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Hot Topics in Feline Medicine Online 'Mini Series'

Session 1: Yellow Cats
Liver Disease & Pancreatitis

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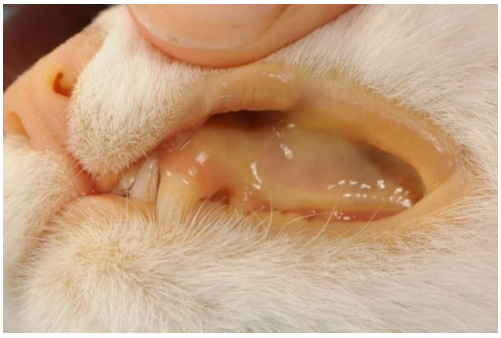


Hot Topics in Feline Medicine Part 1

Yellow cats – liver disease and pancreatitis, unravelling the complex cases

Feline liver disease in practice

Frustratingly, vague signs such as lethargy and anorexia are very common presenting signs in feline practice and a wide number of differential diagnoses therefore need to be considered for such cases. **Pancreatic, hepatic and biliary diseases are relatively common causes of these vague signs**, although may also result in additional clinical signs such as vomiting or jaundice, that helps to increase our index of suspicion for this group of disorders. In most cases cats with disease in this area will present with non-specific clinical signs.

Cats suffer from species specific forms of liver disease, i.e. they are not the same as dogs in this respect. Their liver disease can fall into the following categories:

- Hepatic lipidosis (according to studies this is the most common in USA and UK, but I think in UK we less this less frequently)
- Inflammatory hepatopathies: feline cholangitis (I feel the most common in UK)
- Infectious hepatopathies (e.g. FIP, liver fluke (rare in UK), toxoplasmosis)
- Neoplasia (lymphoma most common)
- Other (e.g. hepatic cysts, amyloidosis, portosystemic shunt)

Background –why do cats suffer from hepatobiliary/GI/pancreatic disease?

Hepatobiliary diseases and pancreatitis often occur together in cats, often also in combination with inflammatory bowel disease (sometimes termed **'triaditis'**), which can complicate the diagnosis and management. (Careful if reading any human literature- triaditis is a term used for a specific liver disease rather than the same type of condition). A recent necropsy study showed when looking at cats with cholangitis 50% had IBD, 60% had pancreatitis and 30% had both (plus cholangitis). (paper: Feline cholangitis: a necropsy study of 44 cats (1986-2008), Callahan Clark et al, JFMS 2011 13: 570-578).

The reason for the close association with these other diseases is predominantly thought to be a result of the close anatomical and functional relationship between the major pancreatic duct and common bile duct.

- One of the major differences in the feline pancreas is that the majority of cats have only **one pancreatic duct** which, in contrast to dogs, enters the intestine at the major duodenal papilla together with the common bile duct. This close association makes cats with biliary disease very susceptible to the development of pancreatitis, since any inflammation/blockage of the distal common bile duct may result in reflux of pancreatic secretions up the pancreatic duct.

- Other reasons for the association include the fact that vomiting increases the likelihood of pancreaticobiliary reflux, and in comparison to dogs, cats have a much **higher concentration of intestinal microflora** so when pancreaticobiliary reflux occurs a mixed population of bacteria, bile salts and activated pancreatic enzyme enters the pancreatic and biliary ducts.

Below is an overview of the investigation of liver disease but with each specific disease description later is more information.

