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ISSUES IN GASTROINTESTINAL DISORDERS

Diagnosing Acute Pancreatitis in Dogs

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The pancreas, a tubuloalveolar gland situated in the right cranial quadrant caudal to the stomach, is composed of two separate tissues, referred to as the exocrine and endocrine pancreata.¹ The exocrine pancreas, which comprises about 98% of the pancreatic mass, secretes zymogens (inactive pancreatic enzymes) and active enzymes that enable the digestion of carbohydrates, proteins, and triglycerides.

Pancreatitis causes significant illness in dogs and is considered to be the most common disease of the exocrine pancreas, although the exact prevalence is unknown.¹⁻¹¹ A recent study evaluating dogs that had been necropsied found that 8% showed macroscopic evidence of pancreatitis and approximately 37% had microscopic lesions suggestive of either acute or chronic pancreatitis.¹²

Despite raised awareness of, increased knowledge about, and new diagnostic tests for pancreatitis, the mortality rate in dogs is high, ranging from 27% to 58%.^{2,5,8,10,13,14} These rates are possibly overestimated, as they are based on study

populations from referral institutions. Referred animals often have systemic effects associated with severe, complicated pancreatitis that requires intensive treatment and hospitalization rather than mild, self-limiting disease.^{4,13}

The diagnosis of acute pancreatitis can be difficult because clinical signs and results of diagnostic testing are often nonspecific. This article addresses the diagnostic approach to patients with signs suggestive of pancreatitis; treatment of pancreatitis will be addressed in the March/April issue.

PATHOPHYSIOLOGY

The underlying pathophysiology of acute pancreatitis is incompletely understood, with current understanding extrapolated from human and experimental models.^{1-3,15} The pathogenesis is complex, involving multiple inflammatory pathways.¹⁵ Regardless of the inciting cause, it is accepted that acute pancreatitis is initiated through dysregulated, uncontrolled activation of pancreatic proteolytic enzymes within acinar

BEYOND THE BASICS

Due to the wide range of clinical signs and possible causes for pancreatitis, there are multiple tests and methods for determining the diagnosis of pancreatitis.



cells and the overwhelming of safeguards within the acinar cells and the systemic circulation.^{1,3,4,15-18} Initially, this results in excessive activation of trypsin, which then results in further activation of all zymogens within pancreatic tissue.^{3,15,18,19}

If more than 10% of acinar trypsin is activated and plasma protease inhibitors are depleted, pancreatic safeguards (i.e., pancreatic secretory trypsin inhibitor) are overwhelmed.^{3,15,18,19} Release of the pancreatic enzymes into the pancreatic tissue results in neutrophilic inflammation of the pancreatic and peripancreatic fat. A complex and interdependent “cytokine storm” ensues, leading to production of reactive oxygen species, nitric oxide, and cytokines, as well as activation of other pathways (complement, kinin-kallikrein, and renin-angiotensin systems), which further perpetuates the local inflammatory reaction.³ The end result is digestive enzyme damage not only to the acinar cell, but to the vascular endothelium, producing microcirculatory changes, vasoconstriction, capillary stasis, progressive ischemia, and edema of the pancreas.^{3,18} Generated inflammatory free radicals and inflammatory cytokines may be released into the circulation, which may lead to organ failure (e.g., acute respiratory distress syndrome and acute kidney injury).^{20,21}

CLINICAL SIGNS

Acute pancreatitis is reversible unless the initial trigger persists or there is chronic recurrent inflammation.^{3,5,16,19} In veterinary medicine, the pathologic description depends on histologic descriptions, with different forms of pancreatitis (e.g., acute necrotizing, acute, chronic) overlapping in clinical presentation.^{3,16}

No single clinical sign or combination of clinical signs is pathognomonic for pancreatitis.^{2,10,16} Clinical signs are nonspecific and rely on the degree and severity of local pancreatic inflammation as well as the existence of concurrent systemic complications (**BOX 1**).^{2,3,19}

Dogs with subclinical or mild disease can display nonspecific signs such as intermittent anorexia and weakness with no gastrointestinal signs. These may be self-limiting or may require supportive medical therapy.^{5,8} Dogs presenting with severe disease may present with signs referable to cardiovascular shock, disseminated intravascular coagulation, or multiorgan failure and die within hours of the development of clinical signs.¹⁶

