Management of Chronic Kidney Disease in Cats

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Chronic renal failure is one of the most common illnesses of geriatric cats (Ludich et al., 1992), and in one study the incidence was reported to have increased from 4.5% to 9.6% between 1990 and 2000 (Plotnick, 2007). It is typically a progressive disease resulting in significant morbidity and mortality in cats. The prevalence increases with age and up to 31% of cats over 15 years are affected.

It is presumed that most cats develop chronic kidney disease (CKD) after an earlier renal insult (e.g., infectious, immune-mediated, congenital, metabolic, neoplastic, traumatic, obstructive event) which may have gone unnoticed without causing clinical signs. CKD is an irreversible condition as nephrons cannot be regenerated. Fortunately the kidney has considerable reserves and in early stages nephron loss may pass unnoticed. However, once a critical level of renal damage has occurred, chronic renal failure develops. This can be self-perpetuating and may progress from an asymptomatic non-azotaemic period to end-stage uremia. Acute renal failure (ARF) can be reversible if diagnosed promptly and treated effectively however recurrence (e.g., urethral obstruction) or prolonged ARF can progress to CKD.

Polyuria and polydipsia as a result of inadequate urine concentrating ability (normal urine specific gravity >1.035) are usually the first clinical signs noticed by the owners and occur when renal function is only about one-third of normal. Anorexia only develops when 75% of nephrons are non-functional, thus early detection is crucial in order to implement measures that support renal function and protect against complications (e.g., hyperkalaemia, secondary hyperparathyroidism) associated with the disease. Depending on the stage of CRF clinical signs can be variable.

Clinical Signs
- Polyuria
- Polydipsia
- Anorexia
- Vomiting
- Weight loss and loss of body condition
- Pallor
- Oral ulceration
- Acute blindness secondary to hypertension

Laboratory abnormalities

* Blood
  - Azotaemia
  - Hyperphosphataemia
  - Hyperkalaemia
  - Hypercalcaemia
  - Metabolic acidosis
  - Non-regenerative anaemia

* Urine
  - Isosthenuric urine
  - Proteinuria
  - Urinary Tract Infections

IRIS classification (www.iris-kidney.com)

Accurate staging of chronic kidney disease allows the clinician to choose the most appropriate therapies, monitor the patient and assess prognosis. The “International Renal Interest Society” (IRIS) has produced a set of guidelines which help to stage chronic kidney disease based on serum creatinine values (Table 1), and substage based on proteinuria (Table 2) and systolic blood pressure (Table 3). The creatinine values of stage 1 may be considered within the normal reference range for many labs, however the IRIS staging system takes into account that significant renal disease can be present in the absence of azotaemia. Recent studies have highlighted the importance of proteinuria in renal disease. It is now understood that even low levels (i.e. UP/C >0.4) of
proteinuria are significant whereas previously 0.5-1.0 was used as a cut-off. In a healthy cat the UPC should however not exceed 0.2. However, proteinuria can have many other causes (see below).

<table>
<thead>
<tr>
<th>UPC value</th>
<th>Substage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.2</td>
<td>Non-proteinuric</td>
</tr>
<tr>
<td>0.2 - 0.4</td>
<td>Borderline proteinuric</td>
</tr>
<tr>
<td>&gt;0.4</td>
<td>Proteinuric</td>
</tr>
</tbody>
</table>

Table 2: IRIS staging on urine protein: creatinine ratio (UPC).

<table>
<thead>
<tr>
<th>Systolic BP</th>
<th>Diastolic BP</th>
<th>Substage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;150</td>
<td>&lt;65</td>
<td>Minimal risk</td>
</tr>
<tr>
<td>150-159</td>
<td>95-99</td>
<td>Low risk</td>
</tr>
<tr>
<td>160-179</td>
<td>100-119</td>
<td>Moderate risk</td>
</tr>
<tr>
<td>&gt;180</td>
<td>&gt;120</td>
<td>High risk</td>
</tr>
</tbody>
</table>

Table 3: IRIS staging on blood pressure (BP).

Markers of Renal Function

Creatinine and urea

Although commonly used, creatinine is not the most sensitive indicator of renal clearance, and in early kidney disease, small changes in creatinine may represent large changes in glomerular filtration rate (GFR). As GFR has to be compromised significantly (>75% nephrons non-functional) before plasma creatinine leads to accumulation in the bloodstream exceeding the upper limit of normal there is significant overlap in the creatinine levels of healthy cats and those with early renal disease. Sequential samples may be of greater use than a single value to identify trends. Despite staying in the reference range, sequential increases of plasma creatinine may still be indicative of a progression of the renal disease.

A number of factors can influence urea concentrations in addition to GFR. The most important is dehydration which is a common feature in cats with CKD. The ingestion of protein meals (e.g. food, GI haemorrhage, catabolic state) and-to a lesser extent-its production by the liver can also have an impact on urea measurements. Creatinine measurement is not influenced by diet and is a better marker of GFR than urea. However, poorly muscled, thin cats can have reduced creatinine due to the reduced muscle turnover. Hence, some cats with CKD have normal creatinine and elevated urea.

Proteinuria

A small amount of protein can be found in the urine of healthy cats and may be physiological and transient associated with strenuous exercise, stress or pyrexia. Postrenal causes for proteinuria include trauma, neoplasia, urinary tract infection (UTI), or haemorrhage. Urine dipsticks are more sensitive to albumin but can yield false positive results in concentrated or alkaline urine or false negative results in dilute urine. For example, a trace of protein in a very concentrated sample is less likely to be significant than if the same amount is present in dilute urine.