Laboratory diagnosis of liver disease in cats

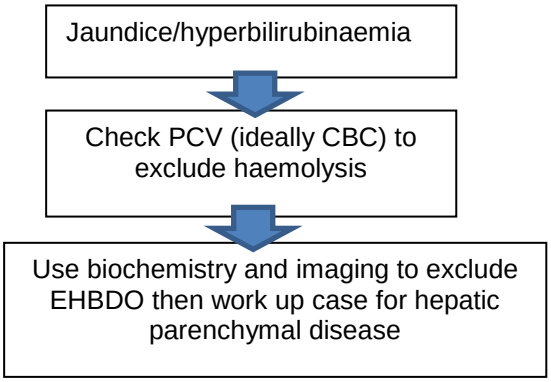
The CBC/haematology is generally not too helpful and maybe normal but you might see a few pointers towards a liver problem:

- Anaemia of chronic disease – normocytic and normochromic and mild
- Microcytic anaemia – this might occur with concurrent GI disease and chronic GI haemorrhage
- Poikilocytosis – changes in membrane lipids cause this abnormality seen in liver disease
- Remember in icteric cats a CBC is important to exclude haemolysis as a cause of the icterus
- You might see an inflammatory leukogram if neutrophilic cholangitis and you can get a neutropenia if septic.
- Think about coagulation times – especially if considering biopsy – coagulopathy occurs due to vitamin K deficiency (malabsorption of vitamin for example) and failure of production of clotting factors. APTT and PT may be elevated but probably all cats with liver disease having a biopsy should be pre-treated with vitamin K.

Biochemistry is going to be abnormal in cats with hepatic disease and clearly abnormal liver enzymes will occur. Remember that the elevations and combinations thereof are unlikely to provide much help in deciding the nature of the disease. Think about the following:

- ALT and AST are hepatocellular enzymes indicating leakage of enzymes from cells but increases also occur with extra-hepatic disease (e.g. hyperthyroidism, sepsis, endocrine disease) so they are not specific for liver disease.
- ALP and GGT are hepatobiliary enzymes so more specific for disease in this area. You don't get the drug induction like in dogs Increases in ALP and GGT are seen in pancreatitis, liver disease and EHBDO.

- Hepatic lipidosis tends to give large increases in ALP but normal GGT whereas pancreatitis and triaditis tends to give similar fold increases in both.
- Hypoalbuminaemia may occur in severe liver disease and/or IBD with malabsorption. To get hypoalbuminaemia from liver disease alone means the liver is severely compromised and clinical signs plus other abnormalities such as hyperbilirubinaemia are likely.
- Hyperbilirubinaemia and clinical jaundice should prompt the following:



- Bile acid stimulation tests are useful to identify reduced hepatic function. They will be normal until a critical loss of function is reached or if the cat has a portosystemic shunt. Abnormal bile acids should prompt work up and treatment for liver failure. There is no point doing a BAST in a jaundiced cat (if jaundice is localised to hepatic) as it will not give additional info.

Urinalysis may help too in the work-up or as a cheap screening test for older/unwell cats:

- All cats with USG< 1.035 should be looked at more closely (not for liver disease necessarily but chronic kidney disease)
- Bilirubin in urine in cats is always abnormal – it needs following up
- Ammonium biurate is always abnormal and suggests a PSS or end-stage liver disease (plus the affected cat may be encephalopathic)

Hepatobiliary imaging

Although radiography will identify large masses, hepatomegaly and other such grossly obvious abnormalities, it will not show detail of the biliary tree, identify parenchymal changes etc and so is not very useful in the investigation of hepatic disease (remember met checks though if neoplasia is suspected). Therefore ultrasound is the much more useful modality. Obviously the info you get depends on your skill and experience but even with a basic machine the following may be appreciate:

- A dilated, tortuous bile duct suggestive of EHBDO – this looks like sausages within the liver and is relatively easy to exclude in cases of jaundice (see case presented in talk)
- A big, bright liver may suggest hepatic lipidosis (but a lymphoma liver will look similar) – histo still needed, inflammatory cholangitis may give a more hypoechoic parenchyma
- A thickened gall bladder wall and bile duct wall may be seen in inflammatory disease (see below)
- **Remember ultrasound is not perfect and is subjective so you will still need a biopsy for a definitive diagnosis**

To biopsy or not to biopsy that is the question

Ideally you need liver tissue to give you a diagnosis but it is not that simple of course! There are risks that need to be considered and of course costs. Think about the following and in the talk we will discuss pros and cons much more:

- FNAs are useful because if lymphoma is a differential it will usually exfoliate and give you a diagnosis (and other round cell tumours e.g. mast cell tumours)
- Similarly hepatic lipidosis is ideally diagnosed with histo but an FNA may be sufficient but remember that the disease may not be uniformly spread out so a lymphoma or inflammatory disease may be missed if concurrent HL is present.
- A risk to benefit assessment should be discussed with owners
- Inflammatory hepatopathies can't be diagnosed on FNA
- Tru-cut biopsies can be useful if obtained by experienced imagers. There is a risk of death from bleeding or a vasovagal syndrome reported when using 'trigger' type biopsy needles. So manual needles are recommended. Coags should be checked before the procedure.
- Gall bladder aspiration for cytology and culture is a relatively low risk procedure if performed correctly
- Laparoscopy is not often performed in the UK but is another option
- Laparotomy allows control of bleeding, larger samples, accurate sampling of abnormal looking areas and if indicated biopsy of other organs (e.g. pancreas, GIT) but clearly is more invasive.
- **ALWAYS save a small sample of liver and some bile for culture NOT JUST HISTO!**

When considering biopsy technique:

- Think likely disease based on signalment, clinical signs, bloods and imaging
- Then consider if an FNA will be helpful
- If inflammatory liver disease is suspected then consider tru-cut or laparotomy after measuring coag times
- Think if other organ biopsy is indicated
- Think owner wishes and finances

Inflammatory liver disease

Inflammatory liver diseases are the most common group of hepatic disorders encountered in cats in the UK. Inflammatory liver diseases in cats can be particularly confusing because of the classification and terminology used. Recently the WSAVA standardised the classification of liver disease in cats and your pathologist should use this system (or find a new pathologist).

Classification has been based predominantly on histopathological features including the nature of the inflammatory infiltrate (neutrophilic vs lymphocytic) and the presence of bile duct proliferation and fibrosis, and in the past a variety of terminology has been used to describe different forms of inflammatory disease.

The most useful and practical way of dividing up feline inflammatory liver diseases is whether they are predominantly **neutrophilic** infiltration, or **lymphocytic** infiltration. The differences in treatment strategies for neutrophilic and lymphocytic inflammation warrant establishing a definitive diagnosis.

Neutrophilic cholangitis

Neutrophilic (previously often termed suppurative) cholangitis and hepatitis is often associated with pancreatitis and IBD (triaditis), although usually the clinical signs associated with the cholangitis/hepatitis predominate. This form of liver disease tends to occur more commonly in **middle-aged to older cats**, although can occur at any age, particularly when there are other predisposing factors.

Common clinical signs/presentation include:

- Acute presentation (rather than prolonged history of illness)
- Jaundice
- Pyrexia
- Anorexia
- Abdominal pain

The most common laboratory abnormalities are elevations in ALT, ALP, GGT, bilirubin and a neutrophilia often with a left shift. Cholelithiasis may occasionally occur and it is unclear whether this is a causative factor, or a consequence of the neutrophilic cholangitis.

Ultrasonography may reveal thickening of the gall bladder wall **(>1 mm)**, distension of the bile duct **(> 5 mm)** and the presence of sludge or inspissated bile within the gall bladder, and there may be a patchy echogenicity to the liver.

Histopathology is necessary for confirming the diagnosis, and samples of liver tissue should also be submitted for bacterial culture and sensitivity. Coagulation times should be assessed prior to taking

biopsies and vitamin K treatment administered if coagulation times are prolonged. If biopsies are obtained at laparotomy, the patency of the bile ducts should also be evaluated, and bile aspirated for cytology and culture. Laparotomy or laparoscopy also allows collection of pancreatic and intestinal biopsies to assess for concurrent disease.

