be subclinical (or be associated with few apparent clinical signs) and serum fPL can be elevated in these cases. It is also possible that increases in fPL occur secondary to pancreatic pathologies other than classical "pancreatitis" (such as benign nodular hyperplasia or cystic-appearing lesions), but this has not been documented, to the authors' knowledge.

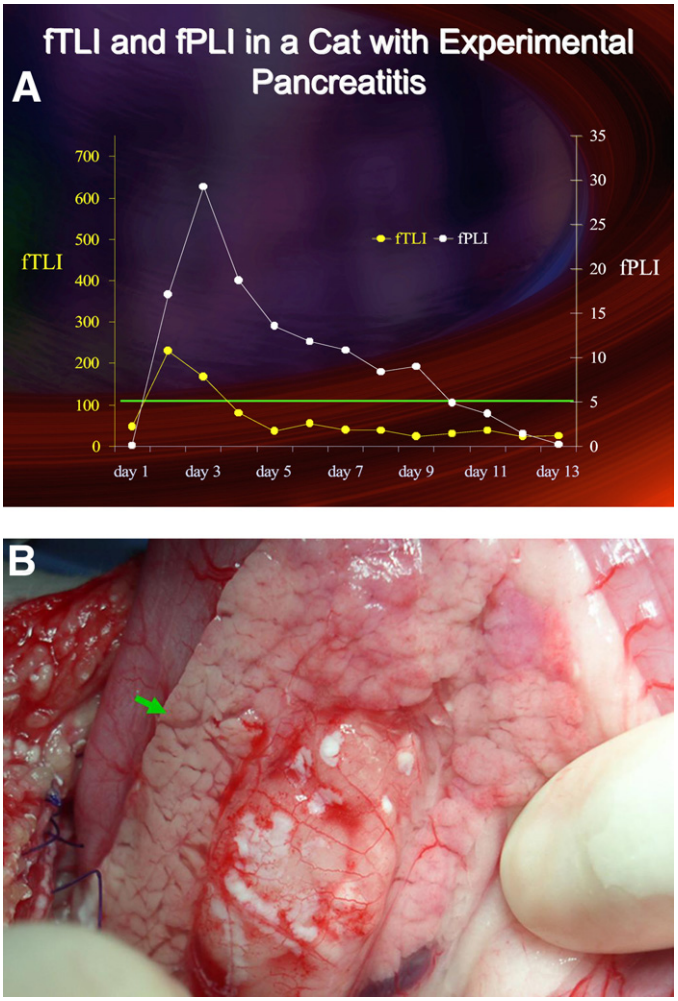

Fig. 1. Serum concentrations (μg/L) of feline trypsin-like immunoreactivity (fTLI) and feline pancreatic lipase (fPLI) **A,** in a cat with mild transient acute pancreatitis **B,** induced experimentally by injection of oleic acid into the pancreatic duct. The horizontal line indicates the upper limit of the reference range for each enzyme. Although both enzymes are pancreas-specific in origin, the increase in serum fPLI is sustained for several days longer than that of serum fTLI. Edema is evident in the fissures between lobules of the pancreas (arrow) and areas of fat necrosis and hemorrhage can be seen in the adjacent peri-pancreatic tissue (Courtesy of M. Zavros).

Despite these limitations and considerations, current evidence indicates that fPL is the most accurate blood test for diagnosing pancreatitis in cats. In a published study, the overall sensitivity (ability to detect pancreatitis) of the fTLI test was 67% (100% in cats with moderate to severe pancreatitis and 54% in cats with mild pancreatitis).[25] The specificity (ability to rule out pancreatitis) of the fTLI test was 100% in healthy cats and 67% in symptomatic cats with histologically normal pancreata, with an overall specificity of 91%. Preliminary results from a follow-up study evaluating the Spec fPL test in clinically well-characterized healthy and sick cats support the diagnostic utility of this test. In 182 cats enrolled in a prospective clinical study, both sensitivity and specificity were about 80% using a Spec cutoff of 5.4 μg/L.[26]

Although serum fPL is almost certainly a highly specific marker for pancreatic acinar cell abnormalities, and is a more sensitive test for pancreatitis than either fTLI or abdominal ultrasonography, it is very important that a positive test result not be considered an endpoint to the diagnostic evaluation. Cats often have concurrent diseases along with pancreatitis, and liver enzymes should be evaluated in the context of possible cholangitis or lipidosis. Consideration should also be given to evaluation for small intestinal disease (even if there are no suggestive signs such as vomiting or diarrhea) by assay of serum cobalamin and folate, because results of these tests are frequently abnormal in cats with increased fPL. Finally, some cases of pancreatitis, or at least of increased serum fPL, may arise secondarily to other problems, for example, severe dehydration or hypercalcemia. Attention should be given to diagnosis of other potential underlying causes for the pancreatic abnormalities such as high intestinal obstruction due to foreign bodies or tumors. In cats, in particular, evidence of pancreatitis should always be considered in the potential context of a wider disease picture.

