Pancreatitis (Canine)

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Synonyms

Acute pancreatitis
Chronic pancreatitis
Pancreatic inflammation

Disease Description

Definition

Pancreatitis is inflammation of the pancreas. It can be acute or chronic in duration, and severity can range from mild to severe.[1-8]

Pathophysiology

Normal Pancreas

The pancreas in dogs and cats is located in the bend of the cranial part of the duodenum, close to the stomach and liver. It consists of a right and left lobe, and a central area where the two lobes meet (**Figure 1**). The pancreas has both endocrine and exocrine functions. Approximately 98-99% of the pancreas is composed of exocrine tissue.

Exocrine function consists of secretion of digestive enzymes, such as protease enzymes that break down proteins. These enzymes include trypsin, chymotrypsin, elastase, carboxypeptidase A and B, and phospholipase A.[1-2] Endocrine functions include production of insulin and glucagon.

Pathophysiology of Pancreatitis

Major digestive enzymes exist in pancreatic acinar cells in inactive forms called *zymogens*. Packaging inactive enzymes into zymogens helps prevent premature activation before they are released into the duodenum.[9] Enzyme inhibitors also exist within the pancreas (e.g. alpha-antitrypsin) and circulate in plasma (e.g. alpha-macroglobulins, antichymotrypsin, alpha-antitrypsin).[1-3] Once zymogens are released into the intestinal lumen, they undergo peptide cleavage by enterokinase secreted by duodenal mucosal cells. This breakdown activates pancreatic enzymes and allows them to begin digesting nutrients.

If inhibiting substances are blocked, or if enzymes are activated while they are still in the pancreas, the pancreas inappropriately begins to digest itself. For example, the conversion of trypsinogen (inactive) to trypsin (active form) can be triggered by enterokinase, bile, lysosomal enzymes, or other stimuli. The result is disruption of pancreatic membranes, arteriolar dilation, increased vascular permeability, edema, and hemorrhage, with subsequent pain, leukocytic infiltration, and peripancreatic fat necrosis.

Reduced pancreatic blood flow and leukocyte infiltration can lead to pancreatic necrosis. Secondary infections can occur due to bacterial translocation from the intestines.[9] Arterial hypotension, portal venous pooling, and hypovolemia may lead to shock.

Peripheral venoconstriction and leakage of pancreatic enzymes into the abdominal cavity and vascular compartment compound the damage. Regional tissue invasion and destruction caused by release of pancreatic enzymes can be extensive. Possible end results include damage to liver,

kidneys, lungs, heart, and abdominal lymphatics.[1-3,9] Pancreatitis can cause extrahepatic biliary tract obstruction.[6] The feline pancreas is also prone to ascending biliary infections and bile reflux because the pancreatic and bile ducts merge before reaching the duodenal papilla.[10]

Disease Description in This Species

Types of Pancreatitis

Pancreatitis in the dog can be classified as acute or chronic. Acute pancreatitis is characterized by an infiltration of neutrophils, moderate to severe pancreatic necrosis, edema, and/or hemorrhage. Acinar tissue and ducts remain intact.[2,9] Chronic pancreatitis is long-standing inflammation associated with low-grade, mononuclear inflammation and fibrosis.[2,5,9] Chronic pancreatitis may be a sequela to recurrent, acute pancreatitis.[2,9] Chronic pancreatitis can eventually lead to diabetes mellitus and/or exocrine pancreatic insufficiency.[2] Acute pancreatitis cannot be distinguished from chronic pancreatitis clinically.[2] Although acute and chronic cases can have mild or severe clinical signs, chronic cases more likely to have mild signs, and acute cases most often have severe signs.[1]

Etiology and Risk Factors

Acute Pancreatitis

The precipitating cause of acute pancreatitis is usually unknown. Risk factors associated with fatal, acute pancreatitis include overweight body condition; presence of diabetes mellitus, hyperadrenocorticism, hypothyroidism or epilepsy; and prior history of gastrointestinal (GI) tract disease.[12] In one study, ingestion of unusual or human foods increased the odds of developing pancreatitis.[45]

Other potential risk factors include high-fat diets, malnutrition, hypertriglyceridemia, exposure to toxins (e.g. zinc, organophosphates), hypercalcemia, pancreatic duct obstruction, reflux of duodenal contents into the pancreatic duct, pancreatic trauma (e.g. surgical, blunt), parasites (e.g. flukes), hepatobiliary diseases, small intestinal disorders, and pancreatic ischemia/reperfusion injury.[9,11,13,43,44] Infection with *Babesia rossi* is associated with pancreatitis; however, infection with *Babesia gibsoni* is less likely to cause pancreatitis.[46,47]

