Chronic Pancreatitis in Dogs and Cats

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ABSTRACT: Pancreatitis is the most common disorder of the exocrine pancreas in dogs and cats. Clinical diagnosis of chronic pancreatitis is challenging because the disease is usually mild or subclinical and because its clinical signs are often the same as those of complicating or concurrent diseases. Obtaining a detailed history, performing a thorough physical examination, and conducting tests that are sensitive and specific for pancreatitis are crucial in diagnosing chronic pancreatitis. Initial management of an acute episode of chronic pancreatitis largely involves supportive and dietary measures, while long-term management of chronic pancreatitis is based on dietary modification. Management of complications and concurrent diseases is crucial in animals with chronic pancreatitis.

Pancreatitis is the most common disorder of the exocrine pancreas in dogs and cats.[1–4] The acute form of the disease is believed to be much more common in dogs, whereas chronic pancreatitis is thought to be the primary form in cats.[1–3,5,6] Although chronic pancreatitis has generally been considered less clinically significant than acute disease, several complications and concurrent diseases can affect the outcome and long-term prognosis of patients with chronic pancreatitis.[2,7–9] Clinical diagnosis of chronic pancreatitis can be challenging because the disease is often subclinical or is associated with mild, nonspecific clinical signs and blood work abnormalities. Consequently, most cases of canine and feline chronic pancreatitis likely remain undiagnosed.[7,10,11]

CLASSIFICATION AND DEFINITION

A standardized classification system for pancreatitis does not exist in veterinary medicine. In contrast, several international symposia have been conducted and a universally accepted classification system for pancreatitis has been established in human medicine. Although disagreements regarding the validity and usefulness of this system exist, the human classifications may be
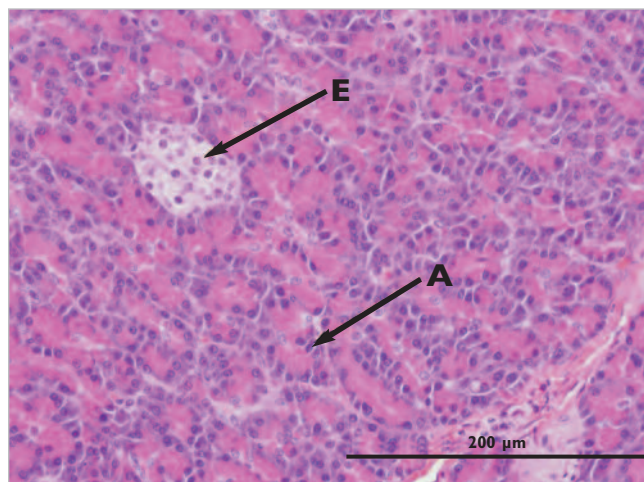
Figure 1. Histologic view of a normal dog pancreas. Most of the cells are exocrine pancreatic cells (A) arranged in acini that subsequently form the pancreatic lobules. Islets of Langerhans are composed of clusters of endocrine cells (E), which have smaller nuclei and a more vacuolated cytoplasm. Hematoxylin and eosin stain; magnification 40×. (Reprinted with permission from Steiner JM. Diseases of the exocrine pancreas. In: *Small Animal Gastroenterology*. Hannover, Germany: Schluetersche; 2008; image courtesy of Dr. Shelley J. Newman, University of Tennessee)
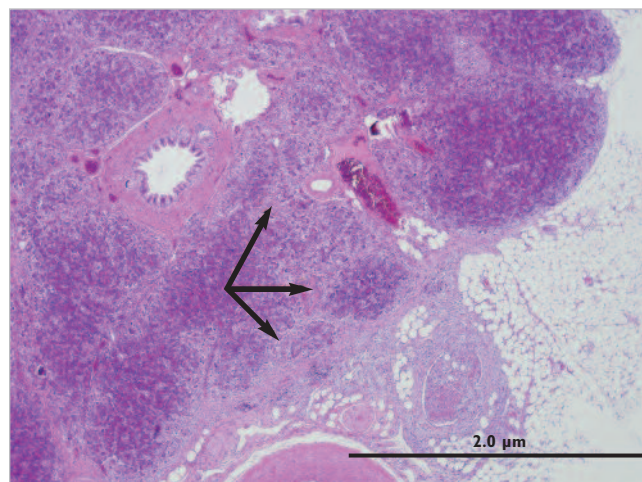

Figure 2. Histologic view of the pancreas from a dog with chronic pancreatitis. Extensive areas of fibrosis and lymphocytic infiltration are noted (some indicated by *arrows*). Hematoxylin and eosin stain; magnification 10×. (Courtesy of Dr. Newman)
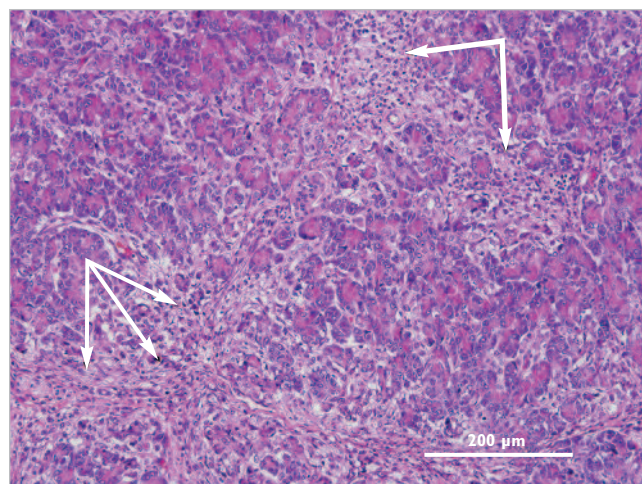

