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Acute Renal Failure in Cats

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Figure 1: Any part of the Lily plant can cause ARF in cats.

INTRODUCTION

Acute renal failure (ARF) is a condition in which there is an abrupt and severe reduction in glomerular filtration rate (GFR). This sudden renal compromise results in severe electrolyte, acid-base and fluid balance derangements and causes dramatic clinical signs. Thankfully acute renal failure is rare in cats, and can be treated effectively if diagnosed promptly. The term ARF describes the condition caused by intrinsic renal disease. Pre- and post-renal causes of reduced GFR and azotaemia will result in intrinsic ARF if left untreated. Acute renal failure can also be superimposed on existing chronic renal failure (so called 'decompensated' renal failure) making the diagnosis more challenging and affecting the prognosis. The aim of this article is to outline the causes, diagnosis and treatment priorities if presented with a cat in ARF.

CAUSES OF ARF

The kidney is vulnerable to damage for several reasons. The kidneys receive a large volume of blood (20% of cardiac output) and have a high metabolic rate. These factors mean that compromise in renal blood flow will result in rapid ischaemic tubular damage and exposure to toxins will result in ARF. See table 1 for causes of ARF.

Unfortunately many nephrotoxic substances are found in the home and many owners are unaware of the potential toxicity. Lilies *(figure 1)* are very frequently found in flower arrangements and tend to be eaten by cats kept indoors. All parts of the plant are toxic. Ethylene glycol is another potential cause of toxicity, see later.

PHASES OF ARF

Toxic or ischaemic renal insults result in tubular cell damage, apoptosis and, in severe cases, necrosis. The proximal tubule is often the most severely affected, whereas the more distal portions of the nephron such as the collecting duct are less severely damaged.

ARF progresses through three phases as follows:

1. INITIATION PHASE

During this initial phase the kidney is exposed to the toxin or ischaemia resulting in tubular damage. Rapid intervention at this phase may prevent the cat developing ARF or at least reduce the severity of damage. Injury during this phase will lead to a reduction in GFR, loss of urinary concentrating ability and anuria/oliguria. It is very difficult to identify this phase of ARF as the cat may demonstrate few clinical signs.

2. MAINTENANCE PHASE

This phase begins after the damage has become irreversible and clinical signs begin to develop. Removal of causative agents/factors at this point will not affect the rate of recovery but will obviously prevent further injury. This phase can last for days to weeks.

3. RECOVERY PHASE

During this phase renal tubular regeneration occurs. This coincides with an improvement in urine production and a reduction in severity of azotaemia, although improvement in biochemical parameters can lag behind microscopic evidence of repair. During this phase the cat is often polyuric. This phase can continue for

CONTENTS

1. ISCHAEMIA	
• Hypovolaemia • Hypotension	shock, blood loss anaesthesia, shock, reduced cardiac output
• Non-steroidal anti-inflammatory drugs (NSAIDS)	Rare causes resulting in reduced renal arteriolar blood flow (polycythaemia, reduced renal arteriolar myeloma, thrombosis, DIC)
2. TOXINS	
• Therapeutic medications	antibiotics (aminoglycosides, tetracyclines) NSAIDS chemotherapeutics (doxorubicin, carboplatin, cisplatin is contra-indicated in cats) antifungals (Amphotericin B) radiocontrast agents
• Heavy metals	mercury, lead, gold salts
• Organic compounds	ethylene glycol, pesticides, solvents.
• Plants	Liliaceae family (Tiger lily, Easter lily, Rubrum lily, Day lily, Glory lily)
• Endogenous toxins	haemoglobin, myoglobin
• Miscellaneous toxins	grapes/raisins, illegal drugs
3. MISCELLANEOUS CAUSES	
• Systemic diseases	hypercalcaemia, SLE, FIP, neoplasia (lymphoma)
• Pyelonephritis	
• End stage chronic renal failure	

Table 1: Aetiology of ARF in cats (SLE = systemic lupus erythematosus, FIP = feline infectious peritonitis).

months after a severe episode of renal damage. Concentrating ability is the slowest to improve.

CLINICAL SIGNS

Due to the rapid onset of this condition, cats are usually still in good body condition. They are often depressed and can be collapsed and unresponsive. Severe uraemia can result in seizures and this is often a terminal event. Uraemic breath may be noted along with oral ulceration and cats may exhibit vomiting and diarrhoea. Depending on the cause the kidneys may be palpably enlarged and painful. Cats with previous unilateral ureteral obstruction which develop obstruction of the opposite kidney may have one small and one large kidney on palpation. The size of the urinary bladder will depend on the volume of urine produced and oliguric or anuric ARF will result in a small, empty bladder. If the animal is hyperkalaemic then bradycardia may be noted.

ARF can be superimposed on pre-existing disease including CRF and therefore clinical signs may vary.

DIAGNOSIS

There may be a history of toxin or drug exposure, although cats with outdoor access could be exposed to unknown substances. The finding of material on the coat or in the mouth will add to this suspicion and the toxin should be identified if possible. The diagnosis will be based on laboratory findings and in the majority of cases the aetiology is unknown. Urine production of less than 0.5ml/kg/hour is termed oliguria and the restoration of urine production is an important aspect of treatment. Urine output should be carefully measured via placement of a urinary catheter and urine collection system (or intermittent drainage) which can be made by using a sterile empty fluid bag *(figure 2)*.

Figure 2: Cat with simple closed urinary collection system.

LABORATORY FINDINGS

By the time of presentation the majority of affected animals will be severely azotaemic. Serum creatinine and blood urea nitrogen (BUN) will increase in proportion to the reduction in GFR. BUN may be disproportionally increased if the cat is dehydrated/hypovolaemic or is suffering gastro-intestinal haemorrhage. Electrolyte abnormalities include hyperkalaemia, which can be severe in cats with anuric ARF and result in cardiac dysrhythmias. A feature of ethylene glycol toxicity is hypocalcaemia due to the complexing of calcium with oxalate metabolites. Hyperphosphataemia often results from the reduced GFR. Cats with ARF can have severe metabolic acidosis, as a result of impaired acid secretion, reduced bicarbonate reabsorption and reduced ammonia generation. This acidosis is worsened by the acid metabolites produced in cats with ethylene glycol toxicity.

Urinalysis is important in distinguishing pre-renal from intrinsic ARF. Cats with intrinsic ARF are isosthenuric as they have reduced tubular function. Urinalysis may also reveal evidence of tubular damage (casts, protein, glucose) and calcium oxalate crystals in ethylene glycol toxicity. Urine bacterial culture is advisable.

DIAGNOSTIC IMAGING

Plain radiography may demonstrate renomegaly *(see figure 3)* and radiodense nephroliths/uroliths can be diagnosed but imaging rarely reveals the cause of ARF. Contrast radiography (intravenous urography) may result in further renal damage in the anuric/oliguric animal and is to be avoided. It can also be unrewarding due to the very slow excretion of the contrast agents. Renal ultrasound may be more rewarding and demonstrate loss of cortico-medullary definition, pelvic dilation, hydronephrosis, and changes consistent with neoplasia, as well as establishing ureter and bladder integrity. Ethylene glycol toxicity can result in a hyperechoic appearance to the renal cortex due to calcium oxalate deposition.

Further investigation such as fine needle aspiration (FNA) or biopsy of the kidney are rarely indicated nor desirable in the cat with ARF due to potential complications and the risk of anaesthesia, the exception may be renal lymphoma which can be diagnosed on renal FNA.