Imaging

Abdominal radiographs in cats with pancreatitis may show a loss of detail in the cranial abdomen, and, in some cases, there may be a suggestion of a mass in the cranial abdomen. Exclusion of some of the other causes of vague gastrointestinal signs, however, is still a major diagnostic rationale for survey abdominal radiographs in cats with the clinical signs described above. Although radiography rarely provides specific evidence of the presence of pancreatitis, sonographic evaluation can be very useful, particularly in the diagnosis of severe acute disease and in evaluation of sequelae of pancreatitis and potential concurrent disease. Paired with the fPL test, abdominal ultrasonography is a key diagnostic test in cats with suspected pancreatitis. The ultrasonographic appearance of normal feline pancreas is well described (Fig. 2).[8,29-32] Findings in cats with pancreatitis may include an

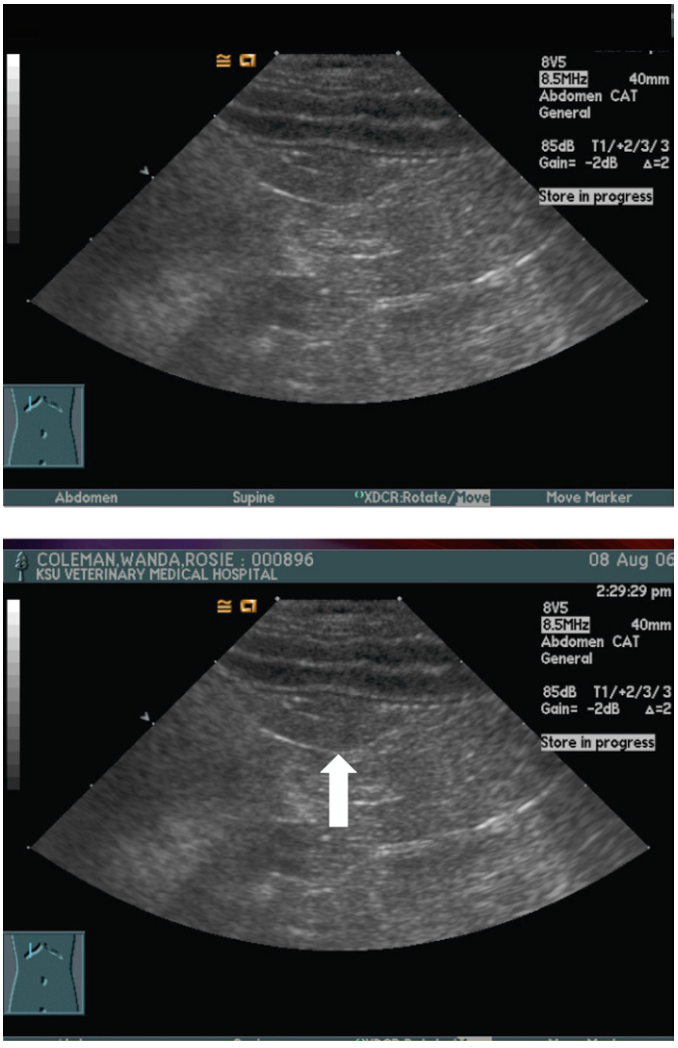

Fig. 2. Ultrasonographic image of a normal feline pancreas (arrow).

enlarged (thickened) pancreas, hypoechoic (pancreatic necrosis) or hyperechoic (fibrosis) parenchyma surrounded by a hyperechoic mesentery (peripancreatic fat necrosis; Fig. 3), peripancreatic fluid accumulation cystic-appearing lesions, a mass effect in the cranial abdomen, and biliary duct dilation. In some cases, however, there will be no detectable abnormalities. Studies have shown abdominal ultrasound has a sensitivity of 24% to 67% for detecting pancreatitis, meaning that 33% to 76% of cats with pancreatitis had no sonographic changes detected in the pancreas or that the pancreas could not be visualized. Abnormal sonographic findings, however, are highly specific for pancreatitis, meaning that a cat with compatible clinical signs and sonographic changes in the pancreas is very likely to be correctly diagnosed with pancreatitis. The low reported sensitivities of ultrasonography probably partly reflect its limited usefulness for detection of chronic disease (Fig. 4), which in many low-grade cases can only be detected by histologic examination of pancreatic biopsies. Conversely, benign hyperplastic nodules and neoplastic disease may be clearly apparent as pancreatic ultrasonographic abnormalities, but they cannot be differentiated from pancreatitis in many instances. Pancreatic enlargement because of hypoalbuminemia or portal hypertension has been described in dogs. Although unlikely to cause clinical confusion with pancreatitis, pancreatic enlargement is not sufficient as a sole finding to diagnose pancreatitis.[33] Sonographic findings should always be interpreted in light of the full clinical picture, and normal findings certainly do not rule out pancreatitis.

