Cats don’t get acute pancreatitis. Well that’s what was generally believed until 2 case series in 1989 and 1993 clearly demonstrated that acute pancreatitis occurs in cats.1,2 The 1st series of 96 cats described pancreatitis as acute in 33% of cats (neutrophils, necrosis, edema, hemorrhage, and no fibrosis), chronic active in 25% of cats (neutrophils, necrosis and chronic mononuclear inflammation, nodular hyperplasia, and fibrosis), and chronic in 41% of cats (mononuclear infiltration and extensive fibrosis).1 The 2nd case series further characterized acute pancreatitis as acute necrotizing (32 of 40 cats: acinar and peripancreatic fat necrosis) and supplicative (8 of 40 cats: little or no fat necrosis and extensive supplicative inflammation).2 Necrotizing pancreatitis was further subclassified by the presence (11 of 33 cats) or absence (22 of 33 cats) of fibrosis. The acute necrotizing classification of Hill and Van Winkle2 seems likely to encompass the majority of cats with acute and chronic active pancreatitis described by Macy.1

Although pancreatitis has been diagnosed as the sole or predominant disease entity in cats at postmortem examination, accounting for 1 in 600 feline fatalities at the University of Pennsylvania,2 pancreatitis has also been variably associated with diseases in other organs such as the liver (cholestasis, cholangiohepatitis, hydropic change, and lipodosis), the kidney (mild to severe nephritis), the endocrine pancreas (diabetes mellitus), the lungs (pulmonary thrombosis), and the intestine (ulceration, inflammation, and lymphoma).2,3 Effusions have also been noted in the pleural and peritoneal cavity. Whether these changes arise as a consequence of pancreatitis, or are associated with disease processes that cause pancreatitis, or are unrelated to pancreatitis is unclear at this time.

Despite these postmortem studies firmly establishing pancreatitis as a clinically significant disease in cats, the antemortem diagnosis of feline pancreatitis has been reported rarely.4,5,6 This is not really surprising when one considers that cats with pancreatitis often present with a history of anorexia, lethargy, weight loss, or vomiting, and display clinical clinicalpathologic findings such as high liver enzyme activities or hyperbilirubinemia that could easily be attributed to organ dysfunction in the absence of pancreatitis. Even cats with experimentally induced severe pancreatitis exhibit clinical signs and clinicalpathologic findings that are subtle and nonspecific. The situation is further complicated by the observation that serum activities of amylase and lipase, biochemical markers that are widely considered helpful for detecting pancreatic inflammation, are often normal in cats with acute pancreatitis. Thus, the clinician wrestling with the possible diagnosis of pancreatitis in a cat is clearly in need of help in the form of noninvasive indicators of pancreatitis.

It is against this background that the study of Gerhardt et al8 was undertaken. In their study, the authors compared the sensitivity of serum trypsinlike immunoreactivity (TLI), a pancreas-specific marker measured by a species-specific radioimmunoassay in the laboratory of Dr David Williams; ultrasonography; and contrast-enhanced computed tomography (CE-CT) for the diagnosis of pancreatitis in cats. The major inclusion criterion for the study was a grossly abnormal pancreas at laparotomy. Twenty-one cats with gross evidence of pancreatitis were evaluated with TLI and ultrasonography. Ten cats with histopathologically confirmed pancreatitis were additionally subjected to CE-CT; a procedure recognized as an accurate indicator of pancreatic necrosis in humans.

The clinical findings of anorexia, lethargy, and vomiting; nonspecific clinicopathologic test results; and the frequent presence of nonpancreatic conditions such as inflammatory bowel disease, diabetes mellitus, and neoplasia in this group of 21 Austrian cats are very similar to those described in North American and British cats with pancreatitis.2,4,5 The authors reported high concentrations of TLI in 33–86% of cats with pancreatitis, depending on the diagnostic cutoff value employed, and demonstrated that TLI is more sensitive than ultrasonography or CE-CT for detecting pancreatitis in cats. This preliminary evaluation is clearly good news for clinicians waiting for a noninvasive test to aid in the diagnosis of pancreatitis in cats. However, as with all diagnostic tests, the TLI test has its limitations. The observation that 3 of 21 cats with acute pancreatitis did not have high circulating TLI is similar to previous studies that documented concentrations of TLI within the reference range in 12 of 24 cats with histologically confirmed pancreatitis,6,7 and clearly impacts the sensitivity of the TLI test. The relationship between pancreatitis and TLI is an important area for further study—i.e., is trypsinogen synthesis downregulated in cats with pancreatitis or could pancreatitis decrease the immunoreactivity of TLI?

In addition to considering the sensitivity of a test, clinicians must also take into account specificity and disease prevalence. Although the study of Gerhardt et al8 does not directly address specificity (no cats without pancreatitis were included), the observation that azotemia, a complication known to increase TLI in other species, was present in 8 of 21 cats has implications with respect to the specificity of the TLI test. Previous studies have also indicated that TLI concentrations may be markedly and persistently high in cats suffering from inflammatory bowel disease or gastrointestinal lymphoma that have normal pancreatic histology.6,7 Abnormally high TLI results in the face of normal pancreatic histology would decrease the specificity of the TLI test. The reason for high TLI concentrations in the face of nonpancreatic disease is unclear; perhaps it reflects increased synthesis of trypsinogen rather than release of active trypsin. This relationship is another area that requires further study. It also should be borne in mind that even if the TLI test was 100% sensitive, the low prevalence of pancreatitis would have a marked impact on the positive predictive value and diagnostic accuracy of the test. In this respect, the combined application of serologic tests and diagnostic imaging may offer better diagnostic accuracy than a single test. For example, the specificity of imaging combined with the sensitivity of TLI may be more accurate than TLI or imaging alone. The application of CE-CT to cats with pancreatitis is perhaps the most novel aspect of the study of Gerhardt et al8 and should not be overlooked. The
absence of visualization of the pancreas in 8 of 10 cats evaluated with CE-CT suggests that this imaging modality will not be as useful in evaluating cats with pancreatitis as it has been in humans.

In conclusion, acute pancreatitis in cats clearly has emerged as a disease of global significance. Accurate non-invasive diagnosis is important and the development of noninvasive tests for feline pancreatitis, such as the TLI test, is a large step in the right direction. The unknowns of feline pancreatitis are many and they will hopefully spur curious clinicians to conduct further studies to solve the problems associated with diagnosing acute pancreatitis in cats, and elucidating its relationship to nonpancreatic disease. Then we can concentrate on preventing and treating pancreatitis!

References


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