Proteinuria is not as common in cats with chronic kidney disease as in dogs but if it is present it is a predictor for progressive renal damage. Persistent proteinuria should always be quantified with the means of urine protein:creatinine (UPC) ratio which is an accurate measurement unaffected by the urine concentration or daily fluctuations. The severity of proteinuria has been found to be associated with survival time. A UPC >0.4 has been linked with a four fold higher risk of death or euthanasia (Syne et al., 2006). Haematuria and pyuria may lead to increases in the UPC, but in these cases an active sediment would be expected. Before staging according to the IRIS system pre-renal (e.g. haemorrhagic, hyperglycaemia, functional renal proteinuria) and post-renal (e.g. lower urinary tract infection) proteinuria as well as concurrent inflammation/infection should be excluded. Large amounts of protein with inactive sediment can also occur in association with glomerular disease (e.g. glomerulonephritis). If persistent proteinuria with the absence of inflammatory urinary sediment is detected, it may be suspicious of early renal injury and warrant intervention.

An even earlier marker of proteinuria in microalbuminuria which is defined as the presence of a very small quantity of urinary albumin (<30mg/dl) below the limit of detection for a dipstick. However, various disease processes (e.g. inflammation, infections) and drugs (e.g. prednisolone) may lead to a positive result and the significance of microalbuminuria is therefore currently not fully understood.

Urine concentrating ability

The urine concentrating ability is the earliest marker of tubular renal disease and any patient suspected of kidney disease should undergo sequential measurements. In azotemic patients determining the specific gravity (SG) further allows differentiating between renal and pre-renal causes. Ideally, urine should be collected at the same time as blood samples and before fluid therapy or administration of drugs (e.g. diuretics, steroids) that could affect the urine concentration.

Phosphate

Hyperphosphataemia occurs commonly in approximately 60-80% of cases of CKD due to secondary hyperparathyroidism. The prevalence rises with progression of disease and declining renal function (DiBartola et al., 1987). In a very recent study carried out by Boyd et al. (2008), phosphate was the only clinicopathologic variable to be predictive of an increased risk of death in the multivariate analysis. With impaired kidney function, phosphate accumulates in the blood stream and perpetuates kidney disease. It was shown that for each 1U (mg/dl) increase of phosphate levels in the blood, there is an 11.8% higher risk of death, thus tackling hyperphosphataemia is crucial in prolonging survival.

Common complications and consequences of chronic kidney disease

Hypertension

One study found that hypertension occurs in almost 20% of cats with chronic renal failure in first opinion practice (Syne et al., 2002). However in cats seen at referral hospitals an incidence of as high as 65% has been reported (Silies et al., 1994). Persistent hypertension increases not only the risk of vascular injury of end-organ (e.g. eyes, brain, kidneys) but also predisposes to uraemic crises and death associated with renal disease. Even though hypertension itself is not significantly associated with survival time, management of hypertension offers indirect benefits to longevity by decreasing the level of proteinuria which is directly correlated to hypertension (Jepson et al., 2007). The complications associated with it can also be serious including e.g. hypoaemia, seizures, left ventricular hypertrophy, etc. hence blood pressure should be monitored in all cats with chronic kidney disease. About 70% of hypertensive cats with chronic renal disease were found to have lesions compatible with hypertensive retinopathy (Syne et al., 2002). The same study found that 50% of hypertensive cats had more serious complications such as hypoaemia or vision loss. Sudden onset blindness is the first sign to alert the clinician of the condition.

Hypertension in cats is defined as an indirect systolic blood pressure greater than 160 or 170 mmHg. Doppler or oscillometric methods have been used to monitor blood pressure. Stress induced hypertension and a "white coat effect" are well recognised in human canine and feline patients requiring consideration in obtaining measurements. For an accurate assessment it is crucial to allow the cat to settle down before taking multiple measurements in a calm environment.

If significant hypertension is detected, antihypertensive treatment is warranted: the drug of choice is amlopidine (Lisenta, Pfizer), a calcium channel blocker which is highly effective and well tolerated at a dose of 0.625-1.25mg PO once daily. ACE inhibitors (e.g. Enalpap®, Novartis, Erbavet®), Metol, only have relatively weak anti-hypertensive properties (reducing the BP only by 3-15 mmHg) but may offer additional intra-renal protection. The aim of treatment is to maintain the systolic blood pressure below 160 mmHg.

Renal secondary hyperparathyroidism

Renal secondary hyperparathyroidism occurs commonly in cats as a result of increased phosphate retention and an impaired ability to produce calcitriol (active vitamin D) (Barber & Elliot, 1998). With declining kidney function, phosphate which is usually filtered and reabsorbed on the proximal renal tubule, accumulates in the blood stream, leading to hyperphosphataemia. This results in a reduction of the ionised calcium which subsequently stimulates the production of parathyroid hormone (PTH).

Further, the decrease of renal functional mass also affects the production of calcitriol (active Vitamin D). In turn lower than normal levels of calcium result in a decrease of intestinal calcium uptake, the mobilisation of calcium from the mineralised reserves (bone) and an increase of the PTH level.

Dietary phosphate restriction and the use of phosphate binders help to resolve hyperphosphataemia and control renal secondary hyperparathyroidism (Barber et al., 1999).

Even though calcitriol given orally would be expected to reduce PTH levels, in a recent study (Horvath et al., 2006) calcitriol failed to normalise the PTH in 10 cats with chronic renal failure. However calcitriol may still be of clinical benefit when managing renal secondary hyperparathyroidism in cats with CKD (Nagode et al, 1996) as long as neither hyperphosphataemia nor hypercalcemia are present. The current evidence is however insufficient to support use against routine therapy with calcitriol and studies are ongoing to elucidate its role in feline patients.

Hyperkalaemia

Increased urinary losses as a consequence of CKD commonly result in hyperkalaemia. In general, hypertensive cats tend to have a significantly lower plasma potassium concentration than normotensive cats (Syne et al., 2002). The degree of hyperkalaemia and severity of associated clinical signs is highly variable. Classically hyperkalaemia presents as ventricularfex of the neck, but signs of generalised muscle weakness may occur. It has been speculated that hyperkalaemia may contribute to the progression of kidney failure (Yobain et al., 2000). Desipramine diets contain increased potassium concentrations to combat this problem. However, in more severe cases, potassium supplementation in the form of potassium gluconate is required to maintain levels within reference range.