Figure 3. Close-up view of the pancreas in Figure 2. Note the marked fibrosis and lymphocytic infiltration of the pancreas (*arrows*). Hematoxylin and eosin stain; magnification 40×. (Courtesy of Dr. Newman)

adapted for veterinary use. Following the human system, canine and feline pancreatitis can be divided into acute and chronic forms based on the presence (chronic form) or absence (acute form) of permanent histopathologic changes such as fibrosis and acinar atrophy[5,12–15] (Figures 1, 2, and 3). Other histopathologic findings (e.g., pancreatic cell necrosis, peripancreatic fat necrosis, predominant type of cell infiltration) and clinical criteria are often used to further classify the disease process in dogs and cats.[16–19] Chronic pancreatitis can be defined as a continuous, usually progressive inflammation of the pancreas characterized by permanent damage of the pancreatic structure that can lead to irreversible impairment of pancreatic exocrine and endocrine function.[3,5,12–15] Clinically, both acute and chronic pancreatitis can be mild or severe.[3,5,13,15]

Some animals show histopathologic evidence of fibrosis and concurrent pancreatic cell necrosis and should be classified as having chronic pancreatitis because of the presence of fibrosis, the key histologic evidence of chronicity.[5,6,11,15,20,21] However, some authors in the veterinary field[16–19,22] consider pancreatic cell necrosis to be the histologic lesion that defines acute pancreatitis, even when mild or moderate fibrosis is present. Especially in cats, the term *chronic active pancreatitis* has been used to describe the simultaneous presence of fibrosis and pan-

creatic cell necrosis.[6] Also, the predominant inflammatory cellular infiltrate (neutrophils or lymphocytes) is often used to describe pancreatitis as suppurative (neutrophils) or lymphocytic (lymphocytes), and suppurative inflammation is considered a component of acute disease while lymphocytic infiltration is considered a component of chronic disease.[16,17] This confusion regarding classification and terminology underlines the importance of determining a multidisciplinary classification

system that applies to dogs and cats and is universally accepted. Until such a consensus has been established, we prefer to apply the human classification system of acute and chronic pancreatitis according to the absence or presence, respectively, of permanent histopathologic changes such as fibrosis and acinar atrophy.

PREVALENCE

Although pancreatitis has been established as a clinically significant disease in cats, its exact prevalence is unknown.[3] Early necropsy studies reported a relatively high prevalence (0.6% to 2.4%) of feline pancreatitis,[1,2] but clinical data indicate that most cases of pancreatitis in cats remain undiagnosed.[2,3,7] In three studies, chronic pancreatitis accounted for 89%,[2] 66%,[6] and 65%[11] of all feline pancreatitis cases. In a recent study, histopathologic examination of 115 feline pancreata from healthy and sick cats revealed findings consistent with pancreatitis in 67% of cases, including 45% of apparently healthy cats.[23] In this study, chronic pancreatitis (including chronic active pancreatitis) was far more common than acute pancreatitis, accounting for almost 90% of all pancreatitis cases.[23] The fact that mild pancreatitis was identified in almost half of the apparently healthy cats raises concerns about the clinical significance of histopathologic findings of pancreatitis in cats, especially mild findings.[23]

Chronic pancreatitis has traditionally been thought to be much less common than acute pancreatitis in dogs.[15] However, because chronic pancreatitis might be mild and subclinical, its true prevalence and clinical importance in dogs are unknown. A recent study examined 73 pancreata from dogs presented for postmortem examination; about 64% showed histologic evidence of inflammation.[15] Surprisingly, the most common lesions detected were lymphocytic infiltration (72%), fibrosis (60%), and a combination of both lesions (47%), findings suggestive of chronic disease.[15] These results indicate that chronic pancreatitis may be more common in dogs than is currently believed. However, the clinical significance of these findings remains to be clarified. Some breeds (e.g., Yorkshire terriers, miniature schnau-

zers) appear to be at increased risk for pancreatitis.[5,24–26] Although studies that detail the histopathologic evaluation of the pancreas in these cases are lacking, it is our clinical impression that many, if not most, cases of pancreatitis in miniature schnauzers are chronic.

ETIOLOGY

Little is known about the etiology of pancreatitis in dogs and cats, and most cases are considered idiopathic because an underlying initiating factor cannot be determined.[3,5,17] It is unclear whether chronic pancreatitis results from recurrent attacks of acute pancreatitis or whether it is a distinct disease. However, it is possible that some animals develop chronic disease after one or more episodes of acute pancreatitis.[5,14] Factors that have been associated with the development of pancreatitis in dogs and cats are summarized in the box on page 170.

Several causes of or risk factors for acute pancreatitis, such as obesity, high-fat diets, pharmaceutical agents, and infectious agents, might also lead to chronic pancreatitis.[24,26] Recent studies suggest that potassium bromide therapy may lead to clinical or subclinical pancreatitis in dogs.[27,28] Several infectious agents have been shown (*Toxoplasma gondii*,[29] *Amphimerus pseudofelineus*[30]) or suspected (feline parvovirus, FIP virus, feline herpesvirus[3]) to be associated with feline pancreatitis, although none has been reported as an important cause of chronic pancreatitis in recent clinical case series.[6,16,20,21,31,32] Recently, pancreatitis has been related to infection with a highly virulent strain of feline calicivirus.[33]