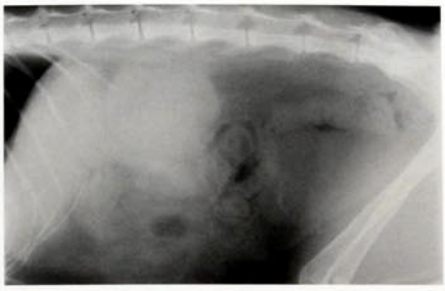

Figure 3: Lateral abdominal radiograph of a cat in acute renal failure showing bilateral renomegaly.

TREATMENT

There are several important treatment priorities when approaching a case of ARF. Obviously the inciting factor should be removed if possible (gastric lavage/ activated charcoal, withdrawal of nephrotoxic drugs – contact the VPIS for more information). Causes of pre and post-renal azotaemia should be promptly addressed. Intrinsic ARF requires a logical approach and appropriate monitoring. The following areas should be addressed:

1. Correction of Fluid Deficits and Restoration of Renal Perfusion

Fluid therapy *(see figure 4)* remains the most important aspect of management of cats with ARF. Most cats will be hypovolaemic on presentation and bolus crystalloids administered over 20-30 minutes should be given to restore renal perfusion. The choice of fluid

Figure 4: Cat receiving fluid therapy.

will depend on the cats electrolyte results. Hyperkalaemic cats should receive 0.9% sodium chloride. Once fluid deficits have been corrected, fluid therapy should be adjusted to meet ongoing losses. Urine output should be measured and replaced, as well as maintenance fluid (2ml/kg/hr) and replacement of ongoing losses in vomit/diarrhoea. Restoration of urine output is often followed by a period of polyuria which must be taken into account. If possible measurement of central venous pressure (CVP) will allow accurate correction of fluid deficits and avoid over-perfusion. Oliguric animals are at risk of over-perfusion and hypervolaemia. Clinical signs include weight gain, tachypnoea, chemosis, serous nasal discharge and crackles on thoracic auscultation.

2. Restoration of Urine Production.

In many cases urine output will improve with correction of hypovolaemia. Monitoring of CVP will allow administration of additional fluids to 'push' the kidneys whilst detecting hypervolaemia promptly. A fluid challenge can be given without CVP monitoring but the cat should be monitored closely for the development of overhydration. If urine production remains inadequate after appropriate fluid therapy then diuretic therapy is appropriate. Furosemide (a loop diuretic) will increase kaliuresis so is also indicated in hyperkalaemic cats (once adequately re-hydrated). An initial dose of 2(-6) mg/kg is given intravenously and repeated after 30 minutes if oliguria persists. If diuresis is induced then the dose can be repeated 6-8 hours later.

Mannitol (an osmotic diuretic) has additional effects of free radical scavenging, increased renal blood flow and GFR. It should be used cautiously (0.25-0.5g/kg) to avoid overhydration and should not be used in patients with pre-existing hypervolaemia.

Cats lack renal dopamine-specific receptors but there are some diuretic effects of dopamine which are explained by adrenergic effects on blood pressure. Low doses (0.5-3µg/kg/min) should be used as higher doses will cause renal vasoconstriction and cardiac arrhythmias.

3. Correction of Electrolyte and Acid-base Imbalances.

Severe hyperkalaemia will result in life-threatening cardiac arrhythmias and requires emergency treatment. Fluid therapy is the mainstay of treatment for hyperkalaemia, however if adequate hydration fails to lower the potassium level additional treatment may be required. There are a number of treatment options:

- Intravenous calcium gluconate is cardio-protective and should be given as a slow bolus (over 20 minutes) whilst the ECG is continually observed (10% solution, 0.5-1.0ml/kg). This treatment will have no effect on the hyperkalaemia which should be addressed whilst the calcium is being administered.
- Glucose stimulates endogenous insulin release which will promote cellular potassium uptake. Hypertonic solutions should be administered into a central vein or a diluted appropriately (0.5-1.5g/kg of 20% solution).
- Treatment with regular insulin works in the same way, but close monitoring for hypoglycaemia and appropriate glucose supplementation is required. Insulin should not be used without concurrent glucose administration.
- Sodium bicarbonate can be used if the cat is severely acidotic, and results in translocation of potassium into cells in exchange for hydrogen. However, very careful monitoring is essential as over correction of acidosis can result in metabolic alkalosis and hypocalcaemia. Sodium bicarbonate should not be used if blood pH cannot be monitored closely.

Hypokalaemia commonly occurs during the polyuric phase of ARF and may require supplementation intravenously or if the cat is eating, orally.

ADDITIONAL TREATMENT

If the cat is vomiting, specific anti-emetic treatment may be required as this will increase ongoing fluid losses

and make it difficult to meet calorific requirements. Metoclopramide infusions may reduce vomiting/nausea and oral sucralfate and H2-receptor antagonists may help reduce gastric acidity. Metoclopramide and H2-antagonists are eliminated by the kidneys to some extent and doses should be reduced accordingly.

Attention to nutrition is important in cats with ARF. If reluctant to eat despite correction of fluid balance, urine output, electrolyte abnormalities and any nausea then assisted feeding may be required. A naso-oesophageal feeding tube should be adequate to provide nutrition until the cat recovers but other options will need consideration if vomiting has not been controlled.

PERITONEAL DIAGNOSIS

Peritoneal dialysis can be considered if the above measures fail to induce diuresis and the azotaemia fails to improve. It involves the instillation of a dialysate solution into the peritoneal cavity to allow the removal of toxins and fluid from across the peritoneal lining. It requires intensive management and is therefore restricted to referral centres. It should be considered early in the management of acute intoxication.

PROGNOSIS

The prognosis for cats with ARF depends on the cause and severity as well as the duration of clinical signs. Prompt treatment can result in complete recovery but the prognosis remains guarded to poor, particularly for cats with toxic renal injury.

SPECIFIC MANAGEMENT OF ETHYLENE GLYCOL TOXICITY

As mentioned previously, ethylene glycol is a potential nephrotoxin. It is used in a variety of anti-freeze and screen wash products. It has a sweet taste and therefore is a more common cause of poisoning in dogs than cats. However, cats seem more sensitive to the effects than other species, with the minimum toxic dose being 1.5ml/kg. The ethylene glycol is metabolised by the enzyme alcohol dehydrogenase in the liver into glycolic acid and other toxic metabolites. The oxalate ions bind to plasma calcium resulting in the deposition of calcium oxalate crystals in the renal tubules causing severe renal tubular damage. Severe metabolic acidosis is also seen as a result of the production of acids during the metabolism of ethylene glycol.

Initial exposure can result in neurological signs as well as gastric irritation. Once the effects of initial exposure wane the animal may seem better only to go on to become severely depressed and anuric as the consequence of ARF become apparent. Exposure may be suspected from the history or by detecting laboratory abnormalities listed in Table 2.