Anaemia

With the progression of CKD renal production of erythropoietin reduces. In conjunction with a shortened
erythrocyte life span and possible uremia induced gastrointestinal haemorrhage many patients develop anemia, typically non-regenerative in nature. Clinical signs may become apparent once the haematocrit falls below 20% and intestinal proteinostases (e.g. sucralfate, S. meprobate, Glaucus, E2 blockers, propranolal), blood transfusions or administration of recombinant human erythropoietin have been suggested. The use of EPO should be reserved until the patient develops clinical signs associated with anemia, due to the risk of development of EPO antibodies and aplastic anemia (occurring in up to 30% of treated cats). Anabolic steroids to boost appetite and red blood cell mass are sometimes prescribed in practice, however there is no evidence of effectiveness and they are not recommended in the management of CKD.

Gastrointestinal signs
Nausea, vomiting and anorexia occur frequently in cats with CKD. In particular, the inability of the failing kidneys to excrete excess gastrin, a digestive hormone, results in increased gastric acidity and possibly gastric ulceration which is reflected in the presenting clinical signs (Goldstein et al., 1998). The accumulation of uraemic toxins further contributes to the development of gastroenteritis. The use of antacids, antacids and gastric proteinostases may prove beneficial in the management of these cases.

Metabolic acidosis
Chronic acidosis is a common feature of chronic kidney disease due to the decreased renal ability to excrete acid. This may exacerurate renal injury and hypokalaemia, thus most prescription diets are alkaline in nature to address this problem. The assessment of blood gases to determine carbon dioxide, bicarbonate levels and blood pH may help in quantifying the acidemia accurately. However, blood-gas machines are readily readily available in practice. Hence the use of alkalising agents (potassium citrate or sodium bicarbonate) is not recommended without adequate monitoring facilities.

Long-term management
Hydration
For cats hospitalised with renal failure, fluid therapy remains the cornerstone of treatment. However, adequate hydration is crucial in the management of chronic cases. Cats should have free access to water at all times. Feeding wet food, additional water bowls or flavoured water (e.g. tuna or prawn) may encourage increasing their water intake further. Some cats prefer drinking from dripping water taps and water intake may be encouraged by water fountains. In some cases, the possibility of administering subcutaneous fluids at home can be considered. Subcutaneous fluids (10-20ml/kg) are given typically by placing a needle in the intrascapular region every 2-3 days. New SQ devices have become available using either a permanent SQ catheter (Endo-Self Subcutaneous Catheter, Simetria, GFL-Ib, Practico) or a permanent "bipod" fashioned in a "Y" shape from which fluids can be injected. This is generally well tolerated by cats and may provide a useful way to prevent dehydration in the more advanced stages. Guidelines for clients are available from the FAB website (www.fabcats.org). There is also an excellent book, "Caring for the Cat with Kidney Failure" which offers valuable advice and guidance to clients (available from www.catprofessional.com).

Dietary Modification
Dietary modification has been advocated for a long time, and remains the single most important factor in preventing and slowing the degeneration in this disease. The use of prescription renal diets even if not fed exclusively has been proven to significantly increase longevity (median survival: 16 months vs 7 months (Flint et al., 2003)): 63% days vs 264 days (Ross et al., 2005)) and reduce the risk of uremic cases (Ross et al., 2005). Renal prescription diets are moderately protein restricted, have higher levels of water-soluble vitamins (e.g. vitamin B complex, vitamin C), potassium & omega-3 fatty acids and lower levels of phosphate & sodium.

<table>
<thead>
<tr>
<th>Stage at Diagnosis</th>
<th>Number of cats</th>
<th>Percentage of cats</th>
<th>Survival Median (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IIf</td>
<td>82</td>
<td>39.4</td>
<td>1.15* (1.04-1.56)</td>
</tr>
<tr>
<td>III</td>
<td>84</td>
<td>40.3</td>
<td>0.679* (0.455-0.810)</td>
</tr>
<tr>
<td>IV</td>
<td>47</td>
<td>20.2</td>
<td>0.35 (0.219)</td>
</tr>
</tbody>
</table>

Table 5: Survival time (Boyd et al., 2008)

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Number of Cats</th>
<th>Survival Median (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosis</td>
<td>211</td>
<td>771 (639-910)</td>
</tr>
<tr>
<td>Weight loss</td>
<td>142</td>
<td>237 (175-444)</td>
</tr>
<tr>
<td>Start of SC fluids</td>
<td>145</td>
<td>245 (81-483)</td>
</tr>
<tr>
<td>Creatinine [mg/dl]</td>
<td>124</td>
<td>100 (35-185)</td>
</tr>
<tr>
<td>&gt;25% weight loss</td>
<td>81</td>
<td>53 (30-194)</td>
</tr>
<tr>
<td>Creatinine [mg/dl]</td>
<td>98</td>
<td>44 (32-97)</td>
</tr>
<tr>
<td>Clinical decompensation</td>
<td>135</td>
<td>40 (31-64)</td>
</tr>
<tr>
<td>Anaemia intervention</td>
<td>42</td>
<td>25 (5-74)</td>
</tr>
</tbody>
</table>

Survival was calculated from the time of diagnosis of CKD, the point of consistent weight loss, the initiation of SC fluids (whether before or following any hospital admission), the 1st time that the creatinine was consistently >5.0 mg/dl, the 1st time that the creatinine was consistently >5.0 mg/dl, the point of clinical decompensation, and the time of intervention for anaemia.

Even though dietary modification is very valuable, the most important priority is intake of adequate energy and protein. It is much more important that the cat eats something even if this is not the ideal diet. Diets can easily be modified by adding an intestinal phosphate binder (IPB) and supplementation of potassium to "recreate" a kidney diet.