Various drugs have been implicated in the development of acute pancreatitis, including thiazide diuretics, furosemide, azathioprine, L-asparaginase, sulfonamides, and tetracycline.[9,11,45] Bromide and phenobarbital administration may also contribute to pancreatitis. In a retrospective study, 10% of dogs receiving combination therapy with potassium bromide and phenobarbital developed pancreatitis compared with 0.3% of dogs receiving phenobarbital alone.[14] In another study of 100 epileptic dogs, 2 dogs receiving only phenobarbital died from acute, hemorrhagic, necrotizing pancreatitis.[15] Keep in mind that because both phenobarbital and bromide can cause polyphagia, dogs may seek out and eat foods that make them more prone to pancreatitis. Glucocorticoids do not appear to increase the risk of acute pancreatitis in clinical cases. Furthermore, experimental evidence suggests that glucocorticoids do not cause pancreatitis.[11]

Chronic Pancreatitis

The cause of chronic pancreatitis is also usually undefined. Chronic pancreatitis can develop as a sequela to recurrent episodes of acute pancreatitis. A particular form of chronic pancreatitis that develops in English cocker spaniels is thought to be an autoimmune disorder that is characterized by pancreatic duct destruction similar to autoimmune pancreatitis in people.[5,48]

Diagnosis

Physical Examination Findings/History: A history of dietary indiscretion, toxin exposure, or drug administration may be present.[9,43] Physical findings can include fever, abdominal pain, a palpable abdominal mass, depression, dehydration, tachycardia, tachypnea, icterus, ascites, prolonged capillary refill time, petechiation, tacky mucous membranes, hypothermia, or fever. Patients with secondary bile duct obstruction may be icteric. Some patients may be presented in shock.[3,9]

Complete Blood Count (CBC): In a study of 70 dogs with severe pancreatitis, neutrophilia with a left shift was found in 55% of the cases.[11] Toxic white blood cells were occasionally seen and suggested a more guarded prognosis.[11] Thrombocytopenia (59%) and anemia (29%) were also frequent findings, and may be early indicators of disseminated intravascular coagulation.[11] Other abnormalities may include leukocytosis, neutropenia, and hemoconcentration.[6,11]

Biochemistry Profile: Abnormalities may include azotemia from dehydration or acute renal failure, and elevation of ALT, AST, ALP, bilirubin, glucose, cholesterol, and triglyceride.[9,18,19] In one study, mild hypercholesterolemia and hypertriglyceridemia were present in 24% and 18% of affected dogs respectively.[18] Hypocalcemia, hypoproteinemia, and hypoalbuminemia may also be noted.[9,18,19] Electrolyte (e.g. sodium, potassium, chloride) values vary but tend to be decreased.[9]

Serum amylase and lipase can be elevated or normal. Serum amylase is normal in 31-47% of dogs with histologically-confirmed pancreatitis.[9] Serum amylase is synthesized and secreted by tissues other than the pancreas, so elevations of amylase are not specific for pancreatitis. Serum lipase is normal in 28-61% of dogs with pancreatitis.[9] In contrast, serum lipase can be elevated in patients without pancreatitis in association with other disorders, such as GI foreign bodies or gastritis.[6] Lipase is produced by the gastric mucosa and can be elevated with gastric inflammation.[6]

Urinalysis: Evaluation of urine specific gravity can help determine if azotemia is pre-renal or renal in origin. Acute kidney injury (AKI) can occur as a comorbidity with acute pancreatitis, as evidenced by the presence of oliguria, anuria, proteinuria, casts and azotemia.[9,49,50] The presence of glucosuria or ketonuria can indicate concurrent diabetes mellitus.[9] Glucosuria must be correlated with blood glucose results since glucosuria with a normal blood glucose can indicate AKI.

Coagulation Profile: Coagulation tests may be abnormal. Both hypocoagulability and hypercoagulability can develop with pancreatitis. In one study of 70 cases, 61% had abnormal coagulation function test results.[11] Various hypercoagulable states can occur, with elevations of D-dimers, fibrinogen, and von Willebrand factor.[9,21] Antithrombin levels may be low, and prothrombin time prolonged.[21]

Canine Trypsin-Like Immunoreactivity (cTLI): Serum cTLI can be elevated with pancreatitis, GI diseases, and azotemia. Sensitivity of cTLI for pancreatitis in dogs is around 35%, making it a poor diagnostic test.[3,6] Decreased cTLI can be used to detect exocrine pancreatic insufficiency that can occur from chronic pancreatitis.