BOX 1 Common Clinical Findings in Dogs With Acute Pancreatitis^{6,9,22-25}

- Decreased appetite/anorexia
- Vomiting
- Abdominal pain
- Weakness/lethargy
- Diarrhea
- Dehydration

RISK FACTORS

Most cases of acute pancreatitis are considered to be idiopathic, with no identified inciting cause.^{1,16} Dogs of any age can be affected; most dogs are middle-aged or older (>5 years).^{6,14,16,26} Several epidemiologic factors and pathologic conditions have been identified as potential risk factors (**BOX 2**). Obese or overweight dogs older than 7 years are at a greater risk.^{3,6,14,27} Terriers (e.g., miniature schnauzers, Yorkshire terriers) and nonsporting breeds (e.g., miniature poodle) are at an increased risk, although lifestyle (i.e., diet and exercise) may be factors that contribute to the development of pancreatitis in these breeds.¹⁴ Neutered status has been associated with the development of acute pancreatitis;²⁷ however, in the author's opinion, this may not be a true association, as it cannot be distinguished from the effects of diet, lifestyle, and exercise. Cause-and-effect relationships have not been established for most published risk factors, with the veterinary literature consisting of single case reports and relatively small retrospective studies. The presence of a factor(s) along with compatible clinical signs should raise suspicion for pancreatitis.^{3,16}

Ingestion of unusual food items or garbage is linked to the development of pancreatitis (i.e., inappropriate food rather than the fat/protein content of food).^{3,27}

Certain breeds likely have a genetic predisposition.¹ Genetic studies of acute pancreatitis have focused on *SPINK1* gene variations in miniature schnauzers.^{28,29} The *SPINK1* gene codes for the pancreatic secretory trypsin inhibitor, which acts as a defense mechanism against premature activation of trypsinogen. Recently, the significance of previous findings has been called into question, with one gene variant being commonly identified in schnauzers both with and without a

history of pancreatitis. Furthermore, a relationship between the *SPINK1* gene variant and clinically detectable pancreatitis could not be confirmed.³⁰

DIAGNOSTIC APPROACH AND DIFFERENTIAL DIAGNOSIS

Diagnosis and the diagnostic approach to acute pancreatitis remain challenging due to the variable severity and inconsistent presenting clinical signs associated with the disease. In dogs presenting with acute gastrointestinal distress and abdominal pain, surgical disease requiring intervention must be initially ruled out (**FIGURE 1**). At the author's institution, acute pancreatitis is considered a diagnostic differential in animals presenting with anorexia, vomiting, dehydration, and abdominal pain.

BOX 2 Risk Factors Associated With Acute Pancreatitis^{1,3,6,16,27}

Epidemiologic factors

- Breed (terriers, schnauzers, poodles, Cavalier King Charles spaniels)
- Age
- Dietary factors (low-protein/high-fat diets, ingestion of garbage and unusual food)
- Obesity
- Neutered/spayed

Pathologic conditions

- Previous surgery (e.g., adrenalectomy, neuter, spay)
- Hyperlipidemia
- Endocrine disease (diabetes mellitus, hyperadrenocorticism, hypothyroidism)
- Hypercalcemia
- Adverse drug reactions (azathioprine, L-asparaginase, sulfonamides, zinc, clomipramine, hydrochlorothiazide, potassium bromide with phenobarbital)
- Duct obstruction (i.e., ductal hypertension)
- Duodenal/biliary reflux
- Ischemia
- Infectious (e.g., babesiosis, leishmaniasis)
- Toxins (organophosphates)

Hematology, Serum Biochemistry, and Urinalysis

Results of a complete blood count (CBC), serum biochemistry panel, and urinalysis convey general information about the patient and are useful in the diagnosis or exclusion of other diseases.¹⁶ However, these results are often nonspecific and highly variable and may be within normal limits in mild cases.^{16,18} Abnormalities, when present, are the result of hypovolemia and inflammation. In brief, changes include leukocytosis, thrombocytopenia, azotemia, and increased liver enzymes. Variable electrolyte abnormalities have been identified. In some cases, coagulation abnormalities may be present (i.e., prolonged activated clotting time, prothrombin time, or partial thromboplastin time), which are invariably associated with spontaneous bleeding.¹⁶

Diagnostic Imaging

Abdominal Radiography

Abdominal radiographs are insensitive for detecting abnormalities associated with pancreatitis.^{3,4,16,17} When present, radiographic findings are nonspecific and may include increased soft tissue opacity, decreased serosal detail in the right cranial abdomen, widened pyloric duodenal angle, displacement of the stomach and/or duodenum from their normal position, gaseous dilation of bowel loops, and abdominal effusions (**FIGURE 2**).^{3,6,31} In dogs presenting with acute gastrointestinal distress and abdominal pain, plain abdominal radiographs are cost-effective, logical, and practical when surgical disease is a possibility (**FIGURE 1**).