Laboratory findings following Ethylene Glycol Toxicity

- Metabolic acidosis
- Increased anion gap
- Hypocalcaemia
- Calcium oxalate crystalluria
- Azotaemia
- Isosthenuria
- Hyperphosphataemia (due to phosphate containing 'rust prevention' additives)
- Increased serum osmolality
- Ethylene glycol detectable in the blood (test available for humans)

Table 2: Laboratory abnormalities resulting from ethylene glycol toxicity

TREATMENT

Unfortunately ethylene glycol is rapidly absorbed making treatments aimed at reducing gastrointestinal absorption (induction of emesis, activated charcoal) ineffective. It is also worth remembering that activated charcoal reduces absorption of ethanol, used in the management of ethylene glycol toxicity, so should not be used if oral ethanol is planned.

Ethanol is also a substrate for the enzyme alcohol dehydrogenase (ADH) and competitively occupies binding sites. It is not a perfect antidote however as it causes further central nervous system depression. It also has a short half life so repeated doses are required. If given orally it can result in gastric irritation and vomiting, therefore intravenous administration is recommended. Ethanol at 20% (higher concentrations are irritant) should be given at a dose of 5ml/kg every 6 hours for 5 treatments and then every 8 hours for 4 treatments.

Fomepizole is another product used to inhibit ADH activity in dogs. However, it does not inhibit the enzyme effectively in cats and is therefore not recommended.

Aggressive treatment for ARF is also vital in patients with ethylene glycol toxicity, as described above. Despite this, the prognosis for cats with ethylene glycol toxicity remains poor.

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Feline Hyp
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Feline cardiomyopathy is defined as a primary disease of the myocardium. Six types of cardiomyopathies are recognised in cats: (1) dilated cardiomyopathy, ***(2) hypertrophic cardiomyopathy (HCM),*** (3) restrictive cardiomyopathy, (4) arrhythmogenic right ventricular cardiomyopathy, (5) unclassified cardiomyopathy and (6) secondary cardiomyopathy (secondary myocardial disease). Of these, ***HCM is the most common*** (60-70%) and will be covered in this article. HCM refers only to primary or idiopathic hypertrophic cardiomyopathy while hypertrophic cardiomyopathies secondary to hyperthyroidism, hypertension or acromegaly are included within the group of secondary cardiomyopathies.

DEFINITION

HCM refers to an idiopathic disease of the ventricular myocardium (primarily left ventricle) characterised by mild to severe thickening of the papillary muscles and wall. A percentage of HCM cats (25%-67%) show dynamic left ventricular outflow tract obstruction due to ***systolic anterior movement of the mitral valve (SAM)*** and are subclassified as having ***hypertrophic obstructive cardiomyopathy (HOCM).***

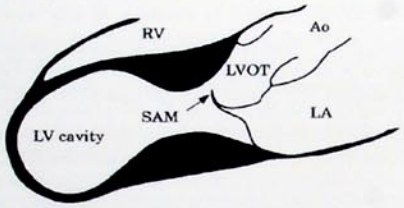

Fig. 1: Systolic anterior movement of the mitral valve (SAM) causes obstruction at the left ventricular outflow tract (LVOT). ***LV: left ventricle. Ao: Aorta. LA: Left atrium. RV: Right ventricle.***

PREVALENCE

The prevalence of HCM is high and varies among different populations and geographical areas (8-16%). Male predisposition has been widely reported; however, a study in Maine Coons suggested that males may have appeared predisposed clinically because they develop more severe and earlier disease.

AETIOLOGY

The exact cause of feline HCM is unknown, however, the disease has been shown to be heritable in many breeds such as ***Maine Coon, Persian, British Shorthair, Norwegian Forest, Ragdoll, Turkish van and Scottish fold.*** The disease is also common among ***mixed breed cats*** and heritability has also been shown in a family of mixed breed cats. Other breeds such as Siamese, Abyssinians and Burmese appear to be uncommonly affected. HCM has been suggested as an animal model for human hypertrophic cardiomyopathy. In human hypertrophic cardiomyopathy many genetic mutations have been identified as the direct cause of the disease. Similarly, two separate genetic mutations in the myosin binding protein C gen have been found as responsible for some cases in Maine Coons and Ragdoll.

PATHOPHYSIOLOGY

HCM causes abnormal diastolic function which in moderate to severe cases leads to congestive heart failure. Thickening of the myocardium itself increases chamber stiffness and together with the ischaemia, cell death and fibrosis associated with the hypertrophy caused decrease in ventricular compliance (filling phase) and also in relaxation. Factors associated with the disease which can initiate or contribute to the development of clinical signs include (1) ***arrhythmias*** (mainly supra- and ventricular tachyarrhythmias) which can reduce diastolic filling, increase outflow gradients and reduce forward cardiac output; (2) ***SAM,*** which increases systolic intraventricular pressure, increasing myocardial wall stress and stimulating further ventricular hypertrophy and (3) ***mitral regurgitation,*** (commonly secondary to SAM but also seen in non obstructive HCM) which can increase preload. Although HCM is primarily a diastolic disease, pulsed tissue Doppler imaging has shown that mild systolic impairment also occurs from early stages of the disease unrelated to SAM or heart failure. With conventional echocardiography, regional systolic dysfunction or end-stage myocardial (generalised systolic) failure can also be observed in a small number of cases.

CLINICAL PRESENTATION

Four main clinical presentations of HCM:

(1) THE ASYMPTOMATIC CAT:

These cats may be identified because they undergo screening investigations as they are breeding cats belonging to a high risk family or a high risk breed (e.g. Maine Coon or Ragdoll) or because an abnormal heart sound is detected during a routine clinical examination or while investigating other disease problems. These include (a) gallop sounds, (b) murmurs and (c) arrhythmias.

(a) Gallop sounds are a sequence of three sounds due to an audible extra S3 (protodiastolic), an S4 (presystolic) or summation of both. Gallop sounds always indicate abnormal diastole. It is usually difficult to differentiate whether a gallop sound is S3 or S4 on auscultation, this is however of no importance clinically as ***gallop sounds generally represent significant cardiac pathology*** and cardiac investigation should always be performed. Gallop sounds in HCM cats are usually S4 as it is a sound that occurs with atrial contraction and is common in diastolic restrictive disease. On rare occasions a gallop sound may be due to an extracardiac disease such as anaemia, or may be confused with a more benign sound such as a systolic click. However, the vast majority of gallop sounds indicate the presence of significant heart disease.

(b) Murmurs in adult cats ***can be physiological*** due to extra-cardiac disease (e.g. anaemia), recognised benign murmurs (e.g. dynamic right ventricular outflow tract obstruction) or be of uncertain origin; ***however, many are associated with structural heart disease.*** The most prevalent cause of a murmur in cats with hypertrophic cardiomyopathy is the presence of SAM which causes a left basilar systolic (due to left ventricular outflow tract obstruction) and/or left apical systolic (due to mitral regurgitation) murmurs. Left sided systolic heart murmurs in HCM can also be due to a basal septal bulge which causes narrowing and turbulence of the left ventricular outflow tract (left basilar) or due to mitral regurgitation caused by the compromised apposition of the mitral valve leaflets related to papillary muscle hypertrophy (left apical). Septal hypertrophy may also result in dynamic right ventricular outflow tract obstruction creating a systolic murmur best detected on the right sternal border.

Heart murmurs or gallops in an individual cat ***may be very variable,*** and may be very ***obvious on some occasions and absent on other examinations.*** These variations may be ***heart rate or stress/relaxation dependent.***

c) Arrhythmias can be detected after careful auscultation in some HCM cases. ***Tachyarrhythmias*** including supraventricular or ventricular premature complexes are not uncommon while ***bradyarrhythmias*** (e.g. third degree A-V block with a junctional escape rhythm) are occasionally detected.