Treatment of neutrophilic cholangitis:

Antibiotic therapy is the most important treatment for neutrophilic cholangitis. E.coli, and mixed growth including anaerobes is most frequently found, reflecting ascending infection from the intestine.
The author usually initiates **amoxycillin-clavulanate** or **cephalexin** whilst pending culture and sensitivity results, and then adjusts treatment as appropriate. Antibiotic treatment should be maintained for at least **4-6 weeks** to reduce the risk of recurrence. Supportive treatment such as intravenous fluid therapy and nutritional support are also usually required:
Ursodeoxycholic acid is a choleretic that may have anti-inflammatory, immunomodulatory and antifibrotic properties in addition to increasing the fluidity of biliary and promoting bile flow. Although there is no evidence that it is of benefit, it has been widely used and so is indicated as an additional treatment in inflammatory liver disease, provided there is no evidence of extra-hepatic biliary obstruction. The recommended dose is 10-15mg PO once daily.

S-adenosyl-L-methionine (SAMe) is a nutraceutical agent believed to restore glutathione levels that are reduced in liver disease resulting in oxidative damage. No clinical trials have been performed to confirm potential benefit, but it may be a useful supplement for any cat with liver disease.

Lymphocytic cholangitis

Lymphocytic cholangitis usually occurs in younger cats with Persians overrepresented. The aetiology of the disease is unknown, but immune-mediated mechanisms may be involved.
Common clinical signs/presentation include:

- In contrast to suppurative inflammatory disease, these cats are often bright, appetite is usually maintained and pyrexia is unusual.
- Significant weight loss may occur in some but not all cases.
- Some cats may actually be polyphagic.
- The most striking clinical finding is jaundice, sometimes accompanied by ascites.
- Hepatomegaly and mild generalised lymphadenopathy is also often present.
- Laboratory findings are similar to those found with suppurative disease, the main difference being that hyperglobulinaemia is frequently present, neutrophilia is less common and a left-shift would not be expected. If ascitic fluid is present, it is usually very proteinaceous with a low mixed inflammatory cellularity. FIP and lymphoma are important differential diagnoses.

Treatment of lymphocytic cholangitis:

Corticosteroids are the mainstay of treatment for lymphocytic cholangitis. Prednisolone is usually used at a dose of **1-2mg/kg twice daily**, with the dose gradually reduced over 6 –12 weeks if good response is seen. Additional treatments that may be beneficial include choleretics, nutritional support and SAMe as described above. Colchicine (0.03mg/kg once daily) may be of benefit in an attempt to reduce fibrosis, although there is no evidence for this.

Mixed inflammatory hepatopathies

Occasionally, a mixed inflammatory infiltrate may be observed on histopathology of liver biopsies. These cats may have a history of an initial acute illness, followed by gradual loss of condition, inappetance and lethargy. The dilemma with these cases is whether it is antibiotic or corticosteroid treatment that is required. The author usually initiates antibiotic treatment initially whilst bacterial culture of liver tissue is pending. If bacterial cultures are positive, a 4-6 week course of antibiotics is administered, but if there is not complete clinical improvement after 1-2 weeks, an anti-inflammatory dose of prednisolone (0.5-1mg/kg daily) is also initiated. If cultures are negative, anti-inflammatory doses of prednisolone are initiated earlier, and antibiotics continued for 2-3 weeks.

Cases with no histopathology:

- Consider clinical signs, lab results and severity of illness
- Start with broad spectrum antibiotics for 1-2 weeks (amoxyclav plus metronidazole)
- If no improvement add 0.5mg/kg pred SID/BID
- Address other issues as a priority (pain, nausea, hypokalaemia)

'Triaditis'

Neutrophilc cholangitis and pancreatitis often occur together in cats, often also in combination with inflammatory bowel disease (IBD) ('triaditis'), which can complicate the diagnosis and management.

When managing triaditis other factors should be considered:

- Have you got a diagnosis? What is PLI and have you biopsied the gut?
- How will you manage the combination of conditions? Think diet for IBD, antibiosis for neutrophilic cholangitis and analgesia, fluid therapy (see later) for pancreatitis
- Think about ongoing management after acute disease treatment i.e. diet for IBD

- Don’t rush into prednisolone as this could be the wrong decision if the cat has neutrophilic cholangitis.