Several studies have shown an association between chronic pancreatitis and inflammatory diseases of the liver and intestine in feline patients.[6,31,34] The term *triaditis* has been used to describe the coexistence of these three disorders. In one study, pancreatitis was reported in 50% of cats with cholangitis; 39% of these cats also had inflammatory bowel disease (IBD).[34] In another study, cholangitis was present in 64% of cats with histologically confirmed pancreatitis.[6] It is unclear which disease occurs first and what role the initial disease plays in the pathogenesis of the other disorders. Although some dogs with chronic pancreatitis also have evidence of

> *The true prevalence of chronic pancreatitis is unknown, but recent evidence suggests that it is more common than clinically diagnosed.*

Factors Potentially Associated with the Development of Pancreatitis in Dogs and Cats

Nutritional (only in dogs)
• High-fat diets/dietary indiscretion
• Obesity

Drugs
• Organophosphates (cats)
• L-Asparaginase
• Azathioprine
• Potassium bromide (dogs)
• Antimonial derivates (dogs)
• Many others

Infectious agents
• *Toxoplasma gondii*
• *Amphimerus pseudofelineus*
• Feline calicivirus (virulent strain)
• FIP virus?
• Feline parvovirus?
• Feline herpesvirus?
• Other

Endocrinopathies (only in dogs)
• Hyperadrenocorticism
• Hypothyroidism
• Diabetes mellitus

Breed (hereditary predisposition)
• Miniature schnauzers
• Terriers
• Domestic short-haired cats
• Siamese

Other factors
• Hyperlipidemia (dogs)
• Bile reflux (cats with concurrent cholangiohepatitis?)
• Reflux of duodenal contents (cats with concurrent inflammatory bowel disease?)
• Duct obstruction
• Hypercalcemia
• Ischemia
• Trauma
• Pancreatic tumors

IBD, the pathogenetic connection between these two conditions is even less clear. Tumors of the pancreas (most commonly pancreatic adenocarcinoma) and pancreatic ductal obstruction (e.g., by intra- or extrapancreatic neoplasia, other masses, or hepatic flukes) may also lead to pancreatitis in cats and dogs.[3,35,36]

Endocrinopathies such as hypothyroidism, hyperadrenocorticism, and diabetes mellitus may also be associated with increased risk for pancreatitis in dogs, but a clear cause-and-effect relationship has not been described.[24,26,37] Several types of hyperlipidemia have been associated with frequent episodes of pancreatitis in humans, and such a relationship has been suggested in dogs but not in cats.[38–40] Secondary hyperlipidemia may, therefore, be responsible for the increased risk for canine pancreatitis associated with hypothyroidism, hyperadrenocorticism, and diabetes mellitus.[26,37] Hyperlipidemia has also been observed in obese dogs, providing a possible explanation for why obese dogs are more prone to pancreatitis.[41] In human patients, hypertriglyceridemia can also be the result (rather than the cause) of pancreatitis, and the same relationship has been suggested in dogs.[5,42] However, in one study in which acute pancreatitis was experimentally induced in dogs, there was no evidence of hypertriglyceridemia 96 hours after the induction of pancreatitis.[43]

As stated above, miniature schnauzers may be at increased risk for chronic pancreatitis. A possible hereditary mechanism has been suggested for this breed predisposition.[5,44] In humans, hereditary pancreatitis is an important condition that can be caused by mutations of several genes (e.g., cationic trypsinogen gene, cystic fibrosis transmembrane regulator gene, pancreatic secretory trypsin inhibitor [PSTI] gene).[45] Initial studies in miniature schnauzers with pancreatitis failed to identify any mutations of the cationic or anionic trypsinogen genes.[44,46] Because idiopathic (primary) hypertriglyceridemia is common in miniature schnauzers in the United States,[47] and because hyperlipidemia has been associated with pancreatitis in humans,[42] another study investigated the association between pancreatitis, hyperlipidemia, and potential mutations of the lipoprotein lipase gene (the gene encoding the major enzyme involved in triglyceride clearance) in miniature schnauzers.[25] However, this study failed to identify any mutations of the lipoprotein lipase gene.[25] Recently, preliminary studies have identified a number of mutations of the PSTI gene in miniature schnauzers.[48] The clinical significance of these mutations and their association with pancreatitis are still being investigated.[48]

DIAGNOSIS

Overall, careful evaluation of the animal's history, physical examination, and routine clinical pathology findings as well as the results of highly specific and sensitive tests (pancreatic lipase immunoreactivity [PLI], ultrasonography, histopathology) is crucial to correctly diagnose pancreatitis. In chronic cases, clinical and clinicopathologic evidence of pancreatitis is often absent

during remissions, and serial diagnostic efforts may be necessary before a diagnosis can be made.

Signalment

Cats of any age, breed, or sex can develop pancreatitis. Older cats appear to be more likely to develop chronic disease.[2,20,23] Domestic short-haired and Siamese cats have been reported to be at increased risk.[16,17,20]

Most dogs that present with pancreatitis are older than 5 years.[4,24,26] Miniature schnauzers and Yorkshire terriers may be at increased risk for pancreatitis.[24,26] However, a diagnosis of pancreatitis has not always been definitely confirmed in published reports; thus, more conclusive data are needed to confirm or refute these breed predilections. No clear sex predisposition has been identified. Many dogs are obese or overweight.[5,24,26]

Clinical Features

Acute and chronic pancreatitis cannot be differentiated clinically in dogs and cats.[3,13,16] Most cases of chronic pancreatitis are considered to be mild, and many remain sub-

in dogs and cats.[5] Other clinical signs may be consequences of concurrent diseases (e.g., polyuria/polydipsia in animals with diabetes mellitus; diarrhea, weight loss, polyphagia, and poor haircoat in animals with exocrine pancreatic insufficiency [EPI]).[8,9,19]

Routine Clinical Pathology

A complete blood count (CBC), serum biochemistry profile, and urinalysis should always be conducted in dogs and cats suspected of having chronic pancreatitis.[10] Although the results of these tests are nonspecific and cannot confirm or rule out pancreatitis, they are extremely useful for the diagnosis and exclusion of other diseases and give important information about the general condition of the animal.[10] These tests also help identify specific abnormalities (e.g., electrolyte disturbances) that must be corrected as part of patient management.[6,10] CBC, biochemistry profile, and urinalysis results may be normal in some cases.