(2) THE CAT IN CONGESTIVE HEART FAILURE (CHF):

Impaired ventricular relaxation produces elevated atrial and venous pressures, which lead eventually to left-sided heart failure: ***pulmonary oedema and/or pleural effusion.*** Pleural effusions are usually modified transudates although chylous effusions are occasionally encountered. Unlike dogs, ***pleural effusions in cats can occur in left-sided heart failure*** due to drainage of the visceral pleural veins into the pulmonary veins. Clinical signs of concurrent right-sided heart failure (e.g. ascites, jugular distension, peripheral oedema) rarely occur. The main presenting clinical signs tend to be ***respiratory distress or severe tachypnoea*** due to pulmonary oedema or pleural effusion and associated signs of malaise, such as inappetance, anorexia or lethargy. ***Coughing can occur but it is uncommon.*** Because of the sedentary and/or outdoor nature of most cats subtle signs such as mild tachypnoea or exercise intolerance usually go undetected. ***Sinus tachycardia is commonly seen*** but unlike dogs, cats with congestive heart failure ***can show normal sinus rhythm or sinus bradycardia*** on presentation (and obviously ***arrhythmias*** as described above). Fulminant heart failure (particularly in HOCM cats) can be precipitated after severe stress, however, in the majority of cases, the disease has been present for months or years and the heart failure has been gradually worsening over weeks to months. Congestive heart failure can also occur in subclinical cases following the treatment of other diseases (e.g. fluid therapy).

(3) THE CAT WITH SYSTEMIC ARTERIAL THROMBOEMBOLISM (ATE):

The left atrial dilation and the damaged left atrial and left ventricular endothelium that occurs in cats with HCM can lead to ***blood stasis and exposure of subendothelial collagen.*** The hypercoagulable state of these cats might be a further contributing factor in the development of ATE. The incidence of thromboembolism in the HCM population has been estimated to be between 6-17%. The clot forms in the left atrium and then travels out of the heart. In most cats ***(90%) the clot lodges at the aortic trifurcation*** extending to the iliac arteries (saddle appearance). The cessation of blood flow cause uni- or bilateral signs of ***loss of femoral pulses, cold extremities, pain, pale or cyanotic pads or paresis and paralysis of the hindlimbs (see Fig.2).*** Clinical signs and a Doppler device indicating the absence of blood flow is usually enough to reach a diagnosis, however, there is potential for ***misdiagnosis of primary neurological disease.*** Around 10-25% of ATE are due to other underlying conditions such as pulmonary neoplasia. Other locations where the clot may lodge are much less common but include brachial, renal, pulmonary, mesenteric, coronary or cerebral arteries. Cats with ATE are commonly presented with ***tachypnoea (91%).*** One important aspect is to ***determine (by radiography) whether the tachypnoea is due to congestive heart failure (44% of cases)*** or due to pain, acidosis, etc, as these cats tend to be dehydrated and hypothermic and injudicious diuresis may be fatal.

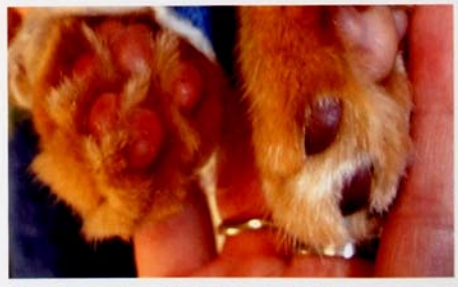

Fig. 2: Cyanotic pads in this HCM cat following a unilateral thromboembolism affecting the right foreleg. ***(Courtesy of Yolanda Martinez-University of Edinburgh).***

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(4) THE CAT WITH SYNCOPE:

This occurs sporadically and may be due to haemodynamic causes ***(severe SAM)*** and/or ***arrhythmias*** (mainly ventricular or supraventricular tachyarrhythmias which may be episodic).

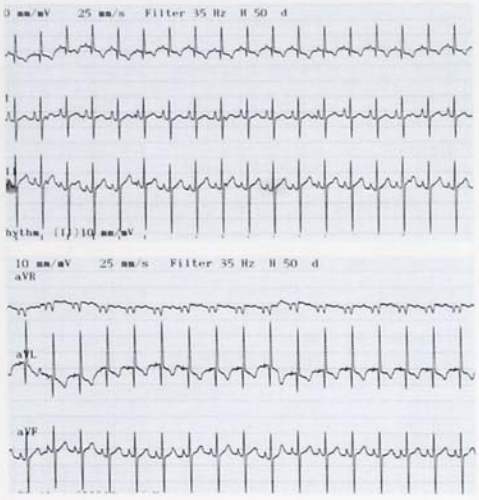

Fig. 3: Left anterior fascicular block in a cat with HCM showing left axis deviation with deep S waves in leads II, III and aVF and qR pattern in lead I and aVL.

DIAGNOSIS

Electrocardiography: ***Arrhythmias or left ventricular enlargement*** (R wave > 0.9 mV tall) can be seen in some cases but its presence is highly variable and nonspecific for HCM. Left axis deviation compatible with ***left anterior fascicular block*** is seen in many HCM cases (11-33%) but rarely in other cardiomyopathies. Some syncopal episodes may be due to episodic arrhythmias and a holter monitor may be necessary for diagnosis *(see Fig.3).*

Radiography: Radiography is most useful for detecting congestive heart failure. Radiography will indicate the presence of ***cardiomegaly and pleural effusion*** and it is the ***only means to determine*** the presence of ***pulmonary oedema*** and vascular changes. There is a great overlap of radiographic findings among different cardiomyopathies and ***it is not possible to differentiate the type of cardiomyopathy based on radiography. A normal thoracic radiograph does not rule out HCM (see Fig.4).***

Genetic Mutation testing: This is available at The Veterinary Cardiac Genetic Lab of the Washington State University for two breeds: Maine Coons and Ragdolls. A positive result means that these cats may show evidence of HCM during their lifetime but does not accurately predict the prognosis for every individual cat. Although a negative result tells you that this cat does not have that particular mutation, the cat may still develop the disease.

Blood markers of myocardial disease: There is increasing evidence that two biochemical markers of cardiac disease, Cardiac troponin I (leakage marker) and NT-pro BNP (functional marker),

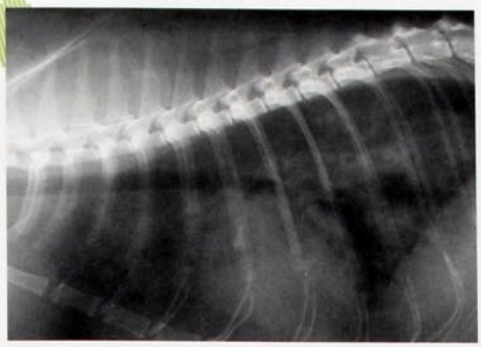

Fig. 4: Pulmonary oedema in a previously asymptomatic cat with mild to moderate HCM following the administration of fluid therapy (twice maintenance crystalloids) which was administered to treat other unrelated condition.

are useful in differentiating cardiac and respiratory disease and possibly CHF cats vs. asymptomatic cats. There is however overlap and these tests must be interpreted carefully together with the clinical signs and other diagnostic investigations. These tests do not differentiate the different underlying heart diseases.