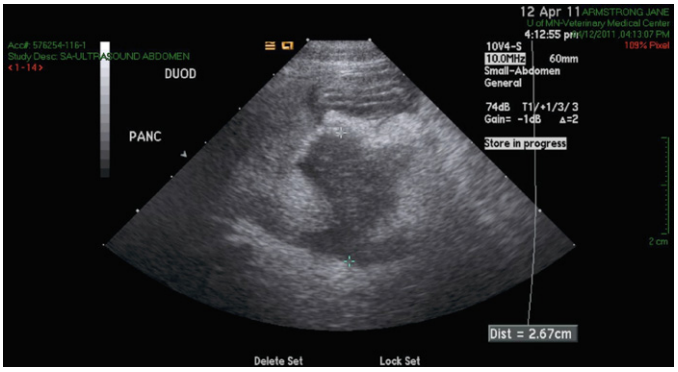

Fig. 3. Ultrasonographic features of feline acute pancreatitis. The pancreas is diffusely and severely thickened and hypoechoic with an irregular margin; surrounding omentum is markedly hyperechoic.

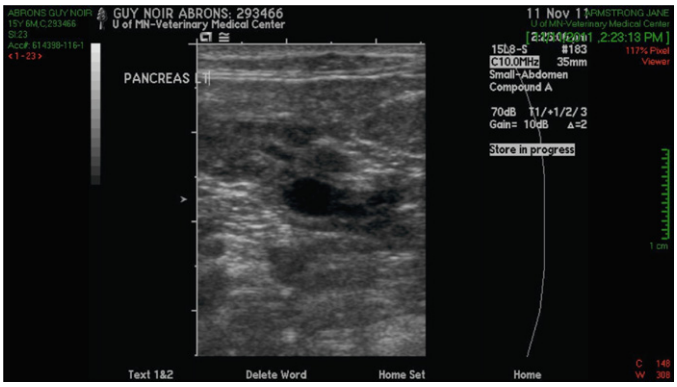

Fig. 4. Ultrasonographic appearance of the pancreas in a 16-year-old cat diagnosed with chronic pancreatitis. There are numerous hypoechoic nodules of varying size throughout the pancreas. The margin of the pancreas is irregular secondary to the nodules. Fine-needle aspiration cytology results were consistent with hyperplastic nodules. At 9-month follow up, the cat remains clinically well on 2.5 mg prednisolone daily.

Endosonography has been evaluated to a limited extent in cats. With this technique, an endoscope with an ultrasound transducer at its tip is positioned in the lumen of the stomach. In 11 healthy cats, general visualization of the pancreas was superior with endosonography compared to transabdominal ultrasound. In 6 cats diagnosed with pancreatitis based on clinical signs and increased fPLI, endosonography provided better resolution of pancreatic margins and parenchyma, but did not alter the diagnosis.[34]

Use of ultrasound-guided fine-needle aspiration cytology of the pancreas is used routinely in the investigation of pancreatic masses and cystic-appearing masses. Increasingly, ultrasound-guided fine-needle aspirates of the pancreas and peripancreatic tissue and/or fluid are being used to assist in making the diagnosis of pancreatitis as well as evaluation of other pancreatic abnormalities[35] (Fig. 5A, B). Bacteriologic examination of material obtained by ultrasound-guided fine-needle aspiration of pancreatic lesions may also be helpful in some cats, especially in establishing that cystic-appearing lesions are sterile on aerobic and anaerobic cultures.