Hematologic findings in cats with chronic pancreatitis may include mild anemia or hemoconcentration and

> *Chronic pancreatitis is often subclinical or characterized by mild nonspecific or intermittent clinical signs.*

clinical.[3,5] Clinical signs are nonspecific and usually transient.[3] However, signs of exacerbation can mimic those of acute pancreatitis.[4,13] In a study of 33 cats with histologically confirmed chronic pancreatitis, the reported clinical signs included complete or partial anorexia (70%), lethargy (52%), vomiting (39%), and weight loss (21%).[16] Diarrhea is reported less frequently.[2,16,21] The most common physical examination findings include dehydration (51%), pallor (30%), and icterus (24%).[16] Tachypnea or dyspnea, hypothermia or fever, tachycardia, signs of abdominal pain, and a palpable abdominal mass may also be noted.[2,3,6,7,16,17]

Clinical signs in dogs with chronic pancreatitis are not well documented but may include anorexia, weakness, vomiting, diarrhea, and abdominal pain.[4,5,14] The combination of vomiting and cranial abdominal pain is considered especially suggestive of pancreatitis in dogs.[10,19,26] Dehydration, abdominal pain, icterus, and fever may be noted on physical examination.[4,14,19,26]

Severe systemic complications (e.g., cardiovascular shock, disseminated intravascular coagulation, multiorgan failure) are unlikely in cases of chronic pancreatitis

leukocytosis or leukopenia.[6,16,20,31] Elevations in liver enzyme activities and hyperbilirubinemia are common and often reflect concurrent hepatic inflammatory disease or hepatic lipidosis.[2,3,6,10,16,20] Azotemia may be present and can be the result of dehydration caused by vomiting or diarrhea (prerenal azotemia) or, less commonly, concurrent nephritis (renal azotemia).[2,6,10,16,20,34] Other possible findings include hypoalbuminemia, hypercholesterolemia, and hyperglycemia.[2,6,10,16,20,49] The latter can be transient or permanent; permanent hyperglycemia reflects concurrent diabetes mellitus.[6,8] Urinalysis can be used to distinguish prerenal from renal azotemia and concurrent diabetes mellitus from transient, stress-induced hyperglycemia. Electrolyte abnormalities (particularly hypokalemia) and hypocalcemia are common in severe cases.[6,16,17] A recent study reported cobalamin deficiency in cats with gastrointestinal disease, and five of the 22 cobalamin-deficient cats examined had histologically confirmed pancreatic inflammation.[49] However, laboratory findings similar to all those described above have been described for cats with a histologically normal pancreas, indicating that these find-

ings may be the result of concurrent diseases rather than pancreatitis.[31]

CBC, biochemistry, and urinalysis abnormalities in dogs with chronic pancreatitis are not well documented but are probably similar to those reported in cats.[5,14] Hyperlipidemia, which might be either the cause or the result of pancreatitis in dogs,[5,14] is common in dogs with concurrent endocrinopathies, such as hyperadrenocorticism, hypothyroidism, or diabetes mellitus.[24,26] Primary hyperlipidemia in conjunction with pancreatitis is more common in certain breeds (i.e., miniature schnauzers).[47]

Imaging
Radiography

Although conclusive diagnosis or exclusion of pancreatitis is not possible based on radiography alone, radiography remains a logical initial approach to patients suspected of having pancreatitis because it is relatively inexpensive and is useful for the diagnosis or exclusion of other diseases that may cause similar signs. Abdominal radiography lacks specificity and sensitivity in identifying pancreatitis in dogs and cats.[5,10,16–20] In one study of 14 cats with chronic pancreatitis, the most common radiographic finding was loss of peritoneal detail in the cranial abdomen, which was reported in 50% of cases.[16] Other possible findings include increased opacity, presence of a cranial abdominal mass, displacement

diagnosing pancreatitis, especially when conducted by an experienced ultrasonographer.[21,31] If stringent criteria are applied, the specificity of abdominal ultrasonography for pancreatitis in dogs and cats is high, but advances in technology have increased the resolution dramatically, and it is possible to overinterpret findings.[10] Ultrasonographic findings in cats with chronic pancreatitis can include abdominal effusion, hypoechogenicity of the pancreas, hyperechoic peripancreatic mesentery (indicating peripancreatic fat necrosis), pancreatic and biliary duct dilatation, and enlargement, calcification, and cavitation of the pancreas.[5,10,16,31,50] Occasionally, the pancreas is hyperechoic, potentially indicating the presence of pancreatic fibrosis.[9,10] However, a lack of hyperechogenicity in the pancreas does not rule out the presence of pancreatic fibrosis. Ultrasonography is also helpful in detecting concurrent disease. Furthermore, ultrasound-guided fine-needle aspiration is a useful tool for the distinction and management of pancreatic complications of pancreatitis (e.g., pseudocysts).[50]

