



## Feline pancreatitis – an update

By Karen Bailey

Historically pancreatitis has been considered rare in cats, but more recent investigations suggest it is in fact quite common.

**I**n a UC Davis study (De Cock et al., 2007), about 50% of cats had histopathological evidence of chronic pancreatitis, and nearly 10% had evidence of acute pancreatitis at necropsy. Unexpectedly, evidence of pancreatitis was found in 45% of apparently healthy cats, suggesting either a population of cats with subclinical disease or, possibly, overdiagnosis using the current histopathological definition.

Increased clinical awareness and access to testing have been associated with more frequent diagnoses in recent times. The American College of Veterinary Internal Medicine recently published a consensus statement on this condition (Forman et al., 2021), which was written by eight authors, including clinical and anatomical pathologists, radiologists and internists, who referenced more than 200 papers. There was relatively little research on the management of pancreatitis in cats and only a small amount on aetiology and pathogenesis, but slightly more on diagnosis.

### Classifying pancreatitis in cats

The classification of pancreatitis in cats lacks standardisation. Generally, acute pancreatitis is reversible and chronic disease is characterised by irreversible changes in the pancreas. However, this difference is histologic, and clinically it may not be possible to distinguish between acute pancreatitis and a flare-up of chronic pancreatitis.

Both the acute and the chronic diseases can be mild to severe, although acute cases are more commonly severe and chronic cases mild. There is no age, breed or sex predisposition. Associations with body condition, dietary indiscretion or drug history have not been established in cats. While a variety of infections, including parasitic (eg, toxoplasmosis) and viral (eg, coronavirus, herpesvirus) infections, have been associated with pancreatitis, infection is a rare cause in cats. Pancreatic trauma (eg, through road accidents) is an established cause but, importantly, no increased risk has been noted in pancreatic sampling studies.

Neoplasia in the pancreas increases risk but is uncommon in cats. Favourable responses to immunosuppressive therapy in chronic pancreatitis suggest immune-mediated aetiology in some cats. Diabetes mellitus, enteropathy, hepatic lipidosis, cholangitis, nephritis and immune-mediated haemolytic anaemia have also been associated with pancreatitis, but more than 95% of the cases of pancreatitis in cats are idiopathic with no specific cause able to be determined.

A premature activation of pancreatic digestive enzymes leading to pancreatic autodigestion is assumed to be important in the pathogenesis of pancreatitis. Trypsinogen activation is the initiating event for acute pancreatitis, but there is no consensus on how it unfolds. Damage to ductal epithelium appears essential for the development

hyperechoic surrounding mesentery and focal effusion. The reported sensitivity of ultrasonography is 11–67%, but is very operator dependent.

Features of chronic pancreatitis are not well established and ultrasound is poor for assessing chronicity. Nodular hyperplasia can be seen incidentally in older cats and there is an overlap between benign and malignant nodules and pancreatitis, with histology required for differentiation.

Cytology is not often performed on cats with suspected pancreatitis, but samples can be collected safely using ultrasound-guided aspiration with a 20- to 22-gauge needle. Pancreatic cells deteriorate rapidly, so smears should be made gently and quickly.

One study found that about a third (24/73) of samples were non-diagnostic (Crain et al., 2014). In acute pancreatitis, aspirates are often highly cellular with neutrophils predominant. Macrophages, debris and crystalline material may be seen.

Acinar cells may be dysplastic, and distinguishing between pancreatitis with dysplastic epithelial cells and inflamed pancreatic carcinoma is difficult. However, pancreatic tumours are rare, so pancreatitis is generally more likely. In chronic pancreatitis, aspirates are often poorly cellular because of fibrosis.

In both acute and chronic disease, the absence of inflammatory cells in aspirates does not rule out pancreatitis because inflammation can be localised. In acute pancreatitis, fluid may accumulate adjacent to the pancreas and is typically a high-protein transudate or exudate with blood, mineral, neutrophils, macrophages that may contain lipid vacuoles or haemosiderin, and free lipid.

Routine haematology (CBC) and biochemistry, while not specific for pancreatitis, are helpful for screening for other diseases. Findings in pancreatitis cases may include:

- haemoconcentration
- inflammatory leukogram
- increased clotting times (in severe cases)
- hyperbilirubinaemia
- increased liver enzymes
- sometimes azotaemia.

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of chronic pancreatitis in cats. Inflammation in neighbouring organs (eg, intestines, biliary system) may cause pancreatic inflammation, and the shared entry of the common bile duct and pancreatic duct into the duodenum may explain the association between cholangitis or cholecystitis and pancreatitis in cats.