Abdominal Ultrasonography

Ultrasonography is the imaging method of choice and has become a readily available and commonly used imaging modality in general practice.^{3,16} Few studies have evaluated the diagnostic utility of abdominal ultrasonography for the diagnosis of pancreatitis.¹⁶ Based on previous reports, the sensitivity of abdominal ultrasonography is approximately 68% in dogs with severe acute pancreatitis.^{3,4,6,16,32} With the evolution and advancement in equipment quality and ultrasonographer experience, sensitivity has most likely increased; however, further studies are needed to assess this. The specificity of ultrasonography has not been evaluated because histology would be required to establish the diagnosis.^{3,16}

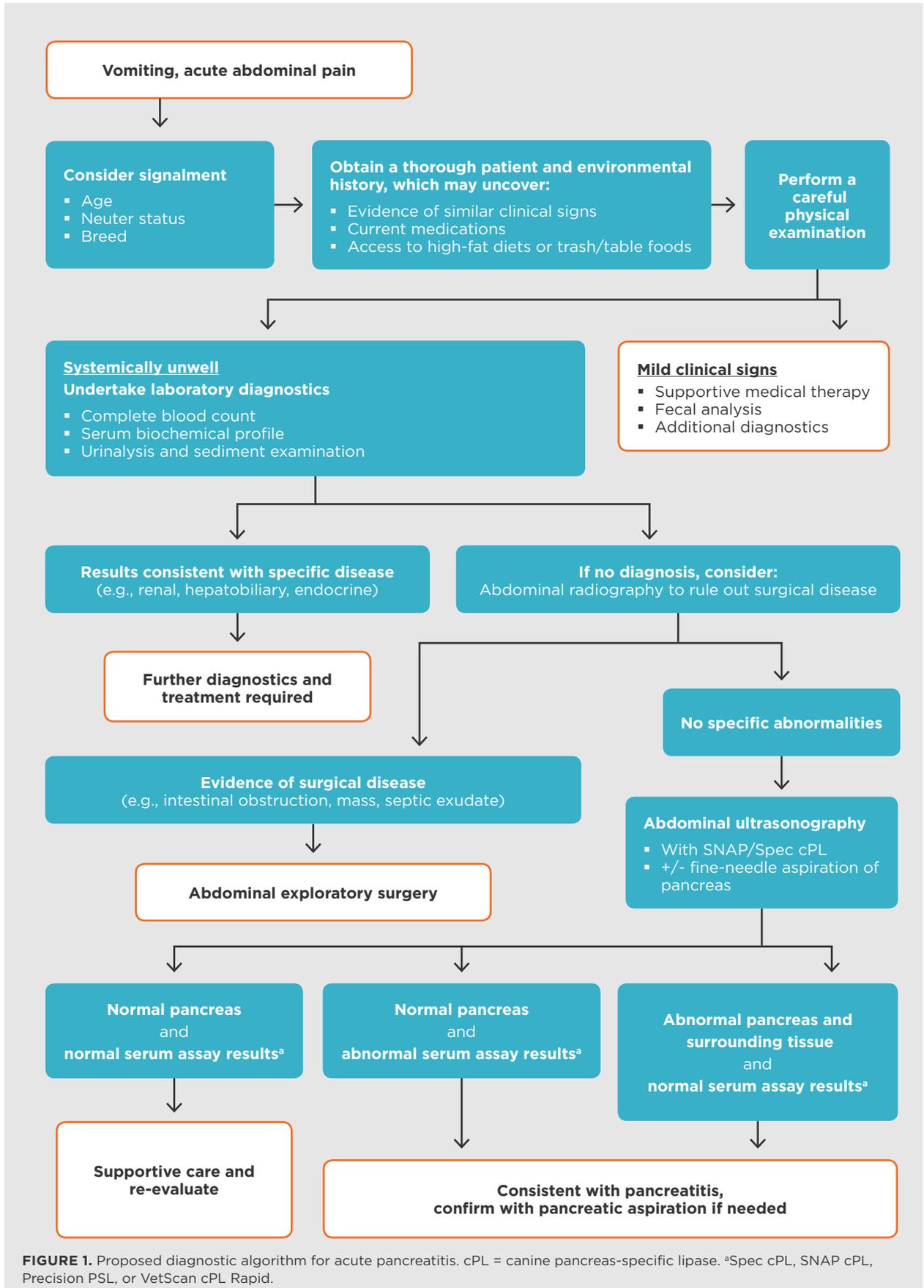


FIGURE 1. Proposed diagnostic algorithm for acute pancreatitis. cPL = canine pancreas-specific lipase. ^aSpec cPL, SNAP cPL, Precision PSL, or VetScan cPL Rapid.

Ultrasonography is better at detecting severe acute necrotizing pancreatitis than mild or subclinical pancreatitis. Ultrasonographic findings suggestive of pancreatitis include hypoechoic areas within the pancreas (indicating necrosis or fluid accumulations); hyperechoic peripancreatic mesentery (due to necrosis of peripancreatic fat); an enlarged, irregular pancreas; dilation of the pancreatic or biliary duct; and abdominal effusion (**FIGURE 3**).^{6,33}

Contrast-enhanced ultrasonography has been investigated as a technique for improving the accuracy

of diagnosing pancreatitis in a small number of dogs.^{4,34} The technique investigates tissue perfusion as well as lesional vascular patterns.⁴ Initial results from several studies identified decreased peak pancreatic perfusion and prolonged retention of contrast in the pancreas, most likely secondary to inflammation.^{4,34} Additional studies are warranted to determine whether this modality is superior to traditional abdominal ultrasonography.

