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Hot Topics in Feline Medicine Online 'Mini Series'

Session 2:
Peeing A Little or Peeing A Lot

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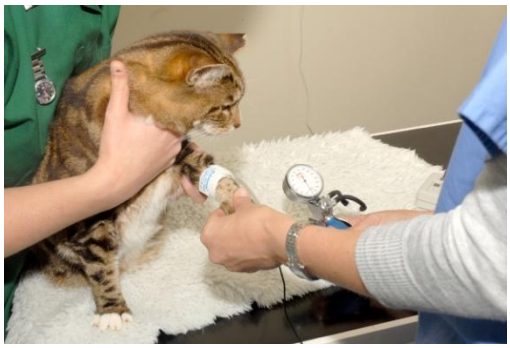


Hot Topics in Feline Medicine Part 2

What's new in feline kidney disease?

New names for old conditions

Traditionally we have called the classic condition affecting old cats with compromised renal function 'chronic renal failure'. However, this is not a very useful term as 'failure' is subjective and this condition is more complex with a progression from initial loss of concentrating ability progressing to failure of excretion of nitrogenous waste products.

Therefore CRF should now be termed **CHRONIC KIDNEY DISEASE (CKD)**. This brings us in line with human medicine and is used to describe structural or functional abnormalities of one or both kidneys that have been present for an extended period (this varies but usually say 2-3 months with duration inferred by clinical signs, physical exam or other findings). CKD now replaces 'renal insufficiency' and 'failure' entirely.

In common with the change in CKD we also need to recognise a change in the terms use for acute kidney problems. So now acute renal failure should be termed **ACUTE KIDNEY INJURY (AKI).** As again 'failure' is a non-specific term and in AKI it is a spectrum of disease, some of which is reversible and some not.

Staging chronic kidney disease

Another recent development is the advice that we should 'stage' CKD. There are several advantages to doing this:

- It allows different clinicians dealing with the case to see at a glance what the severity of the CKD is and apply the most appropriate treatment
- It always guidelines to be created for management of each 'stage'
- It allows you to give owners an accurate prognosis
- It assists with research into the subject as cats are grouped according to stage
- See http://www.iris.com for lots more details

Staging is done as follows:

Firstly the classification is made according to creatinine concentration as well as any other indicators of renal disease. The stage should only be allocated once pre-renal azotaemia has been corrected as many cats will revert to a lower stage once they are re-hydrated. The same is true in acute-on-chronic disease when an improvement is azotaemia will lower the stage following treatment.

Initial stage assessment – serum creatinine

- ***Stage I (non-azotaemia CKD):***

Normal creatinine (<140µmol/l) but another abnormality present indicating renal disease (e.g. renomegaly, proteinuria, poor urine concentrating ability)

- ***Stage II (mild CKD):***

Creatinine above the reference range at 140-250 µmol/l. This lower level (around 140mmol/l) may be within some laboratories reference ranges.

- ***Stage III (moderate CKD):***

Plasma creatinine 251-439 µmol/l.

- ***Stage IV (severe CKD):***

Cats with this stage of CKD have creatinine levels >440 µmol/l.

Substaging CKD – proteinuria

Substaging according to protein level in the urine is of increasing importance as studies identify proteinuria as a significant negative prognostic indicator in feline CKD. Staging is performed according to UPC measurement in patients with inactive sediments. Interestingly in end-stage and severe renal disease the UPC can actually fall as there simply are not enough glomeruli remaining to losing the protein into the urine.

SUBSTAGE	**Non-proteinuric (NP)**	**Borderline proteinuric (BP)**	**Proteinuric (P)**
UPC	<0.2	0.2-0.4	>0.4

Substaging CKD – blood pressure

Cats with CKD frequently have elevated blood pressure and this will cause progression of renal disease as well as clinical signs and reduced quality of life in affected cats. Untreated hypertension causes end-organ damage including damage to the brain, heart and further renal damage. Ocular changes are also common including renal detachment and haemorrhage, and total retinal detachment and blindness.

Diagnosis of Hypertension

The most common methods used to measure blood pressure are indirect techniques Doppler and oscillometry.

Indirect Blood pressure Measurement in Cats

Indirect/non-invasive blood pressure measurement in cats is now a widely used technique in general practice and is really essential for practices seeing cats. Using indirect techniques can provide an indication of systolic blood pressure, although this technique will never be as

accurate as direct methods. Experience of the equipment will improve the ability to achieve accurate results.