Hepatic lipidosis

Hepatic lipidosis is a syndrome characterised by severe hepatocellular lipid accumulation, intrahepatic cholestasis and impaired liver function. A period of anorexia leads to severe protein restriction, mobilization of adipose tissue to the liver, and reduction in synthesis of proteins required for VLDL formation, which leaves the liver unable to remove excess triglycerides. Precise pathophysiology of HL is unknown but proposed mechanisms include metabolic changes associated with starvation and obesity, androgenic release during illness or stress, protein and nutrient such as taurine deficiency, relative carnitine deficiency or insulin resistance. The balance of triglyceride lipolysis and accumulation is modulated by blood glucose concentrations, hormonal, neural and pharmacologic mechanisms. The activity of hormone sensitive lipase (promoting lipolysis) and lipoprotein lipase (promoting fat uptake) directly regulates fat metabolism. Noradrenaline, adrenaline, growth hormone, glucagon, corticosteroids and thyroxine all increase hormone sensitive lipase, whereas insulin inhibits it. Since cats readily release cathecolamines when stressed, stress may therefore exacerbate hormone sensitive lipase activity, and as a result promote lipolysis. Furthermore, in starvation hormone sensitive lipase increases and lipoprotein lipase decreases, favouring hepatocellular fat accumulation.

Hepatic lipidosis may occur as a primary event, or may be secondary to another disease process. Underlying conditions such as cholangiohepatitis, neoplasia, gastrointestinal disease, pancreatitis or endocrine disorders are identified in **over 50% of cats**. **(Note: always look for the underlying disorder).**

In comparison to cats with secondary HL, those with primary idiopathic HL are usually younger, have higher ALP/bilirubin levels, absence of hyperglobulinaemia, normal GGT and a better survival rate. An initiating acute pancreatitis is common, and if present carries a worse prognosis.

Clinical signs are often initially vague consisting of progressive lethargy and anorexia, sometimes accompanied by intermittent vomiting. As the disease progresses, icterus develops, and signs of encephalopathy such as depression and ptyalism can occur.

Diagnosis is based initially on clinical history, physical examination, laboratory findings, ultrasonography and cytology of aspirates. Definitive diagnosis should be made by histopathology of liver biopsies once the cat is stabilised. The diagnosis is made when more than 50% of hepatocytes in an acinus have vacuolar lipid accumulation, and there is no evidence of additional liver pathology, such as inflammatory changes.

The most common laboratory findings include hyperbilirubinaemia and elevations in ALT and ALP (often more than 5-fold) without significant elevations in GGT. In comparison, cats with inflammatory

liver diseases would be expected to have **elevations in GGT** and serum **globulins**. Absence of jaundice does not exclude lipidosis since liver enzyme elevations and histological evidence of hepatic lipidosis occur before cholestasis. Bilirubinuria also precedes increases in serum bilirubin and may be a useful marker of early cholestasis since the presence of any bilirubin in the urine is always an abnormal finding in cats.

Note that bilirubinuria in cats is always abnormal and should be investigated (cf dogs where it can be a normal finding).

Cats with lipidosis are also often hypokalaemic and hyperglycaemia. Haematology is often unremarkable, although poikilocytosis may be evident. Haematology should however be monitored during treatment, in addition to serum biochemistry, as hypophosphataemia can occur with re-feeding, which may result in haemolytic anaemia. A mild to moderate non-regenerative anaemia of chronic disease can also develop.

Coagulopathies are common in cats with a variety of hepatic disorders, including lipidosis. This most commonly results from vitamin K deficiency, but may also be caused by reduced production of clotting factors by the liver, or less commonly a consumptive coagulopathy. Vitamin K deficiency occurs secondary to anorexia and malabsorption resulting from cholestasis, with subsequent lack of intestinal bile salts to aid in absorption of fat and fat soluble vitamins.