As in cats, abdominal ultrasonography has been reported to be highly specific for canine pancreatitis if stringent criteria are used, with a relatively high sensitivity of about 70%.[10,19] However, use of abdominal ultrasonography in dogs has been described only in patients with fatal acute pancreatitis, in which lesions are usually pronounced.[19] The sensitivity of abdominal ultrasound

> *Studies suggest that serum pancreatic lipase immunoreactivity is more sensitive and specific than any other test currently available for the diagnosis of pancreatitis in dogs and cats.*

of abdominal organs (stomach, duodenum), dilatation of the small intestine (with fluid or gas), and hepatomegaly.[10,16,17,20,22] The sensitivity of abdominal radiography was very low (24%) in one study in dogs with fatal acute pancreatitis, and similar results would be expected for dogs with chronic disease.[19]

Ultrasonography

The reported sensitivity of abdominal ultrasonography for the diagnosis of feline pancreatitis ranges from 11% to 67%.[20,22,31] Many cases of feline pancreatitis cannot be diagnosed based on ultrasonographic examination alone, and a diagnosis of pancreatitis cannot be excluded based on a normal ultrasonographic examination.[16,22,31] However, ultrasonography can be effective in

for the diagnosis of chronic pancreatitis in dogs is unknown. Ultrasonographic findings in dogs with pancreatitis are similar to those described in cats.[5,19]

Computed Tomography

Computed tomography conducted in cats with histologically confirmed pancreatitis has shown disappointing results and cannot be recommended.[10,20,21] The use of computed tomography for the diagnosis of pancreatitis has not been evaluated in any large number of dogs.

Pancreatic Enzymes
Amylase and Lipase

Amylase and lipase activities have long been considered markers of pancreatic inflammation; therefore,

these enzymes are often the first tested in animals with suspected pancreatitis. However, these enzymes originate from many tissues, and the traditional catalytic assays cannot differentiate them according to their tissue of origin, making their specificity for pancreatitis fairly low.[10,51,52] In dogs, it has been suggested that only amylase and lipase activities in excess of three times the upper limit of the reference range should be considered suggestive of pancreatitis.[5,10] However, such elevations can result from extrapancreatic disorders (e.g., renal failure, hepatic disease, neoplastic disorders).[5,52,53] Moreover, the sensitivity of these assays for acute canine pancreatitis is not ideal, and their sensi-

of PLI assays has limited the usefulness of TLI in diagnosing pancreatitis in dogs and cats.[10,20,21]

Pancreatic Lipase Immunoreactivity

The PLI assay is a species-specific immunoassay. Pancreatic lipase, which is synthesized in acinar cells, enters the circulation in large quantities during pancreatitis. In contrast to the traditional catalytic assays for lipase, which indiscriminately measure the activity of lipases of any origin (e.g., pancreatic, gastric, duodenal), PLI is specific for pancreatic lipase and, thus, for pancreatic disease. Canine and feline PLI assays are available.[54,55] Studies in cats with spontaneous and experimental pan-

> *The coexistence of exocrine pancreatic insufficiency and diabetes mellitus in some animals is highly suggestive of chronic pancreatitis as the underlying cause.*

tivity for chronic canine pancreatitis is likely even lower.[10,17,19,51,52] Normal amylase and/or lipase activities cannot rule out chronic or acute pancreatitis because many dogs with pancreatitis may have normal activities of these enzymes.[10,19,51,52] In any case, more specific and sensitive tests should be considered. These two tests are of no clinical value in cats and should not be used for the diagnosis of chronic or acute feline pancreatitis.[7,10,17]

Trypsin-like Immunoreactivity

The trypsin-like immunoreactivity (TLI) assay is a species-specific immunoassay that measures circulating trypsinogen and, when present, trypsin. The specificity of feline TLI (fTLI) for pancreatitis has been questioned because high TLI concentrations have been reported in cats with gastrointestinal disorders (usually IBD or gastrointestinal lymphoma) and no demonstrable pancreatic disease.[31,49] Also, possibly because of its short half-life, fTLI has a low sensitivity (28% to 40%) for pancreatitis.[5,11,20,21,31] In one study involving 18 cats with histopathologic evidence of chronic (active) pancreatitis, the sensitivity of fTLI was 28%.[21] Nevertheless, some studies have found serum fTLI to be more sensitive than abdominal ultrasonography for the diagnosis of pancreatitis in cats.[20,31,32]

As in cats, the sensitivity and specificity of serum canine TLI (cTLI) for the diagnosis of pancreatitis are less than optimal (sensitivity: 36%).[10,51] The availability

creatitis have shown promising results.[21,56] In one of these studies, in which most cats had evidence of chronic pancreatitis, PLI appeared to be more sensitive and specific than serum fTLI concentration; it had the same sensitivity as, but higher specificity than, abdominal ultrasonography.[21]

Preliminary investigations suggest that canine PLI (cPLI) is more sensitive and specific than any other test available for the diagnosis of pancreatitis in dogs.[57–61] The reported sensitivity of cPLI for the diagnosis of canine pancreatitis is more than 80%, which is higher than that reported for abdominal ultrasonography (68%), amylase (62% to 69%), lipase (39% to 73%), and cTLI (36%).[19,51,57,61] However, the sensitivity of the cPLI assay has not been evaluated in dogs with histopathologically confirmed chronic pancreatitis. Experimentally induced chronic renal failure and prednisone administration were not found to affect cPLI concentration.[58,59] A commercial assay for the measurement of serum cPLI concentration (Spec cPL, IDEXX Laboratories) was released in 2005. This assay shows the same clinical performance as the original cPLI assay and can be used interchangeably with the original cPLI assay. A more recently released point-of-care test for the estimation of cPLI (SNAP cPL; IDEXX Laboratories) can be used early in the diagnostic workup of dogs suspected of having pancreatitis to include or exclude pancreatitis from the differential diagnosis. A positive test result should be followed by laboratory measurement of serum cPLI concentration.