Phosphate restriction
Phosphate restriction plays a vital role in preventing renal secondary hyperparathyroidism which can promote chronic kidney disease and soft tissue and renal mineralisation (Barber et al., 1999; Chew D, 2008). Phosphate and protein restricted diets have been shown to slow down the progression of disease (Elliott et al., 2000) and their use is considered to be the first step in addressing hyperphosphataemia. If hyperphosphataemia fails to improve within 4-6 weeks despite having instigated dietary modification, an additional intestinal phosphate binder (IPB) should be added in. These must be administered with food to be effective and work by binding phosphate in the intestinal lumen, reducing intestinal absorption. IPB are aluminium, calcium and lanthanum based. In the past aluminium salts (aluminium hydroxide) have been used as a first choice treatment, however toxicity in human patients have lead to difficulties in sourcing these drugs. A study by Wagner et al. 2004 confirmed the beneficial effects in decreasing intestinal phosphate absorption when using a phosphate binder containing calcium and chitosan (Hydralyte®, Veraphos®) which is available in the US, Calcium IPB are however contra-indicated in the face of hypercalcaemia. Lanthanum salts have been developed for use in human CKD but are not available for calcium and aluminium based products. A new product containing lanthanum carbonate (Renolut® Brager) has recently been marketed. Although limited trials (Schmidt et al., 2006) have been carried out and its long-term use requires further investigations, it appears to have a dose-dependent effect and successfully reduces phosphorus absorption in combination with maintenance and phosphate restricted diets.

Omega-3 fatty acid supplementation
Recent studies (Brown et al., 1998 & 2000) suggest that diets high in omega-3 unsaturated fatty acids may help to preserve renal function in dogs. The underlying mechanism is contributed to a reduction in intra-glomerular pressure and renal inflammation. However, there are currently no studies in cats confirming these findings in this species.

ACE inhibitors
The benefits of ACE inhibitors in proteinuretic patients have been well documented (King et al., 2000; Mizutani et al., 2006) and result from the decrease in effluent arterial resistance in the glomeruli. ACE-inhibitors are recommended in cats with a LUPC over 0.4 and/or severe hypertension. Although the value of therapy in non-proteinuretic, non-hypertensive patients is currently unknown there are some studies that indicate that treatment may lead to an improved appetite.

Prognosis and survival times
The management of a cat with chronic kidney disease requires a considerable amount of financial and emotional commitment from the owner. Thus accurate prognostic information is essential to educate owners and help them making decisions based on realistic expectations. A study published in 2008 (Boyd et al., 2008) looked specifically at survival times in cats with chronic kidney disease. This study found a median survival time of 2.1 years (771 days) regardless of the time of diagnosis, but it also indicated that cats diagnosed early (Stage IIb) were documented to live up to 5.8 years, with a median of 3.315 years (1,351 days) (see Table 5 & 6). The association of laboratory variables and survival were also investigated and only phosphate (P<0.005) was found to be a prognostic factor in the final multivariate model. An increase of 1 mg/dl was associated with a 12% higher risk of death. Hypertension has also been associated with decreased survival. Age of diagnosis, albumin, urea, creatinine, calcium, bicarbonate, potassium, and haematocrit were not found to be predictive of survival.

Summary
Early diagnosis of chronic kidney disease gives us veterinarians the opportunity to promptly implement treatment that may successfully slow the rate of renal damage. Evaluation and monitoring of laboratory parameters (blood and urine) along with identification and management of associated complications (e.g. hypertension, renal secondary hyperparathyroidism, anaemia) are important in order to formulate an individual management plan. With judicious care, the prognosis of a patient diagnosed early may be favourable and result in a prolonged survival time.

References are available on request.

Utrasound of a cat with Chronic Renal Disease: The left kidney appears small and irregularly marginated with varying opacity of the cortices. Shadowing (highlighted by white arrows) consistent with a nephrolith can be observed.
CASE REPORT: 'TIGGY'
5y 6m FM SIAMESE WITH PANCREATITIS

Background
Tiggy is a five year old female, neutered Siamese who had been obtained from a breeder as a kitten. She had been vaccinated annually (FIHV, FCV, FIP and FeLV) and wormed at vaccination consultations with a veterinary licensed product. She had received intermittent prophylactic flea treatment with a POM spot on. She was housed with an unrelated Siamese who had not exhibited any clinical signs. Tiggy was an indoor/outdoor cat fed on a varied diet of wet and dry cat food and frequent treats. She had received veterinary attention on one previous occasion for an episode of hypovolaemia which had resolved without treatment. A cause was not established.

Clinical History
Tiggy presented with a one week history of anorexia and lethargy with occasional vomiting and one episode of diarrhoea. A cat bite wound in the region of the ventral neck had occurred prior to the presenting clinical signs. The referring vet had treated Tiggy with intravenous fluid therapy, amoxicillin (50mg/kg bid), prednisolone (5mg/kg q day) and uncoordinated acid (75mg po sid) but Tiggy had remained profoundly anorectic and had become jaundiced in the days prior to referral.

Clinical Examination
Tiggy had jaundiced mucous membranes, third eyelid protrusion, mild submandibular and preauricular lymph node enlargement and hepatomegaly. A small, healing bite wound was present over the ventral neck. There was mild hypotension, systolic BP (Doppler) 110mmHg (120-180). Parameters were otherwise within normal limits, HR 180bpm, RR 28, T 36.7°C.

Problem List
1. Anorexia
2. Lethargy
3. Jaundice

Differential Diagnoses
Anorexia and lethargy are very vague clinical signs seen in a variety of conditions. Jaundice can be considered as pre-hepatic, hepatic or post-hepatic.

In pre-hepatic jaundice, increased bilirubin occurs as a result of sepsis or haemorrhage. This can be caused by a variety of infective factors including *Mycoplasma haemofelis* infection, FIV body haemolysis secondary to toxicity, most commonly onion or paracetamol, FeLV infection or primary immune mediated haemolytic anaemia.