Diagnostic imaging often reveals hepatomegaly and diffuse hyperechogenicity. This is not a sensitive or specific finding for lipidosis however, since clinically normal obese cats and those with hepatic lymphoma may also have hyperechoic liver parenchyma. Ultrasonography helps to exclude extra-hepatic biliary obstruction and other processes that may be involved with secondary hepatic lipidosis, such as pancreatitis. It can also be used to guide fine needle aspirates and/or tru-cut biopsies.

Since these cats are often very sick, a period of stabilisation is usually required before it is safe to anaesthetise them for liver biopsies. Fine needle aspirates are very useful to perform initially, to obtain a tentative diagnosis when initiating appropriate treatment. Typically cytological evaluation of fine needle aspirates are not very useful in the diagnosis of liver disease. Hepatic lipidosis is one exception to that where extensive vacuolization of hepatocytes consistent with fatty infiltration can usually be appreciated. Biopsies should however still be taken once the animal is stable enough, since aspirates will not exclude underlying concurrent disorders such as cholangiohepatitis. Pre-treatment with subcutaneous vitamin K1, or demonstration of normal coagulation times is advised prior to obtaining biopsies.

Generally liver FNAs are not useful in the diagnosis of inflammatory liver disease BUT they can provide a diagnosis of lipidosis and lymphoma.

Cats with hepatic lipidosis require aggressive nutritional support as soon as possible so this should be initiated immediately if hepatic lipidosis is suspected based on the history and clinical findings. Most cats will require enteral assisted feeding for at least a few weeks, often longer, and therefore oesophagostomy or gastrostomy tubes are the method of choice. Naso-oesophageal tube feeding can be useful for the first few days, until the cat is stable enough to undergo anaesthesia for the placement of a longer term feeding tube. The author prefers to endoscopically place a gastrostomy tube, and obtain endoscopic gastrointestinal biopsies at the same time.

In contrast to dietary management of other liver diseases, a high protein diet is required to treat hepatic lipidosis, unless the cat is initially showing signs of hepatic encephalopathy. Cats with lipidosis should be feed 60-80kcal/kg/d.

Additional treatment with L- Carnitine (250mg/d; for potential effects in increasing mitochondrial fatty acid oxidation), taurine (250mg/d; to prevent deficiency) and vitamin E (50mg/d; as an antioxidant) once daily and s/c vitamin B_1 (100mg), vitamin K_1 (2mg; to prevent vitamin K deficent coagulopathies) twice daily for 3 days and vitamin B_{12} (250µg twice weekly) are also recommended, since early provision of these is thought to improve clinical outcome.

Other treatments that are often required include antiemetics and promotility drug. Gastric stasis is common and aggressive treatment may be required with constant rate metoclopramide infusions (1-2mg/kg/day) and/or pro-kinetics such as cisapride or tegaserod.

Careful monitoring and frequent reassessment of haematological and biochemical parameters is essential to ensure improvement is occurring, and to monitor for complications such as hepatic encephalopathy, hypokalaemia, hypophosphataemia and subsequent haemolytic anaemia. Prompt treatment is required should any of these complications occur. Additional treatment may also be required for any underlying disease if another disease process is identified following initial stabilisation.

Does this cat have pancreatitis & if so how do I treat it?

Pancreatitis is becoming increasingly recognised as a clinical entity in cats. It has always been notoriously difficult to diagnose but more recently diagnosis has been aided by newer, more sensitive diagnostic tools. **We are probably underdiagnosing this condition.**

The most common clinical findings are ***lethargy, anorexia and dehydration***. Chronic pancreatitis (CP) is more common in cats than the acute form (acute necrotizing pancreatitis, ANP) and is often associated with IBD (70% cats with IBD had elevated PLI in one study) and/or cholangitis/hepatitis (often termed 'triaditis'), so clinical signs associated with these conditions may predominate. CP often involves lymphocytic infiltration of the pancreas.