Figure 4. Gross appearance of the pancreas from a dog with chronic pancreatitis. Note the irregular appearance indicative of pancreatic fibrosis. (Courtesy of Dr. Newman)

Histopathology

At present, a definitive diagnosis of pancreatitis can be made only by histopathologic examination of the pancreas.[10] Histopathology is also the only diagnostic method by which acute and chronic pancreatitis can be differentiated. However, it is the most invasive and least commonly conducted test for pancreatitis, and exclusion of pancreatitis may still be challenging. Although gross lesions (Figure 4) may confirm the diagnosis of pancreatitis in some cases (and these lesions usually suggest preferred sites for biopsy), they appear to be very uncom-

pancreas should be evaluated in cats with IBD or cholangiohepatitis.

TREATMENT

Although the etiology of most chronic pancreatitis cases is never determined, all potential etiologic factors should be investigated and, if possible, properly managed.[5]

Fluid Therapy

Many animals with pancreatitis present with some degree of dehydration because of ongoing vomiting or diarrhea.[5] Aggressive intravenous fluid therapy is the most important priority in severely dehydrated animals, in which maintenance of tissue and pancreatic perfusion is crucial.[5] Mildly dehydrated animals respond well to subcutaneous fluid therapy. The most commonly used fluids are lactated Ringer's solution and 0.9% saline. Hypokalemia is often present as a result of potassium loss from a combination of diarrhea, vomiting, fluid therapy, and food withholding or anorexia.[5] Therefore, serum potassium should always be measured, and potassium must be added to the intravenous fluids when necessary.[5] Other electrolyte and acid–base balance disturbances should also be identified and corrected.[5]

Analgesic Therapy

Analgesic therapy is very important in the management of animals with pancreatitis and is indicated in all animals with an acute episode of pancreatitis, regardless

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Histopathology of the pancreas is the only way to confirm chronic pancreatitis, but standardized criteria for sampling and histologic interpretation are lacking.

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mon in dogs[15] and are not always apparent in cats.[22] In one study, macroscopic lesions were seen in only 8.5% of 47 dogs with histologic evidence of pancreatic inflammation.[15] Also, inflammatory lesions of the pancreas are often highly localized, and multiple sections of the pancreas must be evaluated to increase the likelihood of finding microscopic lesions.[15,17,22] Therefore, histopathology must be evaluated very carefully when histopathologic evidence of pancreatitis is absent, especially when only one section of the pancreas has been examined.[10,15] Because triaditis appears to be a common problem in cats, hepatic and intestinal biopsies should be considered in cats suspected of having pancreatitis. Likewise, the

of clinical evidence of pain.[3] Initial pain management can be accomplished by opioid administration[5,62] (Table 1). Injectable opioids, such as morphine, meperidine, or buprenorphine, are effective and provide fast results.[5,62] After initial analgesia has been achieved with injectable opioids, transdermal fentanyl patches can be used to maintain patient comfort. These patches are practical and safe and provide a longer duration of analgesia compared with other treatments[62]; however, they take longer to achieve their effect and, therefore, should not be used for initial pain management. Long-term pain management is essential in animals with chronic pancreatitis that experience chronic pain, and fentanyl

Table 1. Drugs Commonly Used to Treat Chronic Pancreatitis in Dogs and Cats

Category/Agent	Dose	Route	Interval
Antiemetics			
Dolasetron	0.6 mg/kg	PO, SC, IV	24 hr
Maropitant	1 mg/kg	SC	24 hr
	2 mg/kg	PO	24 hr
Ondansetron	0.1 mg/kg	IV (slow)	6–12 hr
Analgesics			
Buprenorphine	0.005–0.015 mg/kg	PO, SC, IM, IV	6–12 hr
Fentanyl	0.004–0.01 mg/kg	IV, IM, SC	2 hr
	0.001–0.004 mg/kg/hr	CRI	N/A
	Patch size is based on patient size	Transdermal	3–4 days
Meperidine	5–10 mg/kg (dogs)	SC, IM	30 min–1 hr
	2–5 mg/kg (cats)	SC, IM	30 min–1 hr
Morphine	0.5–2 mg/kg (dogs)	SC, IM	3–4 hr
	0.1–0.4 mg/kg (cats)	SC, IM	3–6 hr

patches or NSAIDs might be useful in these cases.[62] NSAIDs should be used with caution in animals that are vomiting or have diarrhea because they may contribute to acute renal failure in dehydrated animals by causing renal vasoconstriction and ischemia. Pancreatic enzyme supplementation has been reported to provide pain relief in humans with chronic pancreatitis.[63] However, meta-analysis studies of the effect of pancreatic enzyme supplementation in human patients with chronic pancreatitis did not confirm these findings.[64] Studies are lacking in dogs and cats, but trial therapy with pancreatic enzyme supplementation has been recommended by some authors.[3,5]