Biopsy

Pancreatic biopsy is rarely clinically indicated as a primary procedure and pancreatitis patients are often poor anesthetic risks. If laparotomy (or laparoscopy) is performed for other reasons such as to relieve common bile duct obstruction, gross observation fol-

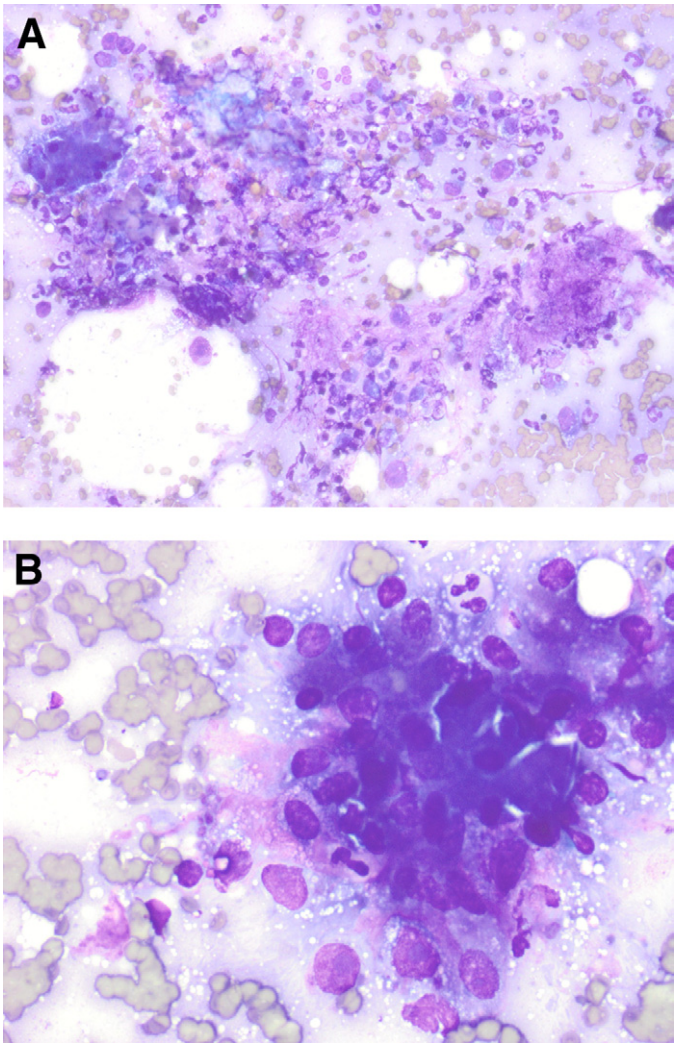

Fig. 5. A, Pancreatic cytology from a cat with necropsy confirmed pancreatitis. The bright pink extracellular material in the lower right corner is mineral, which is commonly observed. There is a cluster of pancreatic acinar cells in the upper left corner of the smear. The remainder of the smear consists of blood, lipid, and increased numbers of neutrophils and foamy macrophages. Wright Giemsa Stain, ×200. Photomicrograph courtesy of Dr. L. Sharkey, University of Minnesota. **B,** Pancreatic cytology from the same case of necropsy confirmed pancreatitis. There is a cluster of hyperplastic exocrine pancreatic acinar cells exhibiting mild disorganization, diminished cytoplasmic granularity with cytoplasmic vacuolization, and increased nuclear to cytoplasmic ratio that often characterize reactive hyperplasia in this cell population. The bright pink extracellular material may represent matrix material or amyloid, both of which were observed in histologic sections. Wright Giemsa Stain, ×500. Photomicrograph courtesy of Dr. L. Sharkey, University of Minnesota.

lowed by pancreatic biopsy may be performed. Previous concerns regarding handling of the pancreas and pancreatic biopsy inducing pancreatitis seem to be unfounded and pancreatic biopsy is routinely performed during celiotomy. Inspection and biopsy of the small intestine and liver (with possible gallbladder aspiration) is also important because of the high rate of concurrent disease in cats. When pancreatic biopsy results are evaluated, it must be kept in mind that a single biopsy may be insufficient to exclude pancreatitis, as the distribution of inflammation within the pancreas may be patchy. The clinical significance of histologic characterization of low-grade inflammation remains to be clarified. Lymphocytic inflammation, in particular, can be found in cats with no history suggestive of classical pancreatic disease; this may reflect true subclinical disease or it may be that the signs of chronic pancreatitis are not appreciated in these cases or are attributed to other concurrent diseases as outlined above.