Canine Pancreatic Lipase Immunoreactivity (cPLI): cPLI may be the most useful laboratory diagnostic test in both the dog and the cat. Although a number of organs synthesize and secrete lipases, cPLI measures lipase that only originates from exocrine pancreas.[22] Reported sensitivity for specific (Spec) cPL is 80-93%.[6,51] However, Spec cPL has a lower sensitivity in dogs with mild pancreatitis compared to dogs with moderate disease.[9,51] In a study of 38 dogs with acute abdominal disease, sensitivity and specificity of Spec cPL was 70% and 77%, respectively.[9] Reported specificity of Spec cPL in another study was 66-77%.[52]

Reported sensitivity for SNAP cPL was 82% in one study and ranged from 91-94% another.[23,52] Specificity ranged from 59-77%.[23,52] One study evaluated SNAL cPL, Spec cPL, VetScan cPL Rapid Test, and Precision PSL.[24] Good to excellent agreement was noted between the four assays.[24] Sensitivities ranged from 73.9%-100%, and specificities ranged from 64.0-83.8%.[24] Both false-positive and false-negative results are possible.[53] It is important to note that results of any cPLI assay alone, without support from other clinical findings, is often insufficient to diagnose clinical pancreatitis.[24]

Fluid Analysis/Cytology: Determination of lipase activity in abdominal fluid of patients with abdominal effusion is a reliable marker of acute pancreatitis. Lipase is significantly higher in dogs with acute pancreatitis than those with other causes of ascites (e.g. cardiac disease), or fluid from abdominal trauma and neoplasia.[25] In one study of 34 dogs, sensitivity and specificity of peritoneal fluid amylase was 71.4% and 84.2%, respectively.[26] For peritoneal fluid lipase, sensitivity and specificity was 92.9% and 94.7%, respectively.[26] Sensitivity of peritoneal fluid cPLI concentration was 100%, and specificity was 94.7% (cutoff value of 500 µg/L).[26] Cytology commonly reveals neutrophilic inflammation (**Figure 2**).

Radiography: Abdominal radiographs may show a loss of detail (**Figure 3**) or suggestion of a mass in the cranial abdomen. Transposition of abdominal organs can also occur, such as displacement of the duodenum dorsally and to the right; the stomach to the left; and transverse colon causally.[5,6] Calcification can be noted in the cranial abdomen in some patients with chronic pancreatitis.[5,6,9]

Contrast Radiography: Contrast radiography is not performed to diagnosis pancreatitis but may be done if other GI disorders are suspected. Delayed passage of barium through the stomach and proximal duodenum may occur in cases of pancreatitis.

Ultrasonography: Abdominal ultrasonography is much better at diagnosing pancreatitis than radiography.[54] In acute cases, an enlarged, hypoechoic pancreas is often surrounded by a hyperechoic area that represents peripancreatic fat necrosis (**Figure 4**).[6,9,27,28] Other abnormalities may include free peritoneal fluid, thickening of the pancreas, dilated bile ducts, enlargement of the gallbladder, evidence of pancreatic masses (e.g. abscess, pseudocyst), and gastric wall edema.[5,6,27-30,55] In one study of dogs with pancreatitis, sensitivity of ultrasonography was 68%.[16] In another study of 157 dogs, sensitivity was 43% and specificity was 92% for pancreatitis if pancreatic enlargement, hyperechogenicity, and altered mesenteric echogenicity were all present.[30] Although not commonly performed, quantitative, contrast-enhanced ultrasonography can detect pancreatic perfusion abnormalities in dogs associated with both experimentally-induced and naturally-occurring pancreatitis.[56-58]

Chronic pancreatitis can be associated with a hyperechoic pancreas (**Figure 5**) from pancreatic fibrosis. Even when ultrasound findings are negative and nonspecific, the procedure helps to rule out other abdominal disorders.

Advanced Imaging: Computed tomographic angiography can be used to aid the diagnosis of acute pancreatitis and to identify pancreatic necrosis.[31]

Biopsy/Histopathology: Histopathology is considered the gold standard for diagnosing pancreatitis, and is the only method for definitively differentiating acute pancreatitis from chronic pancreatitis. Samples may be obtained via exploratory laparotomy (**Figure 6**) or laparoscopy.

Other Tests: Plasma and urinary trypsinogen activation peptides (TAP) have been evaluated in normal dogs and in those with pancreatitis and other diseases.[32] Unfortunately, the wide range of urinary TAP concentration in healthy dogs makes interpretation difficult.[32] Plasma and urinary TAP levels can be increased in dogs with severe pancreatitis.[9] C reactive protein (CRP) may be elevated but it is a nonspecific marker of inflammation.[59,60] Serum 25- hydroxyvitamin D may be decreased in acute pancreatitis but the clinical significance is undefined.[61]

Signalment

One study indicated that increasing age and certain breed types were risk factors for pancreatitis.[11] Breeds with reported higher odds of developing pancreatitis include the miniature schnauzer, dachshund, miniature poodle, Cavalier King Charles spaniel, cocker spaniel, collie, boxer, as well as Yorkshire, fox, and other terriers.[5,43,62] In one study, spayed females and castrated males were at increased risk, compared to sexually-intact males.[11] Most affected dogs are middle-aged.[62]