Computed Tomography Angiography

Three-phase computed tomography angiography (CTA) has been evaluated in dogs with acute pancreatitis as a diagnostic and prognostication tool.^{12,35} Multiple changes have been demonstrated in the appearance of the pancreas, peripancreatic tissues, and adjacent vessels.^{12,35,36} In a recent study, dogs with more heterogeneous contrast enhancement spent a significantly longer duration in hospital (>5 days), had an increased number of relapses, and were significantly more likely to have portal vein thrombosis.¹² Heterogenous contrast enhancement was correlated to increased Spec cPL (IDEXX Reference Laboratories; idexx.com) when compared with homogeneous pancreatic contrast enhancement.¹² Initial findings indicate that CTA is an additional, superior diagnostic modality in evaluating dogs with suspect acute pancreatitis; however, further studies are warranted.

Serum Assays

Based on their increased activity in experimentally induced pancreatitis, serum amylase and lipase were previously used to assess for clinical pancreatitis.^{3,9,16} However, due to their poor sensitivities and specificities, they are no longer used to assess for spontaneous pancreatitis.^{9,16,37-39}

Canine pancreatic lipase originates only from pancreatic acinar cells.^{9,40} During active pancreatic disease, cPL leaks into the serum, leading to an increase in measurable cPL levels.^{9,23,38,39} Initially, a radioimmunoassay measuring cPL immunoreactivity was developed and validated.^{23,38,41} This assay has now been replaced by a quantitative (Spec cPL) and a semi-quantitative (SNAP cPL, idexx.com) colorimetric rapid point-of-care enzyme-linked immunosorbent assay (ELISA).^{9,23,38,39,42} Both tests are now routinely used in veterinary medicine, with the SNAP cPL test being used to rapidly rule out pancreatitis. It is recommended that positive SNAP cPL results be