Therapy of Acute Pancreatitis

The initial medical management of cats with acute pancreatitis must not be delayed until a diagnosis is confirmed. In experimental studies, one of the major factors in the progression of mild pancreatitis to severe pancreatitis is disturbed pancreatic microcirculation. Fluid resuscitation with lactated Ringer's solution (or 0.9% saline) is recommended to correct fluid losses in the third space and maintain an adequate intravascular volume.[36] Attentive monitoring is required, especially in cats, however, to prevent accidental over-hydration. Potassium supplementation (20-30 mEq/L KCl to start) is necessary to replace losses in diarrhea, vomitus, and urine, and to supplement the lack of food intake. Supplementation should be based on serial measurement of serum potassium levels. Symptomatic hypocalcemia (tremors, seizure activity) is a possible complication of acute pancreatitis and requires that calcium gluconate be given at doses of 50 to 150 mg/kg intravenously over 12 to 24 hours and serum ionized calcium concentrations monitored during therapy. Insulin therapy is initiated in patients with diabetes. Colloids such as dextrans are useful in hypoproteinemic cases and may have antithrombotic effects that help maintain the microcirculation. An experimental study in dogs with induced acute pancreatitis showed that dogs resuscitated with lactated Ringer's solution alone required approximately 5 L more fluid to maintain systemic pressures than dogs resuscitated with crystalloids and colloids.[37] Plasma transfusion has potential additional benefits due to its albumin, protease inhibitor, and coagulation factor content, but there are no prospective studies evaluating its usefulness in cats with naturally occurring pancreatitis. Although plasma is expensive, often impractical, and not currently widely used in the therapy of acute pancreatitis in cats, it should be kept in mind that cats with significant hypoalbuminemia should probably be treated with either a synthetic colloid or plasma and that this may help minimize pancreatic edema and promote pancreatic perfusion.[1]

Analgesia is a very important aspect of the treatment of pancreatitis. It can be provided using opioids such as buprenorphine (0.005-0.01 mg/kg subcutaneously or sublingual q6-8 hours) or butorphanol (0.2-0.4 mg/kg subcutaneously q6 hours). A 25 μg/h transdermal fentanyl patch (Fig. 6) is a good way of providing longer duration analgesia (up to 72 hours). Adequate fentanyl blood levels are attained 3 to

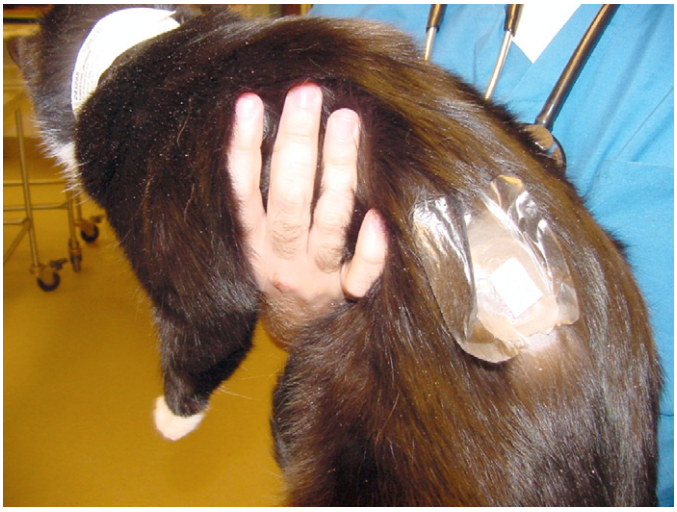

Fig. 6. A 25 μg/h fentanyl patch placed on a cat to provide analgesia transdermally.

12 hours after patch placement in cats, so another analgesic should be administered for the first 12 hours post-placement.

Vomiting and apparent nausea may be severe in cats with pancreatitis. The potent antiemetic, maropitant (Cerenia), an NK_1-receptor antagonist, administered at 1 mg/kg orally, subcutaneously (refrigerate before administering), or intravenously (off-label use) once a day is very useful in controlling vomiting and may assist with control of apparent nausea.[38] Increasing evidence suggests maropitant also helps provide visceral analgesia, making it doubly useful in pancreatitis.[39] Alternative antiemetics are one of the 5-HT_3 antagonists (ondansetron 0.1-1.0 mg/kg or dolasetron 0.5-1.0 mg/kg, orally or intravenously every 12-24 hours). The dopaminergic antagonist, metoclopramide, may be useful to enhance motility in the upper gastrointestinal tract. Its action as an antiemetic is questionable as cats are reported to have few dopamine receptors in the chemoreceptor trigger zone. Additionally, experimental feline studies of acute pancreatitis showed benefit to dopamine infusion, providing theoretic rationale for avoiding use of a dopamine receptor antagonist.[40] Dopamine has no effect on pancreatic blood flow in normal cats. In pancreatitis, dopamine transiently reduced blood flow, but it returned to normal within 1 hour. It was concluded that dopamine ameliorated pancreatitis by reducing pancreatic ductal and/or microvascular permeability rather than by altering pancreatic blood flow.[41]