Clinical Signs

Patients with acute pancreatitis may have mild to severe clinical signs. Patients with chronic pancreatitis tend to have mild, intermittent signs.[5,9] Vomiting is present in approximately 90% and abdominal pain in 58% of dogs with pancreatitis.[16,17] Other possible signs include lethargy, anorexia, diarrhea, cranial abdominal pain, restlessness, hematochezia, and hematemesis. Fever, collapse, and obtundation may occur with severe, acute pancreatitis. Evidence of concurrent coagulopathy (e.g. petechiation), hepatobiliary disease (e.g. icterus), diabetes mellitus (e.g. polyuria, polydipsia), and AKI (e.g. oliguria) may be present.[9,62] Diabetes mellitus is the most common concurrent disease.[62]

Laboratory Profile

Sodikoff's Laboratory Profiles of Small Animal Diseases: Special Tests
Sodikoff's Laboratory Profiles of Small Animal Diseases: Serum Biochemistry Tests
Sodikoff's Laboratory Profiles of Small Animal Diseases: Hepatopathy, Reactive
Sodikoff's Laboratory Profiles of Small Animal Diseases: Pancreatitis, Acute

Genetic Basis

Etiology

Asparaginase
Azathioprine
Babesia canis rossi
Babesia spp.
Biliary disease
Diabetes mellitus
Diet
Dietary indiscretion
Duodenal reflux
Furosemide
Hepatic disease
Hyperadrenocorticism
Hypercalcemia
Hyperlipidemia, lipemia
Hypothyroidism
Idiopathic, unknown
Immune-mediated disease
Infection
Ischemia
Obesity
Organophosphate
Pancreatic duct obstruction
Phenobarbital
Potassium/sodium bromide
Sulfonamides
Tetracycline
Thiazides
Trauma
Uremia
Zinc ingestion

Breed Predilection

American cocker spaniel
Cavalier King Charles spaniel
Dachshund
English cocker spaniel
Fox terrier
Miniature poodle
Miniature schnauzer
Miniature wirehaired dachshund
Terriers
Yorkshire terrier

Sex Predilection

Female spayed
Male castrated

Age Predilection

Mature, middle-aged

Clinical Findings

Abdominal distention
Abdominal pain
AFEBRILE
Anorexia, hyporexia
ASCITES OR ABDOMINAL DISTENTION
Capillary refill time prolonged, >2 seconds
Collapse of patient or syncope
Dehydration

Depression, lethargy
DIARRHEA
Ecchymoses, purpura, bruising
Exercise intolerant, reluctant to move
FEVER
Hematemesis
Hematochezia
HEMORRHAGE
Hypothermia
ICTERUS, JAUNDICE
MASS
Mucous membranes pale
Nausea
Oliguria, urine output decreased
Overweight, obese
PAIN
PETECHIAE
Polydipsia
Polyuria
Pulse weak
Shock
TACHYCARDIA
Tachypnea, hyperpnea, polypnea
VOMITING
Weakness: Asthenia or Paresis
ZZZ INDEX ZZZ

Diagnostic Procedures

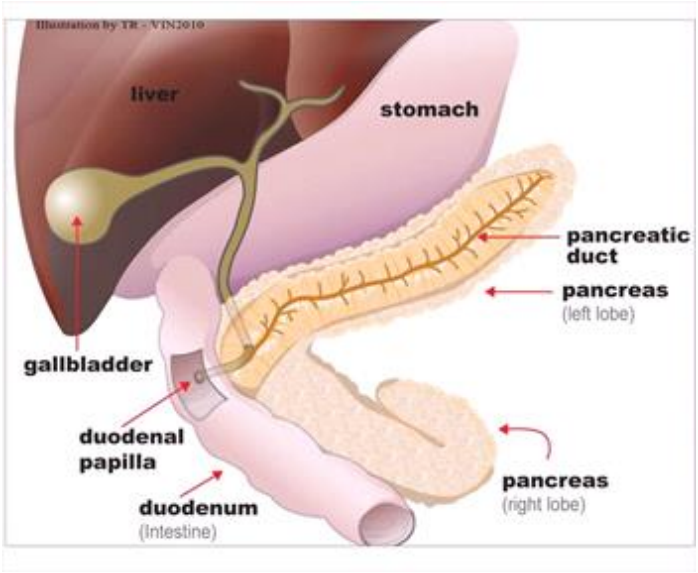
Diagnostic Procedures:	Diagnostic Results:
Pancreatic lipase immunoreactivity in serum	Pancreatic lipase immunoreactivity elevated
Complete blood count (hemogram)	ANEMIA Hemoconcentration or polycythemia Leukocytosis Neutropenia, neutrophils decreased Neutrophilia Thrombocytopenia
Urinalysis	Bilirubinuria, urine bilirubin increased Glucosuria, glycosuria Ketonuria Proteinuria, albuminuria URINE CASTS Urine specific gravity increased
Radiography of abdomen	Abdominal effusion Duodenal displacement to right Opacity right cranial abdomen Pyloric displacement to left
Serum biochemistries	Alanine aminotransferase (ALT) increased Alkaline phosphatase (ALP) increased Amylase increased, amylasemia Aspartate aminotransferase (AST) increased Azotemia/uremia Blood urea nitrogen (BUN) increased Gamma-glutamyl transferase (GGT) increased Hyperbilirubinemia, bilirubin increased

