Pancreatitis: More Common and Sometimes More Severe Than You Think

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Background

Pancreatitis is a common condition in dogs and cats, but its diagnosis and management remain challenging. Acute pancreatitis (AP) is defined by the presence of neutrophilic inflammation within the body of the pancreas, and potentially extending to the peri-acinar fat, whereas the presence of fibrosis or acinar atrophy characterize chronic pancreatitis. Inflammation of pancreatic tissue leads to the passage of pancreatic enzymes into the peritoneal cavity and into the portal circulation potentially resulting in significant systemic complications such as ileus, hepatic necrosis, acute kidney injury (AKI), etc. Severity varies from mild to necrotic, often fatal, hemorrhagic pancreatitis. Although most often considered idiopathic, many etiologies or risk factors have been proposed, notably in dogs, such as hypertriglyceridemia, endocrine disease, adverse drug reaction (ex: steroids, furosemide, azathioprine, sulfas), prior surgery and dietary factors (fatty diet, dietary indiscretion). Although most often sterile (33/46), feline pancreatitis may be infectious as demonstrated using fluorescence in situ hybridization, particularly moderate to severe (11/31) compared to mild forms (2/15).

Diagnostic

Histopathology remains gold standard for diagnosis but is rarely performed due to its invasive nature and inherent limitations including missing localized lesions. Therefore, clinical signs combined with ultrasonographic findings and measurement of serum pancreatic lipase immunoreactivity (cPLI or fPLI) are routinely used. Mild to moderate pancreatitis is characterized by clinical signs such as anorexia, abdominal pain, vomiting and lethargy. In severe pancreatitis, acute pancreatic necrosis results in more severe clinical signs and multisystem complications such as systemic inflammatory response syndrome (SIRS), multiple organ dysfunction syndrome, or disseminated intravascular coagulation (DIC). Anorexia is commonly the only finding in cats. Improved technology and training have improved the diagnostic sensitivity of abdominal ultrasound (AUS), but it remains limited. A retrospective study in dogs concluded once more that AUS should not be used alone to diagnose pancreatitis and is a poor indicator of severity. AUS changes included pancreatic enlargement, echogenicity and altered mesenteric echogenicity with sensitivity and specificity of 89% and 43% (one criteria), and 43% and 92% (three criteria). Two-dimensional shear wave elastography may enhance diagnostic confidence as it was recently shown to be capable of assessing pancreatic stiffness in canine AP and correlated moderately to Spec cPL. Advanced imaging such as endoscopic ultrasound, CT or MRI have yet been established as a routine diagnostic tool. Assays that detect lipase specific to the pancreas have become widely available and are considered the clinicopathological tests of choice for the diagnosis of both canine and feline pancreatitis. Cage-side semi-quantitative tests perform reasonably well. The sensitivity and specificity of qualitative tests in dogs ranges from 74-100% and 64-83%, respectively,

with a better performance as the severity of pancreatitis increase. Given the limitations of the assays, it is crucial to interpret the results based on clinical signs and the presence of concomitant diseases. In both dogs and cats, increased DGGR lipase has been shown to correlate closely with increased pancreatic specific lipase (Spec PLI) which may be a useful economic tool for diagnosis and follow-up. To allow early prediction of short-term death, a Canine Acute Pancreatitis Severity (CAPS) score was proposed based on identified independent risk factors: presence of SIRS, coagulation disorders, increased creatinine, and ionized hypocalcemia.

Management

Early targeted fluid therapy, aiming at correction of dehydration, acid-base and electronic imbalances, is critical in severe AP. Crystalloids (Lactated Ringer preferred) are the first line fluids used, with colloids reserved for patients with proven low oncotic pressure. Unless DIC is present, there is no benefit in using fresh frozen plasma in terms of alpha-macroglobulin intake. Multi-modal analgesia, such as combination of opioids with lidocaine +/- ketamine, is often required. In human medicine, thoracic epidural may have prognostically beneficial effects due to suspected anti-inflammatory effects and increased splanchnic perfusion. Interestingly, early refeeding is part of the strategies to reduce pain. Early interventional feeding is now advocated in AP and is usually well tolerated. Antibiotic administration remains controversial but may be considered in patients with high risk of bacterial translocation or in severe feline pancreatitis. Corticosteroids are typically reserved for patients non-responsive to fluid resuscitation, although a recent study has suggested their use in the initial treatment of canine AP, resulting in earlier improvement of clinical signs.

Complications

Systemic complications such as AKI, acute respiratory distress syndrome, diabetes ketoacidosis or cardiac arrhythmias are treated on an individual basis. The coagulation status should be carefully monitored, and antithrombotic therapy initiated in most severe cases to avoid thrombotic complications. In case of local complications, such as acute fluid collections or extra-hepatic bile duct obstruction, ultrasound-guided intervention and/or biliary stent placement may be warranted. Surgical interventions result in high mortality rate and are usually reserved for infected pancreas (ex: abscess) or gallbladder rupture.

References available upon request.