Nutrition

"Resting the pancreas" by withholding food and water is a common strategy in humans and animals with pancreatitis.[3,5,6,65] However, the benefits of this approach remain unproven in either humans or animals, and it is possible that the usefulness of this strategy is limited to controlling vomiting and abdominal pain.[3,5,6,65] Recent investigations indicate that humans and dogs with acute pancreatitis may benefit from early nutritional support, and the same may be true for cats.[65–68] Also, recent studies suggest that enteral nutrition is superior to total parenteral nutrition in the treatment of acute pancreatitis in humans and animals.[65,68,69] In dogs with experimentally induced acute pancreatitis, early intrajejunal nutrition did not exacerbate pancreatitis and had beneficial effects, which were attributed to reduced bacterial translocation from the intestine and a reduced systemic inflammatory response rather than a decrease in pancreatic stimulation.[66,68]

In general, oral feeding is not contraindicated in nonvomiting patients with mild or moderate pancreatitis. Even vomiting may not be an absolute contraindication to oral feeding; in one study,[70] dogs with severe vomiting and diarrhea due to canine parvoviral enteritis were successfully managed with early enteral nutrition (nasoesophageal tubes) and showed more rapid clinical improvement (resolution of vomiting and diarrhea) compared with a group that was offered nothing by mouth. If vomiting is severe and there is a risk of aspiration pneumonia, oral intake of food and water can be restricted until vomiting is absent for 12 to 24 hours.[3,5,71] After this period, small amounts of water can be introduced and, if vomiting does not recur, small amounts of a low-fat diet can be offered. Introduction of enteral nutrition during oral food restriction seems to be beneficial. Enteral nutrition is strongly recommended for dogs and cats given nothing by mouth for more than 3 to 4 days or 2 days, respectively.[3,5,71,72] This is particularly important in cats that present with a history of anorexia of several days' duration because additional food restric-

tion could induce or worsen hepatic lipidosis.[3,18,72] Esophagostomy, gastrostomy, or jejunostomy (i.e., distal to the site of pancreatic stimulation) tubes are useful for nutritional management of anorectic animals.[3,5,72]

Dietary modification is probably the most important component of long-term management of dogs with chronic pancreatitis. As stated earlier, hyperlipidemia and obesity are both risk factors for canine pancreatitis, and these two conditions can be effectively managed with dietary modification in most cases. Fat restriction prevents or corrects hyperlipidemia and reduces pancreatic stimulation.[71] Lifelong fat restriction has been recommended for all dogs with chronic pancreatitis, regardless of whether they are hyperlipidemic.[5] Obese dogs also benefit from low-fat diets as part of a weight management program. Obese dogs and dogs with primary hypertriglyceridemia should be fed a high-quality, balanced, low-fat diet (fat content <10% of dry matter), and table foods and treats should be avoided.[38,39,71] In about 10% of hyperlipidemic dogs, low-fat diets alone are not enough to effectively reduce the serum triglyceride concentration to 500 mg/dl or less.[39,71] Use of gemfibrozil, fish oils, and/or niacin may help in these cases, but limited or no data are available about the use of these therapeutic agents in dogs.[39] No relationship between pancreatitis and hyperlipidemia or obesity has been reported in cats; therefore, the need for long-term fat restriction is less clear in this species. Protein restriction may be beneficial for animals with chronic pancreatitis because amino acids stimulate pancreatic secretion.[71,73] Protein levels (dry matter basis) of 15% to 30% for dogs and 30% to 45% for cats have been recommended.[71]

Antiemetic Therapy

When vomiting is severe or persists despite fasting, antiemetic therapy should be initiated. We have found dolasetron, a serotonin antagonist, to be very useful in treating nausea and vomiting in dogs and cats with acute or chronic pancreatitis. A new antiemetic, maropitant, has recently become available and been shown to have superior antiemetic efficacy in dogs.[74] This antiemetic is not labeled for use in cats, but anecdotal evidence of its use in cats suggests that it is also effective in this species.

Antibiotic Therapy

The routine use of antibiotics is considered to be of no benefit in animals with pancreatitis because, in contrast to human pancreatitis patients, bacteria do not seem to play a role in pancreatitis in small animals.[3,5] However, antibiotics are recommended in dogs and cats with pancreatitis when infectious complications have been identified or are suspected (e.g., toxic neutrophils, persistent fever).[3,5] Antibiotics should also be used in cats with concurrent neutrophilic cholangitis. Enrofloxacin and ampicillin seem to penetrate well into the pancreatic tissue.[5]

Other Treatments

Cats (and possibly some dogs) with chronic pancreatitis and concurrent IBD and/or cholangitis can be a therapeutic challenge. Use of oral corticosteroids, which are beneficial for treating IBD and some forms of cholangitis, may be of benefit in these patients because there is no evidence that they aggravate pancreatitis.[3] Budesonide, a potent glucocorticoid analogue that has high topical (intestinal lumen) antiinflammatory activity and minimal systemic effects, might be useful in some of these cases.[62,75] Antibiotic therapy should be initiated in cats with neutrophilic cholangitis, which may be associated with an infectious etiology. Parenteral cobalamin supplementation is recommended for cats with pancreatitis and low serum cobalamin concentration because it has been found to be beneficial in cats with gastrointestinal diseases and hypocobalaminemia.[76] Transfusion of fresh-frozen plasma has been suggested to be a helpful therapeutic measure in animals with severe acute episodes of pancreatitis because it provides natural protease inhibitors (mostly α-macroglobulins) and albumin, but its benefits remain unproven.[5] Surgical treatment might be indicated in cases in which pancreatitis is complicated by pancreatic abscesses, large pseudocysts, or complete bile duct obstruction.