Gastric acid suppression is commonly incorporated into the therapy of pancreatitis in cats, despite a lack of evidence supporting its use in this species. The rationale includes protecting the esophagus from exposure to gastric acid during episodes of vomiting, and protecting against gastric ulceration to which patients with pancreatitis may be predisposed due to hypovolemia and local peritonitis. Additionally, there may other benefits. A higher gastric pH may decrease exocrine pancreatic stimulation but remains undocumented as a treatment for pancreatitis. In human patients with pancreatitis, nasogastric suctioning has failed to show any benefit in reducing pain or shortening hospitalization time. If gastric acid suppression is required, it is theoretically preferable to select a proton pump inhibitor (PPI) than an H_2-receptor antagonist, although the onset of action may be slower. In dogs, PPIs have been shown to be more effective at reducing gastric acidity. Oxidative stress with free radicals is known to play a crucial role in acute pancreatitis in several species. Pantoprazole, although primarily used as a PPI, possesses reactivity toward hydroxyl radicals. An experimental study in rats showed that pantoprazole reduced inflammatory changes and leakage of pancreatic acinar cells in severe acute pancreatitis.[42] The anti-inflammatory properties of pantoprazole are mediated by reduced expression of inflammatory and adhesive proteins with a consecutive decrease in platelet and leukocyte activation. Rats treated with pantoprazole had reduced tissue infiltration of inflammatory cells and less acinar cell necrosis.

In patients suspected of having acute pancreatitis, oral intake has historically been withheld for the initial 24 to 48 hours (or longer) and then gradually re-introduced as tolerated. The theory behind holding patients NPO was to suppress the function of the exocrine pancreas. This rationale has come under close scrutiny in human and veterinary medicine and is no longer the standard of care. Bowel rest is associated with intestinal mucosal atrophy and increased infectious complications due to bacterial translocation from the gut.[43] Currently, antiemetics are used immediately and as required to get vomiting under control, and nasoesophageal feeding (Fig. 7) by slow infusion is begun as soon as possible (or gradual voluntary oral alimentation, if possible). This approach attempts to maintain enterocyte integrity and reduce the risk of bacterial translocation. Recent studies in human patients indicate that enteral nutrition (by nasogastric or nasojejunal feeding), can attenuate the systemic inflammatory response and may decrease complications.[43] From these studies, it is clear that enteral feeding should be provided, but the number of calories and type of nutrient mixtures have yet to be established in cats (and other species). Clinical experience suggests a low fat food does not seem to be necessary. The authors routinely use the liquid diet CliniCare (Abbott Animal Health, Abbott Laboratories, Abbott park, IL, USA) which provides 1.0 kcal/mL with a caloric distribution of 45% fat and 35% protein, and flows easily through a 5 or 8Fr nasoesophageal feeding tube. As the appetite returns, small amounts of food can be offered frequently. The size of the meals should be slowly increased and the frequency of feeding decreased if vomiting does not recur.

Fig. 7. A nasoesophageal tube in place to provide enteral support by frequent small bolus or infusion feeding. A Chinese finger trap knot is usually placed at the lateral edge of the nostril in addition to a simple interrupted suture near the lateral canthus of the eye or on the cheek to secure the tube.

Recent studies in cats using culture-independent methods suggest that greater consideration may need to be given to bacterial infection in cases of feline pancreatitis.[24] Broad-spectrum antibiotics (e.g., amoxicillin/clavulinic acid, marbofloxacin, or ampicillin with marbofloxacin or enrofloxacin) may be warranted in cats with shock, fever, marked leukocytosis, or other evidence of breakdown of the gastrointestinal barrier (see case example).

Based on experimental evidence and experience in human patients, some veterinary centers are using hyperbaric oxygen treatments in patients with severe pancreatitis. No outcome data are available at present.

Surgery is occasionally needed to remove devitalized or infected pancreatic or peri-pancreatic tissue. Surgery may also sometimes be required to relieve an obstruction of the common bile duct related to cholangitis or biliary sludge. Resection or surgical drainage of pancreatic pseudocysts is not always necessary as these can resolve spontaneously or following ultrasound-guided percutaneous drainage.

Therapy of Chronic Pancreatitis

Cats with chronic or chronic relapsing pancreatitis often have a decreased appetite and lose weight despite control of intercurrent diseases. In addition to therapies discussed above to help control vomiting, nausea, and apparent pain, appetite stimulation can play a useful role in maintaining body condition. Mirtazapine is a useful choice as it seems to have appetite-stimulating, antinausea, and antiemetic properties.[44] The pharmacokinetics of mirtazapine have been studied in healthy cats and in cats with chronic kidney disease.[44,45] A dose of 1.88 mg/cat (one fourth of a 7.5 mg tablet) orally q48 hours,

found to be appropriate in the cats with kidney disease, is usually used in cats with chronic pancreatitis and the interval may often be further extended to every 72 hours. A second choice of appetite stimulant is cyproheptadine (1-2 mg/cat q12-24 hours orally).