	Hypercholesterolemia Hyperglycemia Hypoalbuminemia Hypocalcemia Hypochloremia Hypokalemia Hyponatremia Hypoproteinemia Lipase increased, lipasemia Lipemia, lipids increased
Ultrasonography of abdomen	Abdominal mass internal Bile ducts prominent Hyperechoic pancreas Hypoechoic pancreas Intestinal, bowel loops fluid filled Pancreatic mass Peripancreatic fluid accumulation
Fluid analysis, abdominal/peritoneal	Amylase and lipase in peritoneal fluid higher than in serum
Needle aspirate and cytology of lesion/affected tissue	Neutrophilic infiltration
Radiography, contrast procedure	Gastric emptying time delayed
Biopsy and histopathology of pancreas	Pancreatitis
Coagulation tests, clotting factor assay	Antithrombin III activity decreased D-dimer levels elevated Disseminated intravascular coagulopathy present Fibrin degradation products (FDP) increased Prothrombin time (PT) prolonged
Computed tomography (CT) or MRI of abdomen	Characterization and extent of the lesion

Images

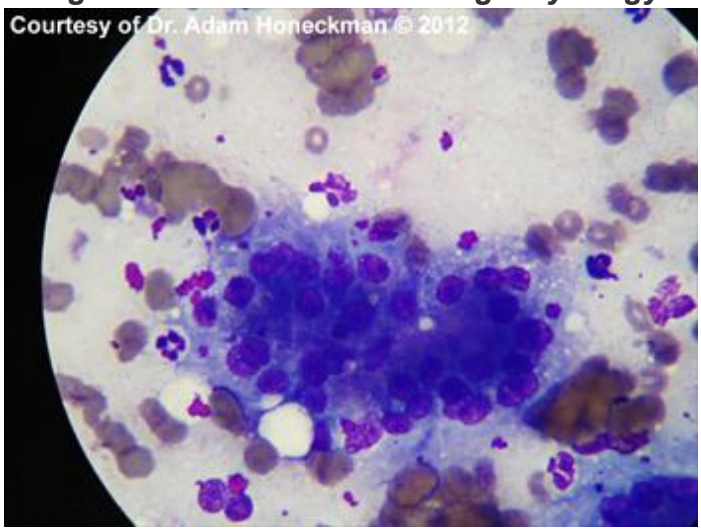
Click on each image to see a larger view

Figure 1. Pancreas area with labels



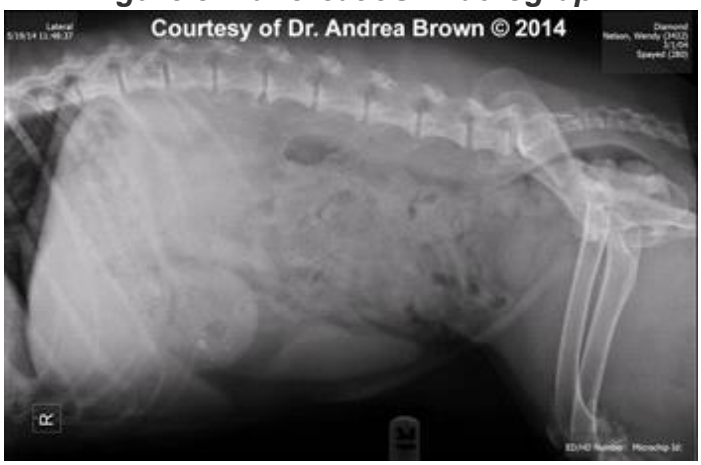
Drawing by Tamara Rees (VIN).