Hepatic jaundice is caused by decreased uptake of bilirubin by the liver and in the cat this can be caused by neoplastic cholangiitis, lymphocytic cholangiitis, FIP, hepatic lipidosis, amyloidosis or due to drug induced hepatopathy. In post-hepatic jaundice there is decreased excretion of bilirubin due to either intrahepatic or extrahepatic biliary compression. The former is caused by hepatic cell swelling or cholangiitis whilst extrahepatic biliary obstruction occurs due to pancreatic disease (pancreatitis, pancreatic cyst, abscess or nodule), neoplasia, traumatic rupture of the gall bladder or bile duct (usually following a road traffic accident) or, less commonly, cholelithiasis.

Investigations
Blood was collected for biochemistry, haematology, feline Pancreatic Lipase Immunoactivity (PLI) and FIV/FeLV ELISA. A urine sample was obtained for routine urinalysis, sediment examination and urine protein: creatinine ratio. Thoracic radiographs and abdominal ultrasound were obtained following sedation with ACP (0.02mg/kg im) and Buprenorphine (0.02mg/kg im).

Haematology demonstrated a normal leucocyte count, but the presence of adherent cells on the smear examination is likely to have given a falsely low count. A left shift in the neutrophils in conjunction with toxic changes is consistent with severe inflammatory disease or sepsis. The mild lymphopenia and eosinopenia were not thought to be clinically significant. The thrombocytosis was likely to be artefactual, given the normal platelet count on smear examination.

Results

<table>
<thead>
<tr>
<th>Haematology</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>9.70</td>
</tr>
<tr>
<td>MCH</td>
<td>25.5</td>
</tr>
<tr>
<td>RBC</td>
<td>6.03</td>
</tr>
<tr>
<td>MCV</td>
<td>47.2</td>
</tr>
<tr>
<td>MCHC</td>
<td>16.1</td>
</tr>
<tr>
<td>MCH</td>
<td>34</td>
</tr>
<tr>
<td>PHi</td>
<td>1078</td>
</tr>
<tr>
<td>WBC</td>
<td>9.6</td>
</tr>
<tr>
<td>Band Neutrophils</td>
<td>0.53</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>4.36</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>0.88</td>
</tr>
<tr>
<td>Monocytes</td>
<td>0.88</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0.02</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Further Laboratory Testing
- Ionised Calcium 1.08mmol/l (1.12-1.4)
- PLI (101pg/ml (2-7)
- Virusology
  - HIV and FeLV ELISA negative

Diagnostic Imaging
- Thoracic radiographs were unremarkable
- Abdominal ultrasound demonstrated a markedly enlarged and hypoechoic pancreas with increased echogenicity of surrounding mesentery. No other abnormalities were identified.

Diagnosis
- Vague presenting signs, jaundice, ultrasound changes and hypocalcaemia were consistent with pancreatitis and this was confirmed later by an elevated IPI result. Hypocalcaemia occurs in pancreatitis due to saponification of fat.

Biochemistry

<table>
<thead>
<tr>
<th>Biochemistry</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea</td>
<td>5.8</td>
</tr>
<tr>
<td>Creatinine</td>
<td>64</td>
</tr>
<tr>
<td>Teral Protein</td>
<td>56.4</td>
</tr>
<tr>
<td>Albumin</td>
<td>19.8</td>
</tr>
<tr>
<td>Globulin</td>
<td>38.6</td>
</tr>
<tr>
<td>ALT</td>
<td>72</td>
</tr>
<tr>
<td>ALP</td>
<td>21</td>
</tr>
<tr>
<td>GGT</td>
<td>11</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>173.7</td>
</tr>
<tr>
<td>Sodium</td>
<td>153</td>
</tr>
<tr>
<td>Potassium</td>
<td>2.33</td>
</tr>
<tr>
<td>Chloride</td>
<td>115</td>
</tr>
<tr>
<td>Calcium</td>
<td>2.08</td>
</tr>
<tr>
<td>Phosphate</td>
<td>0.97</td>
</tr>
</tbody>
</table>

Serum biochemistry demonstrated mildly low urea which may have been consistent with liver disease. The hypoproteinaemia and hypocalbuminaemia with normal globulin may reflect liver disease or protein losing nephropathy. The mild elevation in ALT may not be significant but may reflect early/mild hepatic parenchymal damage. The elevation in GGT is indicative of cholestatic disease. The hyperbilirubinaemia is indicative of pre-hepatic or post-hepatic disease but in conjunction with the normal haematocrit would not reflect haemolytic anaemia. The marked hypokalaemia may be due to anorexia or losses via the gastrointestinal tract although these are not reported to be significant. It may also be due to diuresis from fluid therapy prior to referral. The low total calcium could be artefactual due to the hypocalbuminaemia, and assessing ionised calcium is required to evaluate this further.

Urinalysis was unremarkable apart from marked proteinuria, urine protein:creatinine ratio 1.88 (0.4)

Treatment
Tiggy was treated with potassium supplemented intravenous fluid therapy (5.5mKCI 100ml/hr Harmanus) administered at twice maintenance (4ml/kg/h) given the mild hypotension and normal hydration status. IV Calcium supplementation was not given as the hypocalcaemia was very mild, asymptomatic and expected to improve with nutritional support. The serum potassium and calcium concentrations were re-evaluated eight hours later and were both within the reference range. Potassium 4.0mmol/l (4-5) and ionised calcium 1.33mmol/l (1.12-1.4). Oral calcium supplementation was continued over the next couple of days and monitored daily.

Potassium supplementation was continued at maintenance (1.5mKCI 1000ml/hr Harmanus). Analgesia was given in the form of buprenorphine (Vetergesic) 20µg/kg iv tid.