Cats are NOT small dogs!
Pancreatitis in cats doesn't seem to behave in the same way as canine pancreatitis:

- Chronic pancreatitis is more common
- It is often associated with IBD (+/- cholangitis)
- Never starve a cat (actually recent thinking suggests canine pancreatitis patients shouldn't be starved)
- A low-fat diet just doesn't suit cats; they are not designed for it! A diet to manage the concurrent IBD is a better idea.
- Diabetes is a co-morbidity to be assessed and watched out for (diabetic cats have significantly higher PLI than non-diabetic cats)

Diagnosis of pancreatitis in cats can be challenging, in view of non-specific clinical signs, frequent presence of concurrent disease, and limitations in diagnostic testing. However, there are some recent advances which can help the clinician reach a diagnosis of pancreatitis with more certainty.
Liver enzymes are likely to be elevated secondary to hepatic lipidosis, necrosis, or concurrent hepatobiliary disease. Hyperbilirubinaemia is relatively common (although less so than in dogs), and can reflect bile duct obstruction by the inflamed pancreas or be a consequence of concurrent hepatic dysfunction. Electrolyte abnormalities are common, especially hypocalcaemia, which is reported in 30-50% of cats with pancreatitis. Hypocalcaemia may be a result of saponification of fat and soft tissues, changes in acid-base balance, resistance to or decreased production of parathyroid hormone, or increased calcitonin concentrations. Hypocalcaemia has been associated with a poorer prognosis. Hypokalaemia is also common secondary to inappetance.

Amylase and lipase are not considered to be useful in the diagnosis of pancreatitis in cats. They are usually within the reference range in cats with pancreatitis, and in experimental models of feline pancreatitis amylase has been shown to drop to 60-80% below baseline concentrations. Indeed, marked elevations in amylase and lipase are more likely to reflect reduced glomerular filtration than pancreatitis, and are also influenced by glucocorticoids, gastrointestinal disease and peritonitis. Anecdotally, comparison of amylase and lipase in abdominal effusions compared to serum might be of some diagnostic use.

In the last few years, more specific tests for feline pancreatic disease have been developed.
The most useful of these is feline **pancreatic lipase immunoreactivity (fPLI).** It is currently the most sensitive and specific test available for the diagnosis of feline pancreatitis, particularly in cats with moderate-severe disease. Although considered a highly sensitive test, a normal fPLI does not completely exclude pancreatitis as a diagnosis. Until the fPLI test was developed, feline trypsin-like immunoreactivity (fTLI) had been suggested as a useful diagnostic test. However the sensitivity and

specificity of TLI is lower than that for fPLI, and in experimental pancreatitis it is less dramatically elevated and declines more rapidly than fPLI. fPLI is likely to be positive in cases of moderate to severe pancreatitis but in mild cases it will have a lower sensitivity (i.e. false negatives).

Imaging can be useful, but has limitations – the classic loss of detail in the cranial abdomen will only be seen in acute cases and not always. Ultrasonography has a high specificity (>85%, i.e. few false positives) but low sensitivity (<35%, false negatives are common). Findings include hypoechogenicity of the pancreas, a large or bulky pancreas, hyperechogenicity of surrounding fat, effusion, other organ pathology (liver, GIT).

The definitive diagnosis is made with histology – many clinicians are nervous about biopsy of the pancreas but a study (of healthy cats) showed gentle handling and biopsy did not result in pancreatitis. When a high PLI has been obtained you have to decide if treatment or prognostic info given to the owner will be changed by biopsy results. Cases with a normal PLI may therefore be more appropriate to biopsy (i.e. the milder forms of pancreatitis that give a normal PLI). To be discussed further in lecture!

Supportive treatment

FLUID THERAPY: Cats with pancreatitis are often dehydrated, and may also be significantly hypovolaemic (think about this, do you know the clinical difference between these terms?). Crystalloids (Lactated Ringers or 0.9% NaCl) are usually recommended for initial replacement. Hypovolaemic cats (presenting with pale mucous membranes, poor pulse quality, tachycardia or bradycardia, and, in severe cases, low arterial blood pressure) will benefit from an initial bolus of fluids (10-20ml/kg over 20 minutes and repeated as needed). Close attention to cardiovascular parameters is important, and be aware that cats are particularly prone to volume overload, often presenting as pleural effusion, if excessive fluids (especially colloids) are administered.
Plasma administration has been recommended in order to provide a source of protease inhibitors, which in theory should reduce activation of pancreatic enzymes. However studies in cats are lacking, and availability in practice is limited. This will only really be appropriate for ANP.