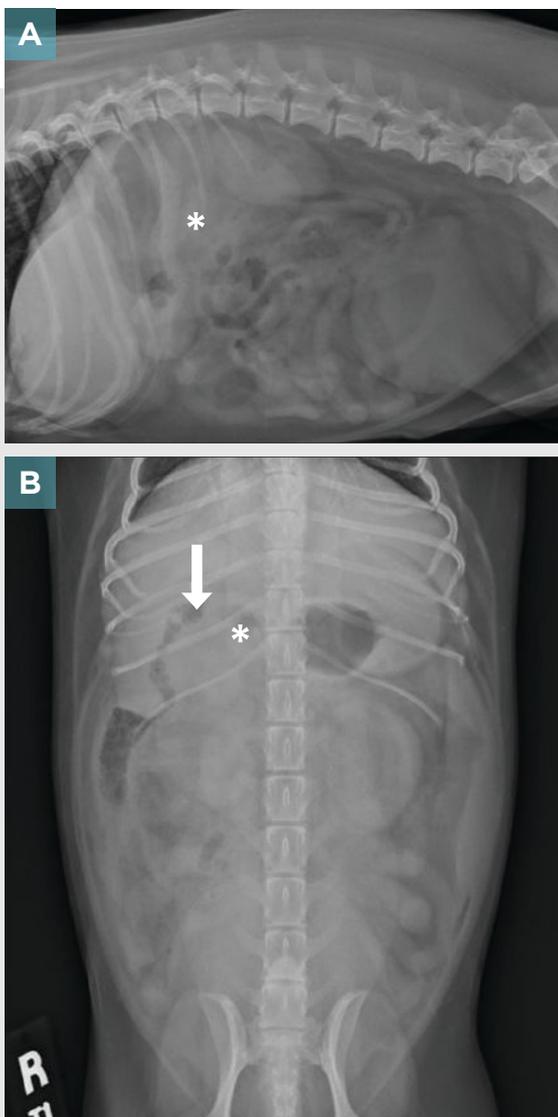


FIGURE 2. Abdominal radiographs from a dog with pancreatitis. In **(A)** and **(B)**, increased soft tissue opacity and decreased serosal detail in the right cranial abdomen can be seen (*). In **(B)**, the pyloric duodenal angle is widened (arrow).

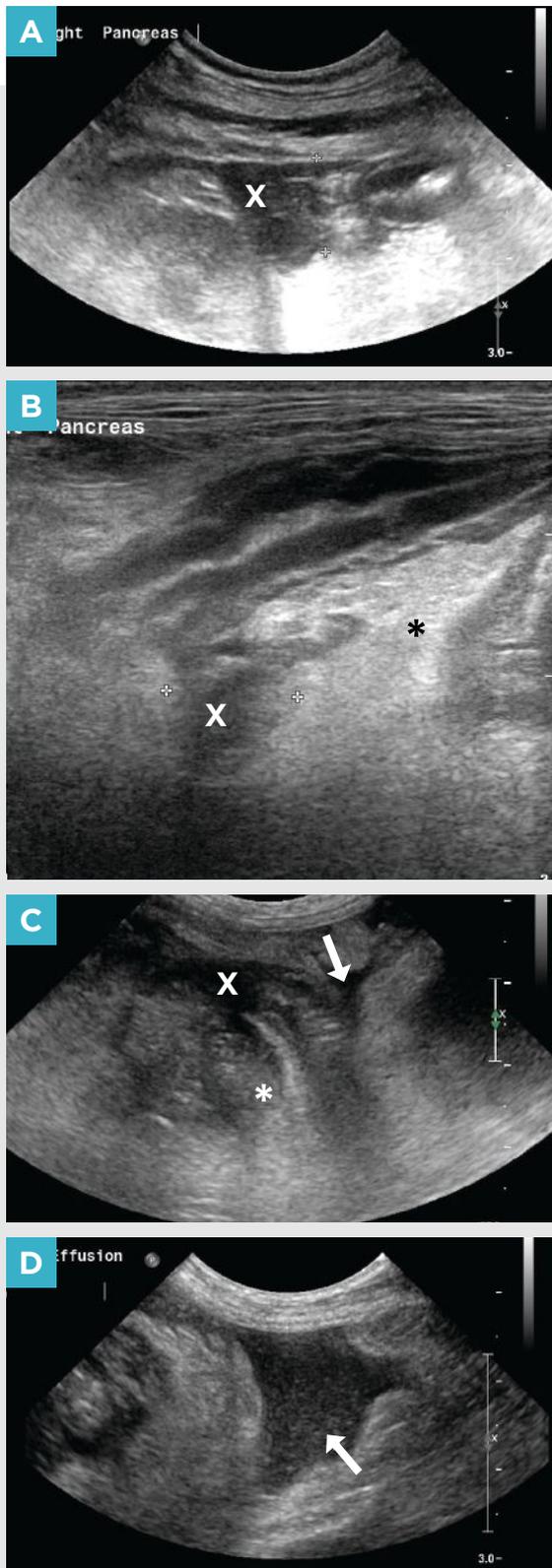


FIGURE 3. Images from a dog with pancreatitis obtained during abdominal ultrasonography. Images (A), (B), and (C) show a hypoechoic pancreas (X); in (B) and (C), the surrounding hyperechoic peripancreatic mesentery is noted (*). In (D), the contour of the pancreas is irregular and abdominal effusion is present (arrow).