Options include:

1. Doppler Ultrasound Technique

This technique has been shown to be the most reliable of the indirect techniques. The procedure involves use of a cuff, sphygmomanometer and an ultrasound transducer with amplifier.
Cuffs need to be the appropriate size with usually 2 options, 2.5cm and 3.3cm. Ideally cuffs are 30-40% of the limb circumference.
This technique is very easy and rapid with practice. In many cases it is better done with the owner present to reassure the cat. Do not restrain the cat if possible and keep the atmosphere calm and quiet (see below). This technique only reads systolic blood pressure (although with experience a diastolic reading can be taken) but this is not a problem as diastolic hypertension is not reported in cats.

2. Oscillometric Technique

This technique is not as reliable in conscious cats. It can be useful in anaesthetised cats as it will document a trend in BP. The author has found this technique unreliable and been referred cases reported to have normal BP on multiple occasions with this technique and found it actually nearing 300mmHg! Multiple readings should be obtained and in aggressive cats this may be easier as it can be put around the tail base and is used 'at a distance' compared to the Doppler technique. Always check the machine is providing as accurate heart rate along side the BP.

Substaging according to results:

>180 mmHg is classified as severe risk of end organ damage (H)
160 to 179 mmHg is classified as moderate risk of end organ damage (M)
150 - 159 mmHg is classified as low risk of end organ damage (L)
<150 mmHg is classified as minimal or no risk of end organ damage (N)

Always take note of the cat's demeanour during measurement and what cuff size was used to allow monitoring.

A further Substage can be assessed on the basis of complications related to the blood pressure. These complications include evidence of end organ damage (see below) and an annotation ‘nc’ for no complications and ‘c’ for complications is given.

Assessing ‘End Organ’ Damage

If you are in any doubt about the SBP readings evidence of end organ damage from the hypertension should convince you it is present and is an indication for urgent treatment.

1. The Eye

This includes retinal haemorrhage, detachment, intraocular haemorrhage and subtle vessel tortuosity (retinal or iris). Sudden onset blindness or anisocoria can occur as a result of retinal detachment.

Ocular examination should include assessment of PLRs, and using direct, or indirect ophthalmoscopy. Indirect takes a little practice but can provide a quicker and wider view of the retina. Eyes may need dilating to fully assess the retina.

2. The Kidneys

This may be the cause or effect of the hypertension. Hypertension may cause glomerular and tubulointerstitial changes. Therefore biochem including urea and creatinine should be performed. Urine protein content has been associated with hypertension, and reduced survival so check a UPC and always a USG. Whether the cause or effect treatment of hypertension will help prevent progression of renal disease.

3. The Heart

Ventricular hypertrophy is a consequence of chronic hypertension. Equally hypertrophic cardiomyopathy can result in hypertension. Studies showed that treatment of hypertension could result in improvement in cardiac parameters. As mentioned before CHF is rare as the result of hypertension alone and cats should be checked for other disease such as hyperthyroidism.

4. The Brain

Neurological signs are rare with hypertension alone but can occur with severe and untreated hypertension. ABP should be checked in all older cats with CNS signs. Differentials include neoplasia, vestibular disease etc.

Once underlying causes have been assessed and treated if necessary then the hypertension should be treated. Early treatment is vital to prevent further damage. Also blind cats with

retinal damage can recover some sight if the hypertension is treated effectively as the retina may re-attach.

Cats with CKD and moderate or high risk of end organ damage should be treated with anti-hypertensive drugs.

Avoiding 'White Coat' Hypertension

- Think about the cat prior to entry to the consult room – the waiting room for example – separate cat room or area away from dogs – cat friendly practice techniques.
- Wear ear phones when using Doppler to prevent noise upsetting the cat.
- If not using headphones then keep volume off until the probe is in position.
- Keep owners present if possible
- Use minimal restraint
- Inflate the cuff gently and not suddenly
- Ignore the first couple of readings
- If the readings are not consistent, have a break, leave the cuff on, reassure the cat and start again.
- Take at least 5 readings and average
- Check heart rate corresponds with readings from machines
- If contact not great with Doppler use more gel
- Clean area with surgical spirit before using gel and Doppler probe
- Do not clip unless very hairy cats and if needed clip quickly then have a break before starting.

Which cats should have their BP checked?