Figure 2. Pancreatitis in a dog – cytology



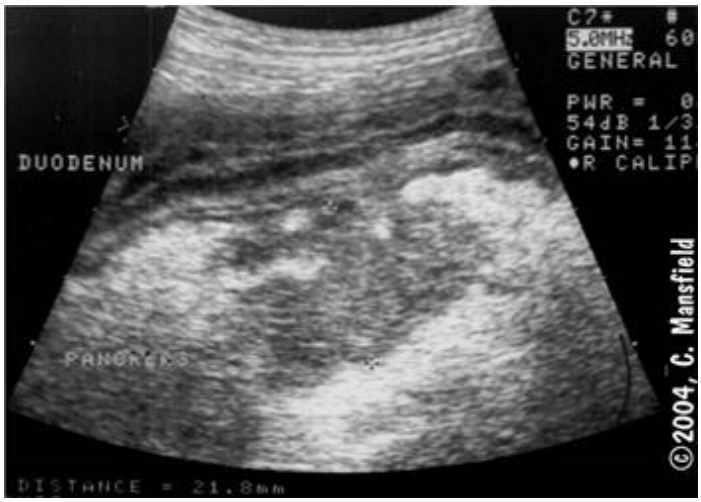
Click here to see board discussion

Figure 3. Pancreatitis – radiograph



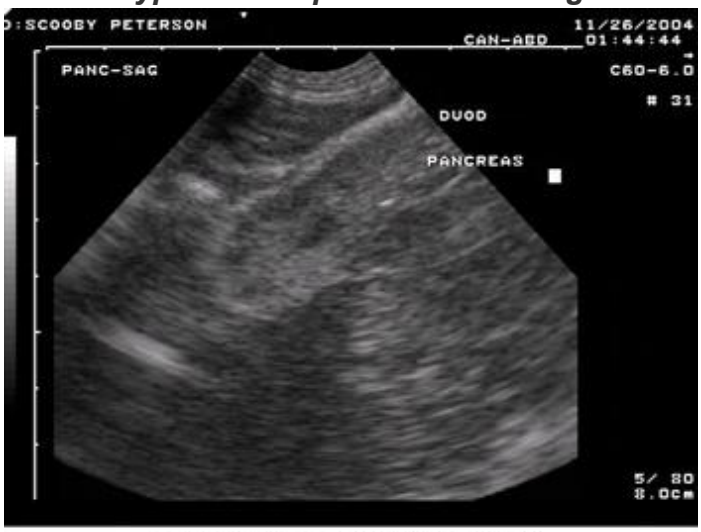
Decreased abdominal detail seen on radiographs from a dog. Ultrasound showed pancreatitis. Click here to see board discussion

Figure 4. Necrotizing pancreatitis in a dog – ultrasound



In this image we can see the hyperechoic areas on the fat surrounding the pancreas and an enlarged pancreas with hypoechoic and hypoechoic areas within it. Click here to see Rounds

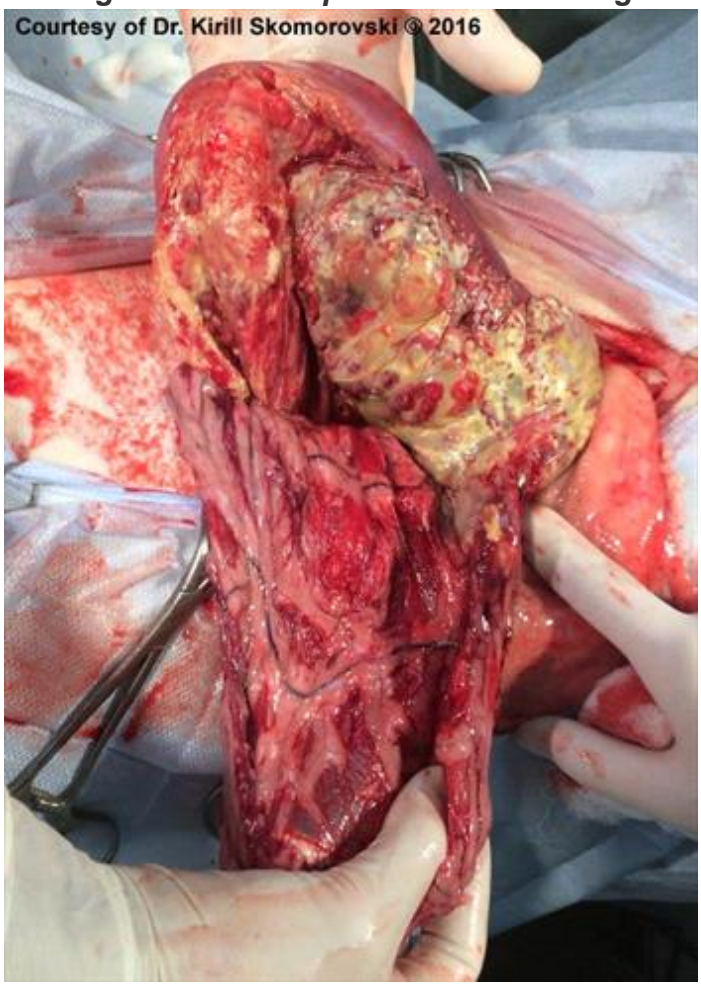
Figure 5. Ultrasonographic image of hyperechoic pancreas in a dog



Courtesy of Dr. Karri Beck © 2004

Thought to be related to prior pancreatitis episode. Click here to see board discussion

Figure 6. Severe pancreatitis in a dog



Courtesy of Dr. Kirill Skomorovski © 2016

click here to see board discussion

Treatment / Management

SPECIFIC THERAPY

No specific treatments exist for pancreatitis, and it is not well understood which supportive therapies are most effective for treating the disease.[6,9,33,34] Therapies aimed at inhibiting

pancreatic secretion (e.g. glucagon, somatostatin) and intracellular activation of proteases (e.g. gabexate mesylate) that have been beneficial in ameliorating the severity of experimental pancreatitis have shown little benefit in the treatment of patients with spontaneous pancreatitis.[9] Appropriate therapy of concurrent diseases is indicated.