Natasha Hetzel BSc BVSc MRCVS Senior Clinician
Tiggy initially ate quite well and a naso-oesophageal tube was therefore not required for nutritional support. Unfortunately, Tiggy's appetite decreased after the first 24 hours of hospitalization and she was then given an appetite stimulant (Dimetapp, 3.75 mg po q 3 days), to which she responded well. As there was evidence of toxic changes and a neutrophilic left shift, treatment with intravenous broad-spectrum antibiotics, amoxicillin clavulanate (Augmentin®) 20 mg/kg iv bid and metronidazole 10 mg/kg iv bid was instigated. Repeat haematology two days later revealed a resolution of the haematological changes and a change to oral antibiotics was then instigated. Amoxicillin clavulanate (Augmentin®) 7.5 mg/kg bid and Metronidazole 40 mg/kg bid. Unfortunately, there was a clinical deterioration in association with the change to oral antibiotics with recurrence of leukocyte adherence on haematology and an increase in liver enzymes ALT 221 IU/L (45-450), ALP 121 IU/L (15-60), AST 74 IU/L (20-120) and GGT 16 IU/L (0-2). This was suspected to represent neutrophilic cholangitis which is often seen in conjunction with pancreatitis in cats. Abdominal ultrasound demonstrated mild dilatation of the common bile duct. Intravenous antibiotics were re-instigated and Tiggy improved from this point. Repeat haematology was normal and biochemistry revealed an improvement in all parameters (ALT 92 IU/L (45-450), ALP 71 IU/L (15-60), Bilirubin 15.2 mmol/L (0-100)). Urinalysis was normal and the urine protein:creatinine ratio had returned to normal. Repeat abdominal ultrasound demonstrated a normal appearance of the liver and pancreas and the diameter of the common bile duct had returned to within normal limits. Tiggy was discharged with a four week course of oral antibiotics and antioxidant therapy, S-Adenosylmethionine (Zentadil®) 100 mg po sid.

**Outcome**

Tiggy made a full recovery and has not had any repeat episodes of pancreatitis or cholangitis, nor shown any clinical evidence of inflammatory bowel disease. Repeat haematology and biochemistry performed at the end of the four week course of antibiotics were normal.

**Discussion**

**Prevalence**

Pancreatitis is the most common disease of the exocrine pancreas in cats. Necropsy studies initially reported a prevalence of 0.6–2.4%. Pancreatitis is thought to be under-diagnosed and in a recent necropsy study of 115 cats, including apparently healthy animals, an overall prevalence of 67% was recorded. Of the healthy cats, 45% had changes consistent with mild pancreatitis. This perhaps questions the clinical significance of mild histopathological changes in the pancreas, particularly when there is little suspicion of pancreatic disease.

**Histopathology**

A classification system for pancreatitis has been adapted from the human field. Cases are defined as acute or chronic, permanent histopathological changes occur in the chronic form but are reversible in acute cases. Cases can be further classified as suppurative (neutrophilic inflammation) or lymphocytic (lymphocytic inflammation). Disease may vary from mild to severe in either acute or chronic forms.

Lesions in chronic feline pancreatitis are similar to histopathological findings in humans where fibrosis is more prominent than inflammation. In acute pancreatitis, neutrophilic inflammation, interstitial oedema and mesenteric fat necrosis predominates. Chronic pancreatitis is reported to be more common in cats whilst the acute form is more frequently seen in dogs.

**Pathophysiology and Aetiology**

Pancreatitis is thought to be due to a failure of the protective mechanisms of the pancreas. These mechanisms ensure that auto-digestion does not occur by four main mechanisms. Firstly, pancreatic enzymes are kept in an inactive form (zymogens) until they enter the duodenum. Secondly, the lysosomal enzymes (which could activate the zymogens) are kept separate from the zymogens by intracellular membranes. Thirdly, a trypsin inhibitor is present in pancreatic juice to counteract any premature activation of trypsin within the acinar cells and ducts. Finally, zymogens are present in the plasma to protect against pancreatic enzymes that may inadvertently reach the circulation.

The ways in which these protective mechanisms may be overcome are not well understood but there is general agreement that trypsinogen activation occurs within the pancreas forming trypsin, which is subsequently capable of activating other zymogens. The pathophysiology of pancreatitis in cats has not yet been determined at a cellular level however, trypsinogen activation may occur when zymogens granules and lysosomal hydrodases coalesce in cytoplasmic vacuoles. This has been demonstrated experimentally but vacuoles have also been observed in healthy rats with no signs of pancreatitis. Chronic hereditary pancreatitis in humans is caused by a mutation of the trypsinogen gene at a trypsin sensitive site. Its loss may thus permit autoactivation of trypsinogen causing pancreatitis.

Once pancreatic proteases have been activated, they enter the pancreatic interstitium and peripancreal caviae, causing tissue damage. Circulating proteases also activate the complement, fibrinogen, coagulation and kinin cascades leading to systemic complications.

**Presenting Signs**

Presenting signs are vague and summarized in the table below.

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Percentage of Cats Affected (Total 40 cats)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lethargy</td>
<td>100</td>
</tr>
<tr>
<td>Anorexia</td>
<td>97</td>
</tr>
<tr>
<td>Dehydration</td>
<td>92</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>68</td>
</tr>
<tr>
<td>Vomiting</td>
<td>35</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>25</td>
</tr>
<tr>
<td>Abdominal mass effect</td>
<td>23</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>20</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>15</td>
</tr>
<tr>
<td>Ataxia</td>
<td>15</td>
</tr>
</tbody>
</table>

**Histopathology**

The most common identified haematological parameters are mild, non-regenerative anaemia and leucopenia. Haemocentration and leukocytosis have also been reported. The most frequent biochemical abnormalities are mild to moderate elevations in ALT, ALP and bilirubin. Azotaemia may be present secondary to dehydration.

Hypokalaemia is seen commonly and hypocacalcaemia may occur due to saponification of periampullar fat. It may also be related to acid-base balance, resistance to or decreased production of parathyroid hormone or increased calcitonin concentrations.

ALP is more likely to be elevated in chronic pancreatitis and hypoalbuminaemia is more likely to occur in acute cases. ALT and ALP are likely to be higher in chronic pancreatitis than in the acute form.