- All cats with diseases known to be associated with hypertension
  - CKD
  - Hyperthyroidism
  - Hyperaldosteronism
  - DM
  - Chronic corticosteroid treatment
- All cats with clinical signs related to hypertension
  - Neurological signs
  - Ocular changes
  - Non-specific signs in older cats
- All senior cats?
  - Debatable issue – early identification prevents end organ damage
  - Senior health clinics?

What's new in treatment of CKD

The mainstays of management of CKD remains the same but recent research suggests focusing on some other treatment targets too. Management should be 'MULTIMODAL' i.e. include diet, medications, lifestyle changes which together improve quality of life and lengthen life. This should include the following:

- Specific therapy for the primary disease (often not available)
- Management of comorbid disease (these are older cats with > 1 disease commonly e.g. hyperthyroidism, osteoarthritis)
- Management of consequences of CKD
    - ANAEMIA – non-regenerative
    - ARTERIAL HYPERTENSION
    - DEHYDRATION
    - HYPERPARATHYROIDISM
    - HYPERPHOSPHATAEMIA
    - HYPOCALCAEMIA AND HYPERCALCAEMIA
    - HYPOKALAEMIA
    - PROTEIN MALNUTRITION AND MUSCLE BREAKDOWN
    - METABOLIC ACIDOSIS
    - URAEMIA
    - PROTEINURIA
    - GI EFFECTS (nausea etc)
    - UTI
- Modify progression of CKD
    - TREATMENT OF PROTEINURIA
    - TREATMENT OF HYPERTENSION
    - MANAGEMENT OF HYPERPARATHYROIDISM

This lecture will discuss dietary management, hyperphosphataemia, proteinuria and hypertension as treatment targets that affect prognosis.

Dietary management of CKD remains very important. The protein restriction is perhaps not as vital as previously considered and it may be that other factors are more important. Components of a renal diet usually include:

- Reduced phosphate
- Reduced (but high quality) protein
- Reduced sodium
- Increased B vitamins
- Increase caloric density
- Polyunsaturated fatty acids (PUFAs)

- Increased potassium
- Reduced acidifying effect
- Antioxidants

Some cats find these diets unpalatable. If so the following may be useful:

- Feed as only part of the diet i.e. normal wet food but dry renal – it will still be beneficial
- Try even cats that only 'eat wet food' on dry and visa versa as sometimes they are surprising in what they will eat
- Try more than one brand: Hill's, Royal Canin and Purina diets taste different and older cats are fussy so try all and owners may need to keep all types and flavours and rotate through to provide variety
- Do not introduce during an inappetant phase or if the cat has been vomiting, food aversions can be permanent
- Use appetite stimulants initially and sometimes cats get used to and start to like the diet as the initial aversion is unfamiliarity (mirtazapine)
- Begin the diet early in the disease before nausea etc have become an issue – DO NOT INTRODUCE DURING A PERIOD OF DETERIORATION, wait until they are stable again
- Transition carefully, offering both old and new diets and slowly withdrawing the old diet
- Think about the bowl – high sides are not popular and a saucer may be better
- Flavour enhancers and warming the food may help

The use of renal diets has been shown to prolong survival in cats. This is likely related to phosphate restriction (see below). Many reports support the recommendation of the use of renal diets in stages II-IV. One study showed that a diet high in essential fatty acids improved survival in cats with CKD and they are included in many renal diets.

Management of hyperphosphataemia

Aggressive management of hyperphosphataemia prolongs survival in CKD. It should be a treatment priority.

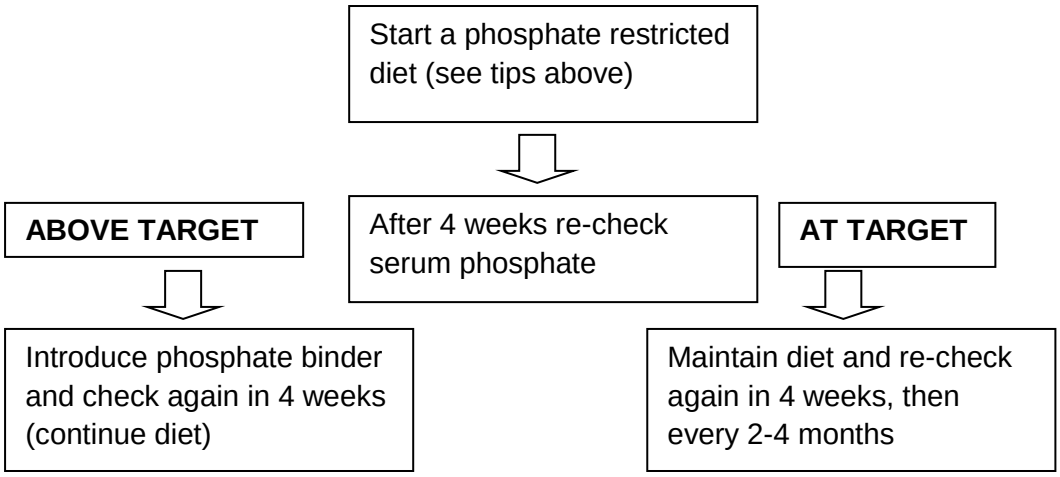
The goal is to maintain serum phosphate at the low end of the reference range if possible, ideally less than 1.45mmol/l. In higher stage cases this may not be possible. Ideally goals are:

IRIS stage II: <1.45mmol/l

IRIS stage III: <1.61mmol/l

IRIS stage IV <1.94mmol/l

This should be done as follows:


Start a phosphate restricted diet (see tips above)
After 4 weeks re-check serum phosphate
ABOVE TARGET
AT TARGET
Introduce phosphate binder and check again in 4 weeks (continue diet)
Maintain diet and re-check again in 4 weeks, then every 2-4 months

It can take 2-3 months for the serum phosphate level to stabilise. In this time the PTH is falling and will continue to fall whilst the phosphate is stabilised. You can measure PTH and in an ideal would this would be the gold standard. However, it is expensive and difficult to measure as sample handling is not easy (needs to be taken onto ice and transported frozen). Therefore phosphate is used as an estimate. Avoid lowering the phosphate too much or hypophosphataemic complications can occur (including haemolysis).

Intestinal phosphate binders

These agents trap phosphate in the gut as insoluble salts so they can't be absorbed. These are a few more agents available currently than previously including:

- Aluminium salts: aluminium hydroxide (Alternagel, Sucralfate) or aluminium carbonate can be used but dosing cats may be an issue palatability wise and the capsules are large and require splitting. Aluminium toxicity has been reported in dogs but is unlikely to be an issue in cats
- Calcium salts: potential problems arise with the use of calcium based drugs including; hypercalcaemia (and then soft tissue mineralisation due to high Ca x PO4 product), over suppression of PTH due to increased calcium and bone mineral breakdown. Ipakitine (Vetoquinol) is used successfully in many patients but calcium levels must be monitored. Avoid if the cats are receiving calcitriol.
- Chitosan: Ipakitine also contains chitosan (shell extract), and studies do show an effect of this drug on PTH and phosphate. Hypercalcaemia remains a concern.
- Lanthanum salts: Lanthanum carbonate (Renalzin, Bayer) is a newer phosphate binder used in humans and now in cats. This drug has also been shown to be effective at lowering phosphate and has the advantage of administration as a gel/foam.
- Sevelamer hydrochloride (Renagel) is a human drug with little current experience in cats. Potential side effects include vomiting and diarrhoea.

Use of calcitriol in CKD

As the kidneys convert vitamin D into it's active form (1,25-dihydrocholecalciferol) and vitamin D levels may be reduced by the increased PTH seen in CKD there is some logic in providing calcitriol supplementation. Calcitriol also inhibits PTH synthesis which we know is increased in cats with CKD. However, vit D supplementation will also increase serum calcium and could risk soft tissue mineralisation. Studies have not been encouraging in cats and so far evidence has not supported its use in cats with CKD. If used then serum calcium must be monitored closely.

Summary of phosphate restriction/hyperparathyroidism in CKD:

- Many cats with CKD (and particularly higher stage CKD) are hyperphosphataemic
- Some cats with normal serum phosphate and CKD still have elevated PTH

- Hyperparathyroidism may contribute to progression of renal disease
- Higher phosphate has been associated with reduced survival
- A phosphate restricted diet has been shown to result in longer survival times in cats with CKD
- Dietary restriction is the first line treatment, followed by intestinal phosphate binders
- Evidence to support the use of calcitriol is lacking but anecdotal reports suggest a possible benefit

Management of Proteinuria:

In the past significant proteinuria was only considered if the UPC was > 1.0. Current thinking concludes that lower levels of proteinuria remain significant. The majority of cats with CKD will have UPCs < 0.5. However, lower levels of proteinuria may be significant. As described above proteinuria likely has a role in the progression of CKD.
Proteinuria in cats with CKD has been shown to be predictive of survival (Syme et al 2006, King et al 2007). It is an independent risk factor i.e. independent of other significant factors such as creatinine level. Even UPCs of 0.2-0.4 had a significant effect on survival compared to UPCs of < 0.2. Therefore we should take seriously the IRIS Substage BP cases. Hypertension also has a relationship with proteinuria. Hypertensive cats are more likely to be proteinuric than normotensive cats, likely due to the glomerular hypertension transmitted. Cats with higher stage CKD are also likely to have higher UPCs.