Many other therapeutic agents (e.g., dopamine, somatostatin, glucagon, H_1 and H_2 inhibitors) have been studied for the treatment of acute and, sometimes, chronic pancreatitis. However, none of these therapies can currently be recommended for clinical use in dogs and cats with chronic pancreatitis.[3,5]

COMPLICATIONS OF CHRONIC PANCREATITIS

Diabetes Mellitus

Extension of inflammation from the exocrine to the endocrine pancreas can lead to progressive destruction of the islets of Langerhans, impaired β-cell function, and, subsequently, diabetes mellitus. Chronic pancreatitis is a well-known cause of diabetes mellitus in humans, and this relationship has also been suggested in dogs and

cats.[4,8,9,77,78] Histologic evidence of chronic pancreatitis has been reported in about 28% of diabetic dogs,[78] and almost 50% of 43 dogs with histologically confirmed chronic pancreatitis also had diabetes mellitus.[4] A recent study of 37 diabetic cats revealed histologic evidence of chronic pancreatitis in 51% of these cats.[8] However, whether chronic pancreatitis was the cause of the diabetes mellitus in the above cases is unclear. Insulin therapy is necessary in cases of chronic pancreatitis complicated by diabetes mellitus, and good glycemic control may be particularly difficult to achieve in these animals as long as pancreatic inflammation is present.[8]

Exocrine Pancreatic Insufficiency

Chronic pancreatitis is a well recognized and common cause of EPI in humans[13] and is believed to be the most common cause of feline EPI.[5,79–91] In contrast, pancreatic acinar atrophy seems to be responsible for most cases of EPI in dogs.[5,82–84] In a recent study, 36% of 11 dogs with EPI had pancreatitis, suggesting that chronic pancreatitis is probably the second most common cause of EPI in dogs.[9] Thus, EPI must be considered as a potential long-term complication of chronic pancreatitis in dogs and cats. The test of choice for diagnosis of EPI is serum TLI, which is indicated when signs such as increased appetite, weight loss, and increased fecal volume develop.[85,86] In some animals, chronic pancreatitis may result in EPI and diabetes mellitus, and animals with both of these conditions are more likely to have chronic pancreatitis as the underlying cause.[5]

Pancreatic Complications

Pancreatic pseudocysts, abscesses, and necrotic masses have been infrequently reported in dogs and cats with chronic or acute pancreatitis.[3,5,87–90] The first two complications have been reported in both species, while the third has been reported only in dogs.[3,7,87–90] Clinical signs of pancreatic complications do not differ from those of pancreatitis and may persist despite treatment of pancreatitis. Ultrasonography and ultrasound-guided fine-needle aspiration can be very useful for diagnosing, differentiating, and managing pancreatic complications.[5,87–90] However, it can be difficult to differentiate a pancreatic mass effect caused by pancreatitis from one caused by a pancreatic complication. Histopathology may be required for definitive diagnosis and differentiation of pancreatic complications in some cases.[90] Surgical treatment might be indicated in some cases to resolve large pancreatic abscesses or remove necrotic masses.[90]

Pancreatic Cancer

It has been suggested that humans with chronic pancreatitis may be at increased risk for the development of pancreatic cancer.[13,91] It is not known whether such a relationship exists in dogs and cats.

PROGNOSIS

The prognosis for dogs and cats with chronic pancreatitis varies widely and cannot always be predicted. For animals with mild, uncomplicated disease, the prognosis is usually good if high-fat diets are avoided. However, for animals that have severe disease with frequent acute episodes or complicating diseases, the prognosis should always be guarded.

In cats with acute pancreatitis, a decreased serum ionized calcium concentration and hepatic lipidosis have been reported to be associated with a poor prognosis.[18,92] Although low ionized calcium concentrations and hepatic lipidosis appear to also occur in cats with chronic pancreatitis, an association between these variables and clinical outcome has not been documented in these cases.[16]

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ARTICLE #3 CE TEST CE

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1. Which disease is most commonly associated with feline chronic pancreatitis?
a. FIV infection
b. toxoplasmosis
c. IBD
d. hyperlipidemia

2. The etiology of pancreatitis in miniature schnauzers is
a. hyperlipidemia.
b. hereditary.
c. unknown.
d. none of the above

3. Which endocrinopathy has not been associated with pancreatitis in dogs?
a. hypothyroidism
b. insulinoma
c. hyperadrenocorticism
d. diabetes mellitus

4. What is/are the most common clinical sign(s) in cats with chronic pancreatitis?

a. vomiting
b. diarrhea
c. abdominal pain
d. anorexia/lethargy

5. Which test(s) or diagnostic method is/are of no clinical use for the diagnosis of chronic pancreatitis in cats?

a. serum amylase and lipase activities
b. serum fTLI concentration
c. serum fPLI concentration
d. ultrasonography

6. The most sensitive and specific test or method currently available for diagnosing pancreatitis in dogs and cats is

a. serum lipase activity.
b. serum TLI concentration.
c. ultrasonography.
d. serum PLI concentration.

7. Acute and chronic pancreatitis in dogs and cats can be accurately differentiated based on

a. the duration and severity of clinical disease.
b. ultrasonographic findings.
c. histopathology.
d. none of the above

8. Which statement regarding chronic pancreatitis is true?

a. It is characterized by histopathologic evidence of fibrosis.
b. It can be clinically characterized by spontaneous relapses and remissions.
c. It can lead to endocrine and exocrine pancreatic insufficiency.
d. all of the above

9. What should be the first step in the management of an animal that presents with severe vomiting and diarrhea caused by an acute episode of chronic pancreatitis?

a. fluid therapy
b. analgesia
c. parenteral nutrition
d. antiemetic therapy

10. The most important component of long-term therapy in dogs with chronic pancreatitis is

a. dietary modification.
b. analgesic therapy.
c. antibiotics.
d. pancreatic enzymes.