Echocardiography is the ***main tool for the diagnosis of feline myocardial diseases.*** In HCM, hypertrophy (thickening) of the left ventricle and on occasions the right ventricle is seen ***with a wall thickness at end-diastole greater than 6mm (see Fig.5).*** The disease is very ***variable*** and can involve the septum and free wall, or both, or may be focal. Relying more on ***multiple imaging views on 2D echocardiography*** (rather than M-mode) may be a solution for detecting focal thickening whilst avoiding inadvertent measurement of papillary muscles but ***care must be taken with the timing.*** Other possible findings include left atrial and often right atrial enlargement, decreased DLV internal dimensions with hypertrophied papillary muscles, mitral regurgitation and normal to elevated LV fractional shortening. ***SAM*** can be identified on ***M-Mode or 2D images*** and it is associated with ***dynamic left ventricular outflow tract obstruction on Spectral Doppler*** measurements (dagger shaped envelope) and a characteristic ***bilobed jet on Colour flow Doppler (see Figs. 6,7 and 8).*** The aetiology of SAM is not completely understood but probably involves the mitral apparatus being pulled into the left ventricular outflow tract by the enlarged papillary muscles during systole together with the associated venturi effect on the leaflet. ***SAM may be labile*** and only present with higher sympathetic tone, hence, stressing the cat during the echocardiography (e.g. by increasing the volume of the Doppler sound) may show a SAM previously unnoticed. SAM often precedes wall thickening and ***can be an early indicator of the disease. Echocardiographic*** parameters considered as ***negative prognostic factors*** include ***severity of thickening, presence and severity of left atrial enlargement, presence of smoke or thrombus or direct evidence of congestive heart failure*** (e.g. pleural/pericardial effusion). ***Concentric hypertrophy can also be secondary to various causes*** including congenital aortic stenosis, ***systemic arterial hypertension or hyperthyroidism.*** Echocardiography, measurement of T4 and blood pressure can be used to rule these out. Less commonly, other diseases such as acromegaly or infiltrative diseases (e.g. lymphoma) may also cause wall thickening. ***Diastolic function*** is abnormal in cats with HCM cats and it is assessed by measurement of transmitral flow patterns, relaxation time, pulmonary venous flow velocities and also by Doppler tissue imaging. ***Three patterns are seen depending on the stage of the disease: impaired relaxation, pseudonormal and restrictive.*** (Further information on diastolic function assessment is beyond the scope of this article.) In some cats, distinguishing the normal from mild HCM may be difficult and these cases are categorised as equivocal and the echocardiography should be repeated (say after 6 months).

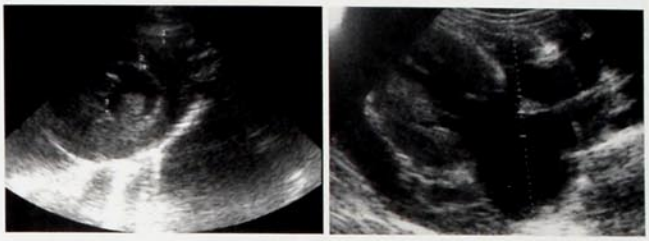

Fig. 5: Right parasternal short axis view (a) of a cat with HCM showing marked asymmetric hypertrophy with the left ventricular wall being more affected. Right parasternal long axis view (b) of a cat with moderate to severe HCM showing a moderate pericardial effusion. Pericardial effusion is occasionally seen in cats with HCM in congestive heart failure, normally together with pleural effusion or pulmonary oedema but sporadically on its own.

TREATMENT

THE ASYMPTOMATIC CAT:

There is no evidence based on placebo-controlled studies to suggest that any of the drugs with theoretical benefits (diltiazem, b-blockers or ACE inhibitors) alter the progression of HCM before the onset of heart failure. Treatment at this stage, therefore, is generally not recommended. However, cats with SAM (particularly if high gradients of obstruction at low heart rates are seen and/or they are outdoor cats) are generally treated by the author with a β-blocker (e.g. atenolol). B-blockers have been shown to be more effective than diltiazem in reducing the degree of outflow tract obstruction caused by SAM. The other group of cats that occasionally receive treatment are patients with severe tachycardia (>240 bpm) which are also given beta blockade if episodes of stress are suspected.

THE CAT IN CONGESTIVE HEART FAILURE:

(1) Acute therapy: The initial treatment for these cats is to remove any pleural effusion present by ***thoracocentesis*** and to relieve congestion through the use of drugs that reduce preload. ***Oxygen therapy*** and therapy with ***furosemide and nitroglycerine*** may be used in the acute setting. An oxygen cage is preferred over other means of oxygen delivery to avoid stress. Thoracocentesis can be performed in the vast majority of cases without chemical restraint with or without local anaesthesia. Furosemide is used for the cat with pulmonary oedema and for those after thoracocentesis for diuresis. Depending on severity, the dose varies from 1mg/kg IV every 1-2 hours until improvement to a standard chronic dose of 2mg/kg BID which can be administered by any route, although parenteral is preferred while the cat is hospitalised. Topical nitroglycerine (0.5-0.75 cm q 8 hours) is also advocated as a venous dilator and preload reducer. ***Response to therapy is assessed by monitoring respiratory rate*** (Decreasing the respiratory rate should lead to reduction in diuretic ·herapy; BID therapy can usually be started when the respiratory rate is below 40-45 breaths per minute). Radiography or a brief thoracic ultrasound can also be repeated every so often (e.g. every 8-12 hours) to detect resolution or worsening of the oedema or effusion. ***Minimizing stress and avoiding severe dehydration are two key issues in the management of these cases.*** It is important to give enough diuresis to relieve the congestion but not to overtreat with diuretics. It is also of importance to monitor ***blood pressure,*** electrolytes and renal parameters. Hypokalaemia can induce anorexia complicating further the situation.

(2) Chronic therapy: ***Furosemide*** is continued at 2mg/kg BID. An ***ACEi*** (see chart for doses and types) is added to diuretic therapy due to its multiple theoretical benefits (e.g. vasodilation or attenuation of sympathetic drive) and its proven benefits in other species (humans and dogs). No clinical studies have clearly shown confirmation of their

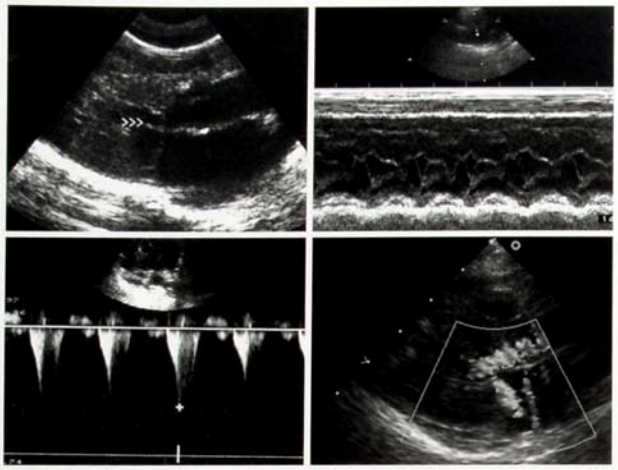

Fig. 6. Systolic anterior movement of the mitral valve shown on a right parasternal long axis view 2D (a) and M-Mode (b) which causes dynamic left ventricular obstruction creating a dagger shaped envelope on Continuous Spectral Doppler of the left ventricular outflow tract on a left parasternal five chamber view (c) and mitral regurgitation (seen with Colour Doppler on a right parasternal long axis view; (d). *(Pictures 6b and 6d courtesy of Laín Garcia-Molins Veterinary Hospital).*