**Specific Biochemical Tests**

Amylase and lipase are of no clinical value in the diagnosis of feline pancreatitis. The Trypsin-like immunoreactive activity (TLI) assay has poor specificity because high TLI concentrations can also be seen in gastrointestinal disease (IBD, GI lymphoma). The sensitivity is low (28–40%) due to its short half-life. The feline pancreas lipase immunoreactivity assay (PLI) specifically measures pancreatic lipase, in contrast to other lipase assays which measure lipase from the stomach and duodenum as well. It has been demonstrated to be 100% specific in healthy cats and 100% sensitive in cats with moderate to severe pancreatitis, although the sensitivity in mild cases was only 54%.

**Imaging**

The most consistent radiographic finding in pancreatitis is a loss of peritoneal detail in the cranial abdomen, but this was only found in 50% of cases in a study of fourteen cats with chronic disease. Abdominal ultrasound provides a good survey diagnostic tool but has poor sensitivity and specificity for pancreatitis.

The sensitivity of abdominal ultrasonography has been reported to vary from 24% to 35% (2%, 10% and 18% for the presence of pancreatitis). The specificity was reported to be 73%. Unfortunately, ultrasonography relies heavily on operator skill and machine technology.

Computed tomography (CT) is a valuable diagnostic tool for human pancreatitis but no significant difference was detected between the healthy and diseased feline pancreas.

**Histopathology**

Histopathology remains the only diagnostic test that offers a definitive diagnosis of pancreatitis. It is performed fairly infrequently due to its invasive nature. It can be performed safely but the disease is often patchy and localized and thus biopsy may not be diagnostic. It should be performed only if anaesthesia is indicated for another reason such as biopsy of other abdominal organs or placement of a feeding tube. Samples should be submitted for histopatology and culture.
Feline Pancreatitis (continued)

Treatment

- Intravenous fluid therapy (Salm: 0.9% or Hartmann's) is vital to correct dehydration, acid base and electrolyte disturbances. Colloidal administration may be required in hypotensive patients.

- Opioid analgesia is vital even if there is no evidence of abdominal pain on clinical examination.

- Buprenorphine (0.1–0.2 mg/kg) subcutaneously usually provides adequate analgesia.

- Broad spectrum antibiotic therapy should be instigated until there is evidence of sepsis.

- Historically, "pancreatic rest" by withholding food has been advised but studies in humans have suggested that nutritional support is essential.1

- The risk of hepatic lipidosis as a complication of anorexia in cats makes this more pertinent. Liquid food (PurinaTM) via a nose-oesophageal or oesophagostomy tube is generally well tolerated.

- Anti-emetic treatment should be instigated when vomiting is severe or protracted. Maropitant (Cerenia®) should be administered first according to the prescription cascade. However if this is insufficient an infusion of metoclopramide at 1–2 mg/kg IV/TID. Alternatively,

- Inapparent cats may benefit from an appetite stimulant. Mirtazapine, a tetracyclic antidepressant used in human medicine also has anti-emetic and appetite stimulating effects by increasing serotonin levels in the CNS but antagonizing serotonin activity in the gastrointestinal tract. A dose of 2.5–3.75 mg/kg (1/6 to 1/4 of a 15 mg tablet) per 3 days is recommended. Caution should be exercised in using this drug in patients with compromised hepatic function and a reduced dose should certainly be given.

- Concurrent disease such as IBD and diabetes should also be treated. Treatment for IBD with corticosteroids is not contraindicated in pancreatitis cases as there is no evidence that corticosteroids aggravate pancreatitis.

Prognosis

The prognosis with mild disease is excellent. Severe cases or frequent episodes carry guarded prognosis. Hypercalcemia and hepatic lipidosis have been associated with a poor prognosis in acute pancreatitis.

References


Launch of Langford Veterinary Services

The University of Bristol Vet School has launched a new company, LVS, at the beginning of March 2009 which incorporates the original clinical services & diagnostic laboratories. The company is a wholly owned subsidiary of the UoB. Its aim will be to provide the very best care for cats in its care as well as excellent customer service. Bristol University is the first Vet School to run its own diagnostic services.

Lynne Hill, Chief Executive, commenting on the new company, said: "No other UK university has put all of its facilities into a subsidiary company like this. A new hospital, surgery and diagnostic imaging centre are currently being planned, with facilities including an MRI scanner and a CT scanner."

The range of veterinary clinics includes first opinion small animal and equine practices, referral services for equine, small animals and exotics, a farm animal practice and diagnostic laboratories. These clinics are supported by highly specialised clinicians, diagnostic imagers, anaesthetists, nurses and other support staff.

Bristol prides itself on providing a premier clinical service for cat patients as well as support for practitioners through providing specialist feline diagnostic services and advice to practitioners in management of cases.

This is an exciting development which will enhance the excellent clinical service we provide whilst emphasising customer services.

Real-time PCR for the detection of Trichromonas foetus in cats

The protozoal parasite Trichromonas foetus (TF) is now well recognised as a cause of chronic diarrhoea in cats. It is a particular problem in multi-cat households, notably pedigreed breeding catteries and rescue shelters, where one or more cats within the group are usually affected. TF-associated diarrhoea is most often seen in cats under 1 year of age, but it has also been reported in older cats. The parasite targets the large bowel causing colitis, with frequent passage of small quantities of liquid to semi-formed faeces often with blood, mucus and straining. Some affected cats develop faecal incontinence. The motile TF trophozoites can be identified in fresh faeces (ideally <2 hours old) by direct microscopic examination, but the sensitivity of this method is very low. Infestation can also be diagnosed by culturing the organism using the commercially available InBact™ TF-kit, which has been marketed for the diagnosis of TF infection in cattle. However, the InBact™ method is laborous and time consuming since pouch contents need to be examined daily by microscopy and results can only be considered negative after 12 days. Additionally, the specificity of the InBact™ system is unknown as a positive result does not preclude the possibility of infection with trichomonads other than TF. More recently, real-time PCR has been recommended as the diagnostic test of choice for TF infection, being more sensitive than both direct examination and culture by the InBact™ method. However, PCR on faeces can be problematic due to the PCR-inhibitory effect of many substances that are co-purified with the DNA during extraction. A real-time quantitative (Q)PCR has recently been developed by the Diagnostic PCR Laboratory, Langford Veterinary Services for the detection and quantification of TF in faecal samples. This new multiplex assay is the first to use an internal amplification control PCR alongside the TF PCR, enabling detection of any inhibitory substances present in the extracted DNA, which could cause false negative TF results. The use of QPCR in this new assay also allows us to report the relative amount of TF present in the faeces. The assay can be performed on a small volume of faeces (2-5ml) at a cost of £55 (+ VAT).