SUPPORTIVE THERAPY

Goals of therapy are to maintain or restore adequate tissue perfusion; limit bacterial translocation; and minimize local and systemic effects of pancreatitis.[9] Supportive therapy varies depending on severity of disease. Some patients can be managed on an out-patient basis while others require hospitalization and intensive care.

Fluid Therapy

Fluid therapy is a critical component of treatment. Subcutaneous fluid therapy may be adequate for some patients, while others require IV therapy. For IV fluid therapy, lactated Ringers solution is appropriate and may be given at the volume needed to correct dehydration, supply maintenance needs (44-66 mL/kg/day), and replace losses from vomiting and diarrhea. Volume deficit can be replaced at an initial rate of 60-90 mL/kg/hr IV, then tailored to maintain tissue perfusion and hydration.[9,34] Dogs with signs of shock require higher fluid volumes.

Monitor serum albumin concentration during fluid therapy. If albumin concentration decreases significantly (e.g. <2.0 g/dL), plasma and/or hetastarch administration may be needed.[6,9,34] Fluid support is also important to prevent and treat disseminated intravascular coagulation (DIC).

Potassium supplementation may be necessary to replace losses in diarrhea, vomitus, and urine, and because of decreased food intake. Potassium supplementation is based on measurement of serum potassium levels. Glucose supplementation may also be needed in some patients.[9]

Fresh-frozen plasma transfusion (10-20 mL/kg IV) may be beneficial in animals have hypoalbuminemia, or evidence of a coagulopathy.[9] Plasma restores circulating protease inhibitors and replenishes antithrombin III, which is a treatment for DIC.[6,34] However, a retrospective study failed to demonstrate any benefit from plasma therapy in dogs with pancreatitis.[6] Monitor serum albumin concentration after administering plasma.[34]

Colloid therapy may be indicated for hypoproteinemia or shock. Dextran 70 (10-20 mL/kg/day IV) or hetastarch (10-20 mL/kg/day IV) given as constant rate infusions may also have antithrombotic effects that help maintain microcirculation.[9]

Antiemetic Therapy

Antiemetics are used if vomiting is severe. Parenteral options include the following:

1) Maropitant at 1 mg/kg SC q 24 hrs has become one of the preferred anti-emetics.[6,33] Maropitant may also have some analgesic effects.

2) Metoclopramide dosage is 0.2-0.4 mg/kg SC, IM q 8 hrs. Metoclopramide is contraindicated in cases with GI obstruction. This dopaminergic antagonist is a relatively weak antiemetic but does enhance motility in the upper GI tract.[33]

3) Dolasetron may be given at 0.3–1.0 mg/kg SC, IV q 24 hrs.[34]

4) Ondansetron dosage is 0.1-1.0 mg/kg IV q 12-24 hrs.[27]

5) Chlorpromazine may be given at 0.5 mg/kg SC, IM q 6-8 hrs. Do not administer until the dog has been rehydrated since the drug can cause vasodilation.

Antacid Therapy

Previously, therapy with H2 blockers was recommended to prevent gastric mucosal ulceration and injury; however, gastric ulceration is not typically noted except with severe pancreatitis. If used, proton pump inhibitors (e.g. pantoprazole, omeprazole) are superior to H2 blockers.[6]

Antibiotic Therapy

Prophylactic, broad-spectrum antibiotics (e.g. amoxicillin, enrofloxacin) may be warranted in patients with shock, fever, diabetes mellitus, or evidence of disruption of the GI protective barrier.[9] However, minimal evidence exists of bacterial infection playing a significant role in most cases of canine pancreatitis.[6]

Analgesic Therapy

Providing analgesia is an important aspect of caring for animals with pancreatitis.[6,9] Possible analgesic options include the following:

1) Buprenorphine may be given at 0.01-0.04mg/kg SC q 6-12hrs. Buprenorphine is a partial agonist, so may antagonize more potent analgesics if given concurrently.[9,33]

2) Oxymorphone can be administered at 0.05-0.1 mg/kg dogs IM, SC q 2-4 hrs or as a constant rate infusion at 0.005-0.01 mg/kg/hr IV.[33]

3) Transdermal fentanyl patch provides longer duration of analgesia but adequate fentanyl levels are not attained until 6-48 hrs after application.[9] See the dosage calculator in VIN's Veterinary Drug Handbook for further information.