followed by laboratory assessment using the quantitative immunoassay (Spec cPL or equivalent).⁹ Spec cPL results ≤ 200 $\mu\text{g/L}$ are consistent with an absence of pancreatic inflammation, while results ≥ 400 $\mu\text{g/L}$ are consistent with a diagnosis of pancreatitis. Results of 200 to 399 $\mu\text{g/L}$ are considered to be equivocal, and further testing using quantitative analysis is recommended in 2 to 3 weeks.^{9,16,23,39,42}

Newer diagnostic assays—the VetScan cPL rapid point-of-care test (quantitative assay; Abaxis, abaxis.com) and the Precision PSL (Antech; antechdiagnostics.com), a nonimmunologic colorimetric lipase diagnostic assay using 1,2-o-dilauryl-rac-glycero-3-glutaric acid (6'-methyl-resorufin) ester (DGGR)—have now been validated for use in dogs.^{7,9,43} All 4 diagnostic assays have been found to have comparable results (TABLE 1). The sensitivity of Spec cPL (or cPL) has been shown to be higher in dogs with increasing histologic severity.^{23,32,37}

No diagnostic test is 100% sensitive or specific, and the clinical diagnosis should be interpreted in conjunction with the signalment, history, physical examination, CBC and serum biochemistry results, and abdominal ultrasonography findings.^{9,23,43} Up to 78% of dogs with upper gastrointestinal obstruction may have an elevated cPL, possibly due to increased leakage of immunoassay-positive lipase into the circulation.⁹ The Precision PSL assay is not specific for pancreatic lipase; therefore, its results may show increased levels in conditions other than pancreatitis.⁹

Histopathology

Histopathology has been previously considered the gold standard for the diagnosis of pancreatitis.¹⁶ This is somewhat inaccurate, as microscopic evidence of pancreatic inflammation does not always translate to clinical presentation or signs. Also, with localized inflammation, representative pathologic samples may not be collected, and the procedure itself is considered invasive, expensive, and not without complications.

Cytology

Pancreatic cytology is gaining popularity in veterinary medicine. It is relatively noninvasive and has good diagnostic yield, with relatively few complications.⁴⁴⁻⁴⁶ Further studies are needed in veterinary medicine to fully evaluate this technique as a diagnostic tool in the diagnosis of acute pancreatitis. At the author's

**TABLE 1 Sensitivity and Specificity of Different Serum Assays for the Diagnosis of Pancreatitis**

ASSAY	CUTOFF VALUE	SENSITIVITY (%)	SPECIFICITY (%)	MANUFACTURER
Spec cPL	≥400 µg/L	70-90.9 ^{9,23,37,39}	74.1-100 ^{9,23,37,39}	Idexx
SNAP cPL	≥200 µg/L	73.9-100 ^{9,23,39}	71.1-77.8 ^{9,23,39}	Idexx
Precision PSL	>216 U/L	85.7-93 ^{9,43}	53.0-74.3 ^{9,43}	Antech
VetScan cPL Rapid	≥400 µg/L	73.9-83.3 ⁹	76.9-83.8 ⁹	Zoetis

institution, fine-needle aspiration of the pancreas is performed to differentiate between pancreatic neoplasia (primary or metastatic) and acute pancreatitis.

CONCLUSION

The diagnosis of acute pancreatitis can be difficult due to the variable and nonspecific spectrum of clinical presentation. A detailed medical history should be initially obtained, with the diagnosis being made based on a combination of presenting clinical signs, serology, abdominal ultrasonography findings, and response to medical therapy. In addition, highly specific and sensitive tests, like cPL, can be used to differentiate acute pancreatitis from other diseases causing similar clinical signs in dogs. Severe acute pancreatitis is often associated with serious systemic complications that, if not accurately recognized and treated aggressively early in the course of the disease, may lead to death.¹⁹ **TVP**

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