Attention must also be paid to electrolyte abnormalities, in particular hypocalcaemia and hypokalaemia. Guidelines for management of hypocalcaemia in cats with pancreatitis are not well established, but the authors have used parenteral calcium gluconate for management of moderate-to-severe reductions in ionised calcium concentration. Note that calcium solutions must not be added to fluids containing bicarbonate. Potassium chloride can be added to maintenance fluids, the rate depending on the severity of hypokalaemia. Care must be taken not to administer potassium at rates exceeding 0.5mmol/kg/h.

NUTRITION: In cats, vomiting is less common than in dogs which allows enteral nutrition to be addressed early in the course of treatment. Hepatic lipidosis is common in cats with pancreatitis and is associated with a poorer prognosis. Appropriate, aggressive nutrition is essential to prevent hepatic lipidosis developing or worsening.

Attentive nursing and appetite stimulants may tempt cats to eat, but rarely results in consumption of the full caloric requirement. If vomiting is not a major feature, then naso-oesophageal or oesophagostomy tubes can be placed with minimal equipment. THINK ABOUT EARLY USE OF FEEDING TUBES IN THESE CASES!

SUPPORTIVE CARE: Pancreatitis is a painful condition, and opioid analgesia is appropriate. Suggested drugs include pethidine (3-10mg/kg q 4-6 hours), morphine (0.1-0.5mg/kg I/M or S/C q 6-8 hours) or buprenorphine (0.01-0.02mg/kg I/M or S/C every 8 hours). Fentanyl patches can be placed on the skin to provide analgesia for up to 72 hours. Non-steroidal anti-inflammatory drugs should be avoided in severe pancreatitis due to increased risks of renal toxicity in dehydrated or hypovolaemic patients.

Although vomiting is uncommon in feline pancreatitis, nausea is probably common and so some cats will benefit from administration of anti-emetics. Metoclopramide can be given be injection or, more effectively, by continuous rate infusion at a dose of 1-2mg/kg/24 hours. This is easily achieved by adding the appropriate amount of metoclopramide to the cat's maintenance fluids. Metoclopramide is light sensitive so fluids should then be protected from light, for example by wrapping the bag and fluid lines in bandage. In patients with intractable vomiting, maropitant or chlorpromazine may be useful, although it can have sedative side-effects. Mirtazepine has an anti-emetic and appetite stimulant effect.

Vitamin B supplementation may be helpful in cats with prolonged anorexia and/or concurrent GI or liver disease, as B12 deficiency is common in these patients.

Often the most important aspect of therapy is the treatment of concurrent disease such as IBD, cholangitis/hepatitis and diabetes mellitus. At this stage, there are no recognised treatments to prevent ongoing inflammation and fibrosis in cases of chronic pancreatitis and management of these cases can be very problematic, relying on symptomatic treatment alone.

Antibiotics are often given but this is likely a sterile process, however ANP cases are at risk of sepsis and may benefit from broad spectrum antibiotics. Research continues on whether there is a bacterial component to pancreatitis in cats. Corticosteroids are only indicated in cases with a biopsy result of lymphocytic pancreatitis, and they often have concurrent IBD. Further work is needed to see if corticosteroids are indicated or not.

Surgery is only indicated in cases of EHBDO and pancreatic cysts or abscessation (although literature describes some pancreatic duct stenting procedures).

When managing a case of pancreatitis it is worth warning owners that there is no ‘quick fix’, recurrence is possible and nursing with close attention to nutrition and nausea are required.

Think – ANALGESIA, NAUSEA, NUTRITION, MANAGEMENT OF CONCURRENT DISEASE!