Cobalamin (B_{12}) deficiency may occur in cats with pancreatitis and concurrent gastrointestinal disease. Hypocobalaminemia can impair nutrient absorption and is occasionally severe enough to produce neuromuscular signs. It is recommended that serum cobalamin be evaluated in cats with chronic pancreatitis; measurement also affords an opportunity to determine serum fTLI concentration and ensure that exocrine insufficiency has not developed as the result of pancreatic parenchymal destruction. The preferred route for cobalamin supplementation is parenteral (250 μg/injection q7 days subcutaneously for 6 weeks, then 1 dose after 30 days, then re-evaluate a serum cobalamin 30 days later). Cobalamin may also have a pharmacologic effect as an appetite stimulant.

Historical reluctance to use corticosteroids in patients with pancreatitis came from the presumption that corticosteroids could lead to pancreatitis, as noted above under "Etiology." Corticosteroids exert a broad anti-inflammatory effect and, in addition, a specific role for corticosteroids in enhancing apoptosis and increasing production of pancreatitis-associated protein, which confers a protective effect against inflammation, has also been proposed. Certainly beneficial effects are seen when cats with concurrent inflammatory bowel disease and chronic pancreatitis are treated with prednisolone, although whether the pancreatic or intestinal disease (or both) are responding positively is not known. Anti-inflammatory doses of prednisolone are increasingly being used in cats with signs of chronic (or chronic relapsing) pancreatitis, anecdotally with good results. Clinical trials are needed to assess the effectiveness of this therapy.

Corticosteroids are now under evaluation in human patients with acute pancreatitis, especially patients diagnosed with immune-mediated pancreatitis. An additional rationale for the use of corticosteroids in patients with acute pancreatitis is relative adrenal insufficiency (now termed critical illness-related corticosteroid insufficiency), a relative glucocorticoid insufficiency along with tissue resistance to the effects of steroids due to a prolonged and severe proinflammatory state. For these reasons, steroid use in cats with acute pancreatitis is increasingly being considered, especially in cases failing to respond to other therapies.

In human medicine, there is compelling experimental and clinical evidence for the important role oxidative stress plays in the pathophysiology of chronic pancreatitis. Increased oxidative stress has been implicated as a potential mechanism in the etiopathogenesis of chronic pancreatitis. A number of studies have demonstrated that patients with chronic pancreatitis have compromised antioxidant status, which may be a contributing factor to the enhanced oxidative state associated with the disease.[46] Supplementation with antioxidants leads to reduction in oxidative stress and can be associated with a reduction in abdominal pain.[47] In most studies, administration of a mixture of antioxidants, such as methionine (which is converted to S-adenosylmethionine [SAMe]), selenium, β-carotene, vitamin C, and vitamin E, was necessary to relieve pain, even if monotherapy improved antioxidant status. Such results may warrant studies on the role of oxidant stress and antioxidant therapy in chronic pancreatitis in cats. An antioxidant commonly used in cats is SAMe, an important hepatocellular metabolite and glutathione donor with hepatic and systemic antioxidant effects. In healthy cats, orally administered SAMe increases plasma SAMe and liver glutathione.[48] The commonly used dosage of SAMe is 35 to 60 mg/kg PO q24 hours. Medical grade SAMe (Denosyl, Nutramax Laboratories, Inc., Edgewood, MD, USA) is recommended. SAMe must be given as an intact tablet on an empty stomach (ideally 1 hour before feeding) for optimal absorption. Based on data from human patients with pancreatitis, consideration may need to be given to using a combination of antioxidants in cats, such as SAMe, vitamin C, vitamin E, and/or selenium.

Human patients with chronic pancreatitis, 25% of which are idiopathic cases, experience chronic pain. Aside from analgesics, enzyme replacement is an important part of treatment. In addition to improving digestion and lessening the effects of exocrine pancreatic insufficiency, pancreatic enzymes may play a role in pain relief. Caution must be used in extrapolating these results to cats, and many cats do not eat well when pancreatic enzymes are added to their food.