The treatment of choice appears to be metronidazole, which is related to metronidazole and is used to treat trichomonads in pigeons. It is not licensed for cats and experience of its use is currently limited, although it appears to be effective. A dosage of 30 mg/kg orally once daily for two weeks has been suggested. The drug does have a narrow safety margin, so cats should be monitored carefully for side effects which usually involve neurotoxicosis. There is a comprehensive information sheet which can be found on the FAB website (www.fabcats.org).

The diarrhoea will usually resolve spontaneously in untreated cats although this may take some time, months or more. Cats in which clinical signs (diarrhoea) have resolved seem to continue to excrete the organism for periods of up to two years.
Welcome to Jim Littlewood the new FAB scholar

Jim graduated from the Royal Veterinary College, University of London, in 2004. Upon graduating he worked as a first opinion small animal vet at a practice in Colchester. It was here that his interest in feline medicine was first ignited. He joined the FAB/ESFM shortly after starting in practice. After 18 months he moved to busy 3-centre practice in Hertfordshire and Bedfordshire.

During his time he saw a wide range of feline cardiology and medicine cases and this drove him to pursue a career in all things feline. He was fortunate enough to be awarded the FAB Scholarship in feline medicine at Bristol University in April 2009.

Jim has a broad interest in feline medicine but has particular interests in anaesthesia, feline cardiology and endocrinology. He hopes to sit his examinations for the RCVS certificate in Anaesthesia next year.

Three years ago Jim and his cat ‘Kit’ were joined by ‘Soopy’, a local stray, after he walked through the cat flap and decided to set up home. Presently he shows little, if any, intention of leaving as both Jim and ‘Kit’ suspect he knows when he is on to a good thing.

Applications are invited for the Fort Dodge Feline Fellowship based at the University of Bristol School of Veterinary Science

This post offers an opportunity for veterinary surgeons with a particular interest in feline medicine to gain specialist experience and expertise in this field. It has been funded by Fort Dodge Animal Health since 1987 and is based at the Bristol University Veterinary School at Langford.

The successful applicant will join a strong team working in the field of feline medicine involving both clinical and research activity. Current areas of particular interest are infectious diseases, feline immunology, endocrinology and gastroenterology. The objectives of the Fort Dodge Fellowship are to provide a link between feline research and clinical work in the department, assist in the development of feline projects and to assist in supporting the busy specialist feline diagnostic service. The Fort Dodge Fellow works very closely with the Feline Advisory Bureau Residents who have responsibility for most of the feline referrals but there is some opportunity for clinical work and there is encouragement to develop a particular aspect of feline medicine. Previous Fort Dodge Fellows have developed a particular interest in FIV, FIP, endocrine diseases (mainly diabetes mellitus) and allergic skin disease.

Nestled within the university and veterinary school, the centre will provide a stimulating environment for a research fellowship. The post offers an ideal setting for developing the skills necessary for a research career.

Further details of the post are available from:

Prol. T.J. Griffith-Jones, The Feline Centre Department of Clinical Veterinary Science Division of Companion Animals, University of Bristol Langford House, Langford, BRISTOL BS40 5DU Telephone: 0117 954 4480

Prospective applicants are invited to visit Langford and to talk to the current Fort Dodge Fellow.
ABSTRACTS

Evaluation of Urine Specific Gravity and Urine Sediment as Risk Factors for Urinary Tract Infections in Cats

Natalie Marcon Whipp, Peter W Nelms, and Virgil Dolan

Preliminary studies have shown an association between low urine specific gravity and urinary tract infections in cats. The current study evaluated the urine specific gravity and sediment characteristics in cats with and without UTIs. The study included 30 cats with UTIs and 30 healthy control cats. The urine specific gravity and sediment characteristics were measured and compared between the two groups. The results showed that cats with UTIs had significantly lower urine specific gravity and higher numbers of leukocytes and bacteria in their urine compared to the control group. These findings suggest that monitoring urine specific gravity and sediment characteristics may be useful in identifying cats at risk for urinary tract infections. Further studies are needed to validate these findings and to determine the most effective strategies for preventing UTIs in cats.

Bacterial UTI Infection Risk: Understanding the Role of Urine Specific Gravity and Sediment Characteristics

Feline UTI is a common and often debilitating disease in cats. Understanding the risk factors for UTIs is crucial for developing effective prevention strategies. In this study, we evaluated the role of urine specific gravity and sediment characteristics as risk factors for UTIs. The study included 50 cats, with 25 cats diagnosed with UTIs and 25 healthy control cats. The urine specific gravity and sediment characteristics were measured and compared between the two groups. The results showed that cats with UTIs had significantly lower urine specific gravity and higher numbers of leukocytes and bacteria in their urine compared to the control group. These findings suggest that monitoring urine specific gravity and sediment characteristics may be useful in identifying cats at risk for UTIs. Further studies are needed to validate these findings and to determine the most effective strategies for preventing UTIs in cats.

Course Notes: Reciprocal Courses from Neutering and Urinary Tract Infections in Cats

James A. Smith, Department of Clinical Veterinary Science, University of Glasgow, Glasgow, Scotland.

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