- Cats with CKD commonly have UPCs < 0.5
- Proteinuria is related to survival, even at low levels
- Control of hypertension may reduce UPC
- Stage of disease is related to level of proteinuria (i.e. serum creatinine)

Which patients benefit from treatment of proteinuria?

Some cats with glomerulonephropathies will have severely elevated UPCs. These cats and cats with amyloidosis (even higher UPCs, often > 10) will undoubtedly benefit from treatment to reduced UPC. The IRIS recommendations state that cats with UPC >0.4 should be treated and if 0.2-0.4 then they should be monitored and the UPC re-checked within 2 months.

The use of ACE inhibitors

ACE inhibitors have a number of functions in the kidney including anti-proteinuric and beneficial haemodynamic effects.
ACE inhibitors:

- Lower glomerular capillary pressure by vasodilating the efferent arteriole
- Lower systemic blood pressure (moderately 10-20mmHg)
- Improve selectivity of the filtration barrier and therefore reduce proteinuria

- Vasodilate the efferent arteriole
- Inhibit other effects of Angiotensin II on mesangial cells and podocytes (negative effects such as contraction and proliferation)

Which cases benefit from treatment?

It has been shown that ACE inhibitors reduce proteinuria in cats with all UPC levels.
However 2 studies have **FAILED** to show a significant increase in survival in cats with CKD treated with benazepril.

NOT EVERY CAT WITH CKD NEEDS FORTEKOR! THINK BEFORE PRESCRIBING AND PRIORITISE OTHER THINGS IF NOT PROTEINURIC (e.g. diet, treatment of UTI)

The studies on benazepril showed the following:

- Benazepril reduced UPC in cats with CKD
- Cats with UPC > 1.0 had an improved appetite when treated with benazepril
- Benazepril did not improve survival

Therefore cats with elevated UPCs (proteinuric Substage UPC >0.4) should be treated with ACE inhibitors.
ACE inhibitors can also be used in hypertensive cats BUT not alone as treatment with amlodipine is required.

- CATS WITH UPC >0.4
- CATS WITH HYPERTENSION AND PROTEINURIA
- CATS WITH HYPERTENSION NOT ADEQUATELY CONTROLLED WITH AMLODIPINE

Management of Hypertension:

CKD is the most common cause of hypertension in cats and approximately 20-30% of cats with CKD are hypertensive. Due to the clinical effects and possible effect on progression (perhaps via proteinuria), hypertension should be managed.

Treatment of hypertension:

The main treatment of choice is:

Calcium channel blocker – AMLODIPINE

This drug vasodilates and is an effective treatment for hypertension. The dose is 0.625-1.25mg/cat/day. This used to mean 1/8 to ¼ of a 5mg tablet. **BUT NOW THE RE-FORMULATED TABLETS ARE AVAILABLE FROM SUMMIT PHARMACEUTICALS!**

These doses are unlikely to cause hypotension. The dose is not predictable between cats and requires re-assessment. Treatment requirement may change as disease progress e.g. renal disease.

Start with the lower dose and re-check BP in 1 week. If the cat is severely hypertensive (>200) with retinal damage then it is reasonable to use a higher dose whilst monitoring closely.
Additional treatments include:

ACE Inhibitors

Alone ACE inhibitors are unlikely to control hypertension. They may provide additional blood pressure control with amlodipine and should be used concurrently if the cat is proteinuric. Hypotension could occur with this combination.

Monitoring

After instituting anti-hypertensive treatment, cats should be assess after 1 week with regards SBP and if indicated ocular changes. If dose adjustments are required then the SBP should always be measured 1 week later. Ideally repeat biochemistry also at this point as changes in BP can affect the level of azotaemia. Once SBP is stable and controlled, then it can be checked after a further 1 month and then every 3 months.

See http://www.catprofessional.com/kidney.html for a great book for owners with information on CKD.

Feline Idiopathic Cystitis