#### **Clinical signs in cats with pancreatitis**

The clinical signs in cats with pancreatitis include lethargy (in 50–100% of cases), anorexia (62–97%), vomiting (35–52%), weight loss (30–47%) and diarrhoea (11–38%). Physical findings may include dehydration (37–92%), hypothermia (39–68%), icterus (six to seven percent), abdominal pain (10–30%), fever (7–26%) and abdominal mass/organomegaly (4–23%).

#### **Diagnosis is challenging**

Radiography is neither sensitive nor specific, but in cases of severe pancreatitis there may be loss of peritoneal detail or a mass effect in the cranial abdomen. Ultrasonography should be routine in cats suspected to have pancreatitis, both to assess the pancreas and to look for other conditions. Findings in acute cases may include pancreatic enlargement,

Hyperglycaemia is sometimes seen, and has been associated with poor outcomes. In cats, unlike other species, hypertriglyceridaemia is rare. Potassium, chloride, sodium and calcium may be reduced.

Amylase is not useful for the diagnosis of pancreatitis in cats. Similarly, feline trypsin-like immunoreactivity, which is not routinely available in New Zealand, has limited usefulness due to poor specificity (increases are seen not only in pancreatitis but also in chronic enteropathy and intestinal lymphoma).

An ideal marker for pancreatitis would be synthesised only by pancreatic acinar cells and not cleared rapidly from blood. Although pancreatic lipase fulfils this, making an assay specific for this is problematic. Many lipases exist in the body, and despite great effort no assay has been shown to be completely specific for pancreatic lipase, with few studies on the use of serum lipase in cats.

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Another option is to measure feline pancreatic lipase immunoreactivity (fPLI). Several commercial assays for this are available, including the Spec fPL test. There is less information on pancreatic lipase immunoreactivity in cats than there is in dogs, but data suggests it is highly specific for pancreatic lipase and quite sensitive for diagnosis. However, sensitivity increases with severity of disease, with a positive predictive value of 90% and a negative predictive value of 76%, so positive results indicate probable pancreatitis but negative results do not rule it out. The patient-side SNAP fPL correlates with the Spec fPL, with 'normal' samples suggesting pancreatitis is unlikely and 'abnormal' samples usually having positive or equivocal results in the Spec fPL (see further reading).

The 1,2-o-dilauryl-rac-glycero-3-glutaric acid-(6-methylresorufin) ester (DGGR) lipase assay used at Gribbles Veterinary laboratories has substantial agreement (Cohen's kappa coefficient of approximately 0.7) with the Spec fPL, so may be useful as a screening test (see further reading).

Agreement is poor between ultrasound diagnosis and Spec fPL or DGGR (kappa 0.26 and 0.22) and also between histological diagnosis and Spec fPL or DGGR (kappa 0.06 and 0.1) (see further reading). However, this study questioned whether mild inflammatory cell infiltrate in the pancreas may be normal, reiterating concern that current histological criteria may be overdiagnosing pancreatitis.

A discussion of pathology is beyond the scope of this article, but it is important to note that lesions may be multifocal, so multiple biopsies are recommended and pancreatitis should not be excluded based on negative biopsy results alone. Although gross lesions may not be apparent at laparotomy, any findings should be communicated to the pathologist. These may include pancreatic haemorrhage, congestion, oedema, fibrin strands, peripancreatic fat necrosis and focal peritonitis.

### **Clinical management**


The clinical management of acute pancreatitis is predominantly supportive. The inciting cause should be addressed if known, but this is rarely possible as most cases are idiopathic.

Treatment may involve fluid therapy, pain management, nausea and vomiting control, and nutritional support. There is little information on appropriate nutritional management, but extrapolation from other species supports the use of enteral nutrition (via stomach tube if necessary) where possible. Although parenteral nutrition can be used when enteral feeding is impossible due to unmanageable vomiting, survival rates are lower. Withholding food can be detrimental and is not recommended.

Acute pancreatitis in cats is usually sterile, so antimicrobial treatment is not generally recommended. Bacterial complications such as pancreatic abscess are rare, and antibiotics should only be used when clinical indications are compelling. Although there have been recent reports of benefits from corticosteroid use in humans and dogs (Okanishi et al., 2019), there is no current information to support its use in cats. There is little research on managing chronic pancreatitis in cats, but it generally involves analgesia, nutritional support, appetite stimulation, and nausea and vomiting control.

In conclusion, although acute and especially chronic pancreatitis are likely common in cats, diagnosis remains challenging and requires the integration of history, clinical findings, imaging, laboratory data

(which may include DGGR lipase or fPLI testing) and potentially cytology and histology.

Management involves the treatment of potential causes (although most cases are idiopathic) or concurrent conditions, analgesics, antiemetics and, especially in acute cases, fluid therapy and nutritional support. Further studies on cats specifically would be welcomed. 

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