Corticosteroid Therapy

Use of corticosteroids is controversial. They are typically reserve for patients with systemic inflammatory response syndrome.[6] In one study, dogs receiving 1 mg/kg/day prednisolone had significantly lower CRP levels on day 3 of hospitalization and earlier improvement of clinical signs.[35] Mortality rate one month after discharge was also lower in the prednisolone group (11.3%) versus the untreated group (46.1%).[35]

Nutritional Therapy

Oral intake has traditionally been withheld for the first 24-48 hrs because food stimulates the pancreas. However, timing of when to feed affected dogs is currently debated. Prolonged withholding of food and liquids can lead to hypoproteinemia; loss of intestinal motility; increased intestinal permeability; decreased intestinal blood flow; villous atrophy; and catabolism.[36] If antiemetics control vomiting, feeding small amounts may be considered as soon as possible either orally or via nasoesophageal feeding tube.[33] One study demonstrated that dogs fed within 48 hours of hospitalization had decreased time to return of voluntary food intake and to maximum food intake, as well as fewer GI signs.[37] Length of hospitalization was not impacted.[37] Administration of enteral nutrition is preferred to parenteral nutrition in patients with acute pancreatitis.[9]

The ideal diet for dogs with pancreatitis is unknown; however, feeding a highly-digestible, low-fat diet is usually the initial choice. A diet with ≤8% fat on a dry matter basis is commonly recommended. Diets designed for obesity management or fiber-responsive conditions are not as highly digestible and may not be suitable.[36] Once recovered, some patients require long-term administration of a highly-digestible, fat-restricted diet, especially those at risk of relapse or with hyperlipidemia. Other patients may be able to transition to a diet that has moderate fat content, i.e. up to 15% fat on dry matter basis.[36]

Other Therapies

Surgery is sometimes needed in patients with persistent biliary obstruction, as well as pancreatic necrosis, abscessation, or pseudocysts .[9]

MONITORING

Monitoring requirements vary depending on the severity of pancreatitis and the presence of other systemic abnormalities. Vital parameters, body weight, pain score, and fluid intake and output are evaluated multiple times daily in hospitalized patients. CBC, biochemistry panel with electrolytes, blood pressure, and coagulation status are repeatedly evaluated. Patients that are treated on an outpatient basis are monitored for improvement in clinical signs. Repeating cPLI and CRP tests may be done to determine if pancreatic inflammation is subsiding.[9]

PROGNOSIS

Patients with mild, acute pancreatitis have a good prognosis. Patients with severe, acute pancreatitis have a more guarded prognosis.[9] Patients with chronic pancreatitis may eventually develop exocrine pancreatic insufficiency.[38] In a study of 138 dogs with acute pancreatitis, 33% died within 30 days of diagnosis.[39] Bilirubin concentration ≥18.7 mg/L, elevated creatinine, hypocalcemia, metabolic acidosis, and AKI of IRIS grade 4 or 5 were associated with increased short-term mortality.[39] In a study of 50 dogs with acute pancreatitis, serum sodium <139 mmol/L was associated with a poor prognosis.[20] In another study, high ALT at the time of diagnosis was associated with longer hospitalization times, and decreasing CRP levels correlated with recovery.[63]

Special Considerations

Other Resources

Recent VIN Message Board discussions on acute pancreatitis
Recent VIN Message Board discussions on acute pancreatitis diagnosis
Recent VIN Message Board discussions on acute pancreatitis treatment
Recent VIN Message Board discussions on chronic pancreatitis
Recent VIN Message Board discussions on chronic pancreatitis diagnosis
Recent VIN Message Board discussions on chronic pancreatitis treatment

Proceedings articles that discuss acute pancreatitis
Proceedings articles that discuss chronic pancreatitis
Proceedings articles that discuss pancreatitis
2013 VIN/VECCS Rounds: Current Concepts in the Diagnosis and Treatment of Acute Pancreatitis
2004 VIN Rounds: Canine Pancreatitis

BestBETS for Vets article entitled Are Antibiotics Useful in Treating Acute Pancreatitis in Dogs?
Client Handout on pancreatitis in dogs

Pain Management for the Small Animal Practioner: Acute Pancreatitis
Small Animal Radiology & Ultrasonography: Pancreatic Masses
Small Animal Radiology & Ultrasonography: Ultrasonography of Pancreatic Abnormalities

Pathology Case 1
For more images see the Small Intestines/Pancreas Radiographs - Dog slideshow in the Image Library
For more images see the Pancreas Ultrasounds/CT - Dog slideshow in the Image Library

Differential Diagnosis

Acute kidney injury
Biliary tract obstruction
Cholangitis/Cholangiohepatitis
Diabetes mellitus
Enterocolitis, acute
Gastric ulceration
Gastric neoplasia
Gastritis, e.g. acute, chronic, hemorrhagic
Hepatobiliary neoplasia
Infectious enteritis, e.g. parvovirus, clostridial
Inflammatory bowel disease
Intestinal obstruction
Intestinal neoplasia
Intussusception
Pancreatic abscess
Pancreatic neoplasia
Peritonitis
Toxins causing vomiting

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