In spite of advances in diagnostic capabilities, especially ultrasonography and fPL, the diagnosis of feline pancreatitis remains challenging in many cases. There is currently no single specific test for pancreatitis and diagnosis is based on a combination of compatible clinical, clinicopathological, and imaging findings. In prospective studies, clinical and laboratory findings need to be correlated with simultaneously obtained ante mortem pancreatic biopsies. Therapy for acute pancreatitis is currently supportive with fluid and colloidal support, antiemetics, analgesics, and enteral nutritional support (as soon as vomiting is controlled) providing the mainstays of therapy. Therapy for chronic pancreatitis may include some of the above therapies, often with the addition of anti-inflammatory doses of corticosteroids, antioxidants, and an appetite stimulant. Prospective trials on the therapy of acute and chronic pancreatitis are sorely needed. Once the diagnostic and therapeutic hurdles are surmounted, further work on risk factors will guide prevention strategies.

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Case Example: Acute Pancreatitis

An 8-year-old male neutered domestic shorthaired cat was examined with a 2-day history of lethargy, anorexia, and anorexia. Subcutaneous fluids and enrofloxacin were started on day 1 of illness. The cat had been diagnosed with diabetes mellitus approximately 2 years previously; twice-daily insulin glargine administration was monitored at home. Blood glucose values over the past 2 days had ranged from 300 to 350 mg/dL, but dropped to 40 mg/dL immediately before his emergency presentation. His last dose of insulin was 6 units 24 hours before presentation. Medical history included 2 previous episodes (1 year and 3 months before) of lethargy, anorexia, and vomiting, from which he recovered with symptomatic therapy. On physical examination, the cat was quiet but alert and responsive, with a temperature of 103.3 F, heart rate 180/minute, respiratory rate of 56/minute, and a body condition score of 9/9 (body weight 9.4 kg). Dehydration was estimated at 7% to 8%. The abdomen was tense on attempted palpation and was suspected to be painful. Results from a complete blood count, biochemistry profile, urinalysis, serum fructosamine and feline pancreatic lipase (fPL) were:

Hct 40% (29-47)
Total plasma protein 9.8% (5.7-7.5)
White blood cell count 5730/μL (1830-16270)
Platelets: Clumped (110.000-413,000)

- Segmented neutrophils 690/μL (1200-13200)
- Band neutrophils 3780/μL (0-160)
- Metamyelocytes 230/μL (0)
- Lymphocytes 400/μL (200-9400)
- Monocytes 460/μL (0-800)
- Eosinophils 170/μL (0-1900)
- 3+ toxic change
- 2+ Dohle bodies
- Nucleated red blood cells 1/100 WBC
- Slight lipemia
- Cr 1.1 mg/dL (0.5-2.1)
- ALT 48 U/L ($<$ 42)
- ALP 17 U/L ($<$ 88)
- Na 146 mmol/L (147-158)
- Cl 107 mmol/L (113-123)
- K 4.6 mmol/L (3.9-5.3)
- Bicarb 17.7 mmol/L (12-20)
- P 2.8 mg/dL (3.3-7.8)
- Cholesterol 396 mg/dL (56-226)
- Glucose 356 mg/dL (74-143)
- Bili 0.2 mg/dL (0-0.3)
- Urinalysis (cystocentesis): specific gravity 1.067 with 2+ glucose, neg ketones, 2+ protein, 1+ bilirubin, inactive sediment, too numerous to count fat/hpf
- fPL 27 μg/L ($\geq$ 5.4 is consistent with pancreatitis)

Fructosamine 426 μmol/L (190-365)

Sonographic evaluation of the abdomen revealed both limbs and the body of the pancreas to be diffusely severely thickened and hypoechoic with irregular margins; the surrounding omentum was markedly hyperechoic. The liver was mildly hyoechoic; the gall bladder was normal. The duodenal wall appeared thickened but layering was maintained. The mesenteric lymph nodes were enlarged. The spleen was mildly enlarged and echogenically coarse. Scant anechoic abdominal fluid was present.

Cytologic evaluation of fine-needle aspirates of the liver and spleen failed to reveal any significant pathology. A diagnosis of acute pancreatitis with possible sepsis was made. Treatments included:

- Fluid therapy
- Buprenorphine
- Regular insulin subcutaneously for 24 hours before restarting glargine
- Maropitant
- Famotidine
- Nasoesophageal tube (CliniCare continuous infusion fed at calculated resting energy requirement)
- Enrofloxacin and ampicillin

The cat was hospitalized for 3 days in the intensive care ward, during which time his temperature reached 105.7 F. The white blood cell count on the second day of hospitalization was much improved, with a total count of 11,870/μL (9850 segmented neutrophils and 710 band neutrophils). When his interest in voluntary food consumption began to return, he was discharged on 4 units glargine BID plus antibiotics for a total of 10 days. Further recovery was uneventful.