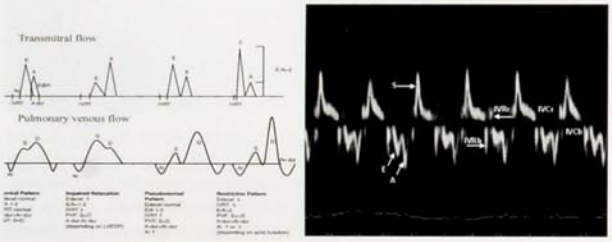

Fig. 7. Transmitral and pulmonary venous flow patterns (a) and an example of E-A wave reversal on Tissue Doppler imaging of a cat with HCM (b). E-Early filling; A-Filling due to atrial systole; Ao-Aortic valve closure; IVRT-Isovolumic relaxation time; A-dur-A wave duration; E-A velocity at point of (incomplete) summation; S-Pulmonary venous flows; D-Pulmonary venous flow; Ar-Pulmonary venous flow atrial reversal wave; decel-deceleration; PVF-Pulmonary venous flow; LVEDP-left ventricular end diastolic pressure. *(Courtesy of Kerry Simpson-University of Edinburgh).*

benefit in cats. ***The respiratory rate at rest*** should be ***monitored regularly by the owner at home*** and the aim is to achieve the ***minimum effective dose of furosemide*** (Absence of congestive heart failure is normally indicated by respiratory rates below 30-35 breaths per minute but there are individual variations). Initially, frequent re-examinations (and frequent telephone updates on appetite, demeanor and respiration rate) until a stable situation is reached are recommended in order to detect early congestion and carry out therapeutic adjustments as one of the most common reasons for euthanasia is recurrence of the clinical signs (i.e. severe pleural effusion or pulmonary oedema that requires re-hospitalisation). Depending on progress, thoracocentesis may have to be repeated or furosemide increased. If dose requirements for furosemide exceed 8mg/kg/day another diuretic (such as spironolactone or a thiazide-see chart) may be used. Spironolactone may be the first choice as it may also have other potential benefits (e.g. reduction of myocardial fibrosis) and is less often associated with complications (e.g. hypokalaemia). Some refractory cases may need higher doses of furosemide that those suggested by the reference ranges. Frequent monitoring of renal parameters and electrolytes is important. A double-blinded study evaluated chronic use of a β-blocker or diltiazem plus furosemide vs. furosemide alone and no evidence was found of any benefit in using any of these drugs. They are also best avoided in the acute situation as they are negative inotropes. Beta-blockers are occasionally used for cats with SAM (as described for the asymptomatic cat) but only once the congestive heart failure has resolved.

PREVENTION AND TREATMENT OF ATE: Anti-coagulant treatment is usually ***started when there is left atrial enlargement, smoke or a clot are seen in the heart or after an ATE episode*** has occurred. ***Aspirin*** is used due to its anti-platelet aggregation properties but its true clinical efficacy remains unknown. Similar results are found when classical or low-dose are used (see chart). ***Clopidrogel*** has shown in vivo and in vitro anti-platelet aggregation activity in cats. It is possibly more potent than aspirin, however clopidrogel is more expensive. A clinical study comparing aspirin to clopidrogel is underway. A third possible drug used by the author is ***Dalteparin*** (low molecular weight heparin - see chart) which inhibits secondary haemostasis and therefore complements the primary haemostastic inhibition of aspirin or clopidrogel. It is given via subcutaneous injection which can be administered by the owner in the same manner as for example insulin. This drug is advised following a successful recovery of an ATE episode or after severe smoke (slow moving blood) or a clot is seen within the heart. It is expensive and requires committed owners. No clinical studies have been performed to determine the real benefit of any of these treatments in preventing ATE. The treatment of ATE is based on cage rest, pain control, supportive fluid and nutrition therapy, prevention of new thrombus formation and treatment of heart failure if present. Reported survival rates are low (35%-39%) and cats with concurrent heart failure have shorter survival times. Many of these cats develop a second thrombus weeks to months after recovery. In many cases euthanasia is elected considering the poor survival rate and the severe underlying cardiac disease. Dissolution of the clot via fibrinolytic agents or surgery is not performed due to the high mortality associated with these procedures.

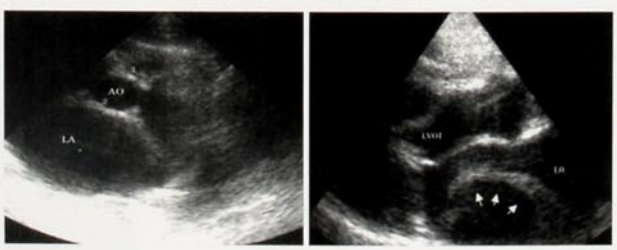

Fig. 8: Echocardiographic images of two HCM cats, one showing left atrial enlargement (left atrium-LA/Aorta-Ao ratio above 1.6/1) on a right parasternal short axis view (a) and the second one with evidence of smoke (slow moving blood) on a right parasternal long axis view (b; arrows). LVOT-left ventricular outflow tract.

Recovery can be very slow and takes a long time. It requires dedicated nursing.

PROGNOSIS

The prognosis for HCM is very variable. Asymptomatic cats with mild to moderate disease and normal left atrium have a good short-term prognosis and many may have a good long term prognosis. Cats with moderate to severe disease that already have mild left atrial enlargement are likely to develop heart failure in the future and if the left atrial enlargement is severe this will probably occur imminently.

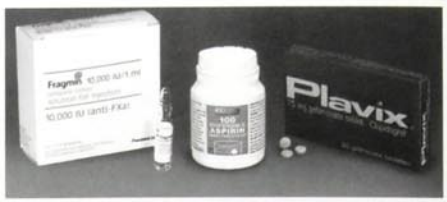

Fig. 9: Aspirin, clopidrogel and dalteparin are used for the prevention of ATE *(Packs not shown to scale).*

These cats will also be at risk of ATE. Cats presented in heart failure have a poor prognosis but some cats (20% in one study) appear to do well for prolonged periods. Many of these cases may represent cats where the heart failure is associated with some trigger factors (e.g. severe stress) and may stabilise well after treatment. Median survival times reported in different studies ranged from 1830 to 1129 days for asymptomatic cats, 654 days for syncopal cats, from 92 to 563 days for CHF cats and from 61 to 184 days for the ATE group. Significant prognostic indicators include SAM which appears to be positively associated with survival time and ***left atrial size and severity of the hypertrophy*** which ***are negatively associated with survival time.***

Table 1: Drugs commonly used in HCM cats

Drug	Preparation	Dose
Benazepril (ACEi)	5, 20mg ***Fortekor®*** (Novartis)	0.25-0.5mg/kg PO q24h
Enalapril (ACEi)	1, 2.5, 5, 10, 20mg ***Enacard®*** (Merial)	0.5mg/kg PO q12-24h
Ramipril (ACEi)	1.25, 2.5, 5mg ***Vasotop®*** (Intervet)	0.125mg/kg PO q24h
Imidapril (ACEi)	150, 300mg ***Prilium®*** (Vetoquinol)	0.5 mg/kg PO q24h
Propanolol (B1, B2 blocker)	10, 40mg ***Inderal®***	2.5-5mg/cat PO q8h
Atenolol (B1 blocker)	25, 50, 100mg ***Tenormin®***	6.25-12.5mg/cat PO q12-24h[1]
Diltiazem (Ca-channel blocker)	10mg ***Hypercard®*** (Arnolds)	10mg/cat PO q8h
Aspirin (platelet aggregation inhibitor)	75mg	High dose: 75mg/cat PO every 3 days Low dose: 5mg/cat PO every three days[2]
Clopidrogel (platelet aggregator inhibitor)	75mg ***Plavix®***	18.5-75mg/cat PO q24 h[3]
Dalteparin (low molecular weight heparin-inhibitor of coagulation)	10.000 IU/ml ***Fragmin®***	100 IU/kg SC q12h
Nitroglycerine (venodilator)	2% topical formulation ***Percutol®***	0.5-0.75 cm q8h over a clipped or hairless region
Furosemide (loop diuretic)	Injectable 50mg/ml Oral 20, 40mg	Variable. PO, SC, IM, IV (dosing discussed in the text)
Spironolactone (potassium-sparing diuretic)	25mg tablet/1mg/ml, 2mg/ml, 5mg/ml,	2-4mg/kg PO q24h

1. Twice a day dosing is preferred if possible (i.e. no sides effects, owners compliance).
2. The author uses 25% of a 75 mg tablet per cat every three days (lowest possible dose without the need for reformulation)
3. The author uses 18.75mg as this dose appears to have the same beneficial effect as 75mg.

Further reading:

FOX, P.R. (1999) Feline Cardiomyopathies. In: Textbook of Canine and Feline Cardiology. Eds P.R. Fox, D. Sisson & N.S. Moise. *W.B. Saunders, Philadelphia.* **pp 621-678.**

KITTLESON, M.D. (2005) Feline Myocardial Disease. In: *Textbook of Veterinary Internal Medicine. Eds S.J.Ettinger & E.C. Feldman. W.B. Saunders, Philadelphia.* **pp 1082-1104.**

Further references available on request.

Welcome to Rachel Korman
the new FAB scholar

Rachel graduated from the University of Queensland Veterinary School in 2000. She worked in a busy small animal clinic in Australia before moving to the UK in 2003. She worked as a locum throughout the country, including time in a feline only practice, before settling down to manage a predominantly cat practice in central London. She spent three years there, and built the clinic up to a Silver award winner in the FAB cat friendly practice scheme. Rachel was also delighted to receive a Cynthia award for her dedication to cats. The statue took pride of place next to her bed for some time!

In 2006 she gained the ESVPS Certificate in Feline Practice. Her interests in feline medicine include geriatric medicine, kidney disease and medical oncology.

Rachel's special little someone is Neko - an elderly rescue cat from the Battersea Dog and Cat Shelter. Neko rules her home with an iron paw and ensures the dog knows her place.

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 major objectives of the Fort Dodge Fellowship are to provide a link between feline clinical and research 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.

Newly qualified veterinary surgeons will be considered for this post but some experience is an advantage. The post is ideal for veterinary surgeons wishing to pursue an interest in feline medicine. It provides an insight into an academic/research career and is particularly suitable for the graduate who wishes to consider this without making a long term commitment. The Fort Dodge Feline Fellowship provides an excellent basis for a subsequent academic or research career and previous Fellows have subsequently undertaken PhD projects arising from their year.

This position normally starts in October and is for one year although reappointment at the end of the first year may be considered.

Further details of the post are available from:

Prof. T.J. Gruffydd-Jones, The Feline Centre
Department of Clinical Veterinary Science
Division of Companion Animals, University of Bristol
Langford House, Langford, BRISTOL BS40 5DU
Telephone: 0117 928 9558

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

ABSTRACTS

Evaluation of trends in urolith composition in cats: 5.230 cases (1985-2004)

Allison B Cannon, Jodi L Westropp, Annette L Ruby et al.
JAVMA 2007; 231(4):570-576.

Urolithiasis accounts for approximately 15%-21% of the diagnoses in cats with clinical signs of disease in the lower portion of the urinary tract. They can lead to serious problems such as ureteral obstruction, acute renal failure, and death. The principal treatment is surgical removal, although medical dissolution, voiding hydropropulsion and lithotripsy can be used for certain minerals.

The purpose of this study was to determine the various types of minerals that were contained in uroliths and submitted to the Gerald V Ling Urinary Stone Analysis Laboratory at the University of California, Davis.

A retrospective computer-based search of records from 1985 to 2004 was carried out. The records of all urinary calculi specimens were reviewed and information obtained from a questionnaire submitted along with the urolith by the veterinarian was analysed.

Analytical methods to identify mineral composition included oil immersion crystallography, polarising light microscopy, infrared spectroscopy, x-ray diffractometry, high-pressure liquid chromatography, scanning electron microscopy with energy dispersive x-rays and electron probe micro-analysis. Any mineral detected was recorded and the percentage of each mineral in each layer was estimated. In calculi composed of a mixture of 2 or more minerals, uniform distribution or distinct layering was reported (e.g. struvite/oxalate containing) and each mineral was counted once leading to totals over 100%.

In total, 5.230 specimens were analysed. Most of them were located in the bladder, however 665 were removed from the lower portion of the urinary tract. 699 (13%) were composed of calculi containing more than one substance.

The number of struvite- and calcium oxalate-containing stones was 2.270 and 2.764 respectively. During the past 20 years, the ratio of calcium-oxalate to struvite containing stones increased significantly ($p<0.001$). In the 1990s, the markedly observed rise in the number of calcium oxalate stones was attributed to overzealous use of acidifying and potentially magnesium restricted diets. Increasing awareness of veterinarians and the pet food industry, however, have reversed this trend and in the last 3 years an emerging trend with more struvite-containing uroliths (44%) than calcium oxalate-containing (40%) has been noted.

Calcium oxalate was detected significantly more frequently in male compared with female cats ($p<0.05$). A significantly increased number of calcium oxalate-containing stones were found in the upper urinary tract (e.g. kidneys and ureters). Persians and Himalayans had significantly more calcium oxalate-containing calculi. The percentage of struvite-containing stones from female cats has decreased significantly in the last 15 years and they were detected significantly more frequently in younger cats than calcium oxalate ($p<0.001$). Manx cats and Siamese had significantly more struvite-containing calculi.

Apatite-containing uroliths (n=296) were predominantly removed from the bladder (71%). Occurrence in cats under 4 years was rare. No sex and breed predilections were found. The decrease in the number of apatite-containing calculi appeared to be secondary to acidifying diets as they commonly precipitate with struvite.

The number of dried solidified blood (DSB)-containing calculi analysed was 60 with a significant increase in numbers detected during the past 15 years. Males were significantly more likely to suffer from those calculi ($p<0.05$). No significant differences regarding age and location were found.

Five hundred seven uroliths contained urate. Despite an increase in detection in the 1980s, the mean percentage of stones containing urate has remained stable at 10%. No sex, age or location predisposition was found. Siamese cats were significantly more likely to have urate containing calculi.

Overall, acidifying diets to decrease the risk of struvite urolith formation, male gender and indoor housing have been reported as independent risk factors for calcium oxalate urolithiasis. It has been hypothesised that breed predisposition of Persians and Himalayans is connected to vascular abnormalities at the tip of the renal papilla. However, the pathophysiology is poorly understood and genetics may play a role.

Outbreaks of renal failure associated with melamine and cyanuric acid in dogs and cats in 2004 and 2007

Cathy A Brown, Kyu-Shik Jeong, Robert H Poppenga et al.
J Vet Diagn Invest 2007; 19: 525-531.

Pet-food contaminated with toxins has caused large outbreaks of renal failure occurring in dogs and cats in 2004 and 2007. In Korea, 6000 dogs and a smaller number of cats developed clinical signs of toxic renal failure which was attributed to mycotoxin contamination of raw materials originating from China in 2004. In 2007, a second outbreak affected large numbers in North America and several major commercial pet food companies recalled more than 1000 potentially contaminated pet food products. It is presumed that melamine added to raise the protein content may combine with cyanuric acid to insoluble crystals that obstruct and damage renal tubules and, hence cause renal failure.

This study investigated histopathologic, toxicologic and clinicopathologic changes in 16 animals affected in these 2 outbreaks of pet-food associated renal failure (2 dogs in 2004; 10 cats and 4 cats in 2007). All 16 animals had clinical and laboratory evidence of uraemia, including anorexia, vomiting, lethargy, polyuria, azotaemia, and hyperphosphataemia. Serum creatinine concentrations ranged from 7 to 15 mg/dl (reference range 0.9-2.1 mg/dl) and blood urea greater than 140 mg/dl (reference range 20-34 mg/dl) as well as hyperphosphataemia 11.3-25 mg/dl (3.2-6.2 mg/dl) were observed in all animals. Serum hepatic enzyme concentrations were normal in animals from both outbreaks. All animals died or were euthanized because of severe uraemia.

Eight of 16 animals had extrarenal lesions related to the uraemia consisting of oral ulceration, mineralization of the gastric mucosa, and mineralization of pulmonary smooth muscle and alveolar wall. Histologic evidence of hepatic injury was not found. Histologic renal lesions were present in all 16 animals with melamine-associated renal failure (MARF), with unique light green to slightly basophilic crystals. In contrast to oxalate crystals, they were present in the distal tubules or collecting ducts, often exhibited a striated appearance and were easily visualised without polarization. Pale green, glassy oxalate crystals were seen in polarized light in the proximal tubules, which were attributed to secondary oxalosis associated with tubular injury.

Eleven animals had lesions confined to the tubules which were interpreted as acute renal MARF. Five animals had in addition to renal tubular necrosis and characteristic intratubular crystals, lesions of mild to moderate interstitial fibrosis and lymphoplasmacytic inflammation which were indicative of chronic stages of MARF. Tubular rupture occurred in some of these chronic cases. In general, larger crystals in the medulla were common and aggregates of crystals were often present in the papilla as grossly visible renoliths. These chronic cases were seen at least 4 weeks after the pet-food recall and more frequently observed in dogs.

Toxicologic analyses confirmed melamine and cyanuric acid in all tested kidney samples, but they were not found in one urine sample of a dog that had not ingested the recalled pet food for 3 months.

Overall, more cats were presented for necropsy in 2007 which appeared to be indicative of the increased susceptibility in cats due to physiologic differences in tubular function between dogs and cats. The characteristic renal lesions, location in the distal tubules and polarizing properties should prevent misidentification as oxalate crystals and attribution to most other nephrotoxins. Chronic interstitial fibrosis as found in chronic kidney disease may mask cases of chronic MARF and sublethal MARF could represent an important, previously unrecognised form of chronic kidney disease in cats and dogs.

Response of feral cats to vaccination at the time of neutering

Sarah M Fischer; Cassie M Quest; Edward J Dubovi et al.
JAVMA 2007; 230(1):52-57.

Feral cats have successfully adapted to almost every ecological niche and the population in the United States is suspected to rival that of the owned cat population (estimated 90.5 million in 2006). They play an important role in spread of infectious diseases. Trap-neuter-return (TNR) programmes to control the feral cat population have been set up. The vaccination policy, however, varies according to the available resources and the effectiveness of a single dose vaccination which is administered under the stressful condition of capture, anaesthesia and surgery are widely questioned.

The objective of this study was to determine whether vaccine administration to feral cats at the time of neutering induces a protective serum antibody titre. Sixty-one cats were randomly assigned to receive either inactivated virus vaccines or modified live vaccines (MLV) against feline herpesvirus (FHV), feline calicivirus (FCV), feline parvovirus (FPV) and feline leukaemia virus (FeLV) immediately after surgery. Blood (6mls) was taken under general anaesthesia and serum stored at -20° C pending analysis.

The cats were recaptured two months post surgery and were given a booster injection. At the same time, blood was collected under a brief sedation.

Lab personnel carrying out the analysis were blinded to the type of vaccination and the timing of sample collection. Reciprocal serum-antibody titres which were considered to indicate adequate response to vaccination were established 40 (FPV), 16 (FHV) and 32 (FCV). A serum antibody titre >25 was considered to be provide effective immunisation against rabies virus (RV).

Statistical analysis did not reveal significant differences between the age groups, gender and type of vaccination. At the time of surgery, protective serum antibodies were found in 33% for FPV, 21% FHV, and 64% FCV suggesting previous exposure or vaccination. Median titre and the proportion of cats that had protective antibody titres for FPV and FCV increased significantly following vaccination and did not differ between the inactivated-virus and MLV vaccine group. Median antibody titres for FHV increased significantly compared to the baseline for both types of vaccine used but cats that received the inactivated virus vaccine developed significantly higher antibody titres than cats from the MLV group.

In 3% of cats, serum antibody titres against RV were identified at the time of surgery. After receiving an inactivated RV vaccine, the proportion of cats that had protective antibody titres increased significantly. Only one cat failed to respond but was tested positive for FeLV and FIV. Another cat infected with FeLV/FIV remained seronegative. This cat also showed no detectable booster effect to FPV, FCV, FHV despite protective serum antibody titres at the time of surgery.

The authors conclude that a substantial number of feral cats had serologic evidence to previous exposure or vaccination. It appears that they are susceptible to infection and that they have a high risk of natural exposure to viral diseases. Serum antibody-titres that would be regarded as protective were found in a high percentage of cats when checking the second blood sample: 90% for FPV; 56% for FHV; 93% for FCV; 98% for RV. The duration of immunity remains unclear and ideally feral cats should be recaptured to receive booster vaccinations and particularly against RV in accordance with the guidelines of the American Association of Feline Practitioners.

The results of this study do confirm that vaccination at the time of neutering does not lead to any significant impairment of the immune response. It is advisable to administer the vaccination during the recovery phase post surgery to lessen the potential of an obscured adverse event. As finances are often limited for TNR programs, the herd health management approach should be embraced with an emphasis on neutering and if universal vaccination is not feasible and a focus on high-risk groups, such as kittens and cats living in colonies with a history of disease.

COURSE NOTES: *Reprints of Course Notes from Feline Update Continuing Education days are available for sale. For an order form please contact:*

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Feline Update is published as a co-operative venture between The University of Bristol and Fort Dodge Animal Health. Any correspondence should be addressed to Prof. T. Gruffydd-Jones, The Feline Centre, Department of Clinical Veterinary Science, University of Bristol, Langford House, Langford, BS40 5DU, or to Matthew Rowe BSc (Hons) at Fort Dodge Animal Health, Southampton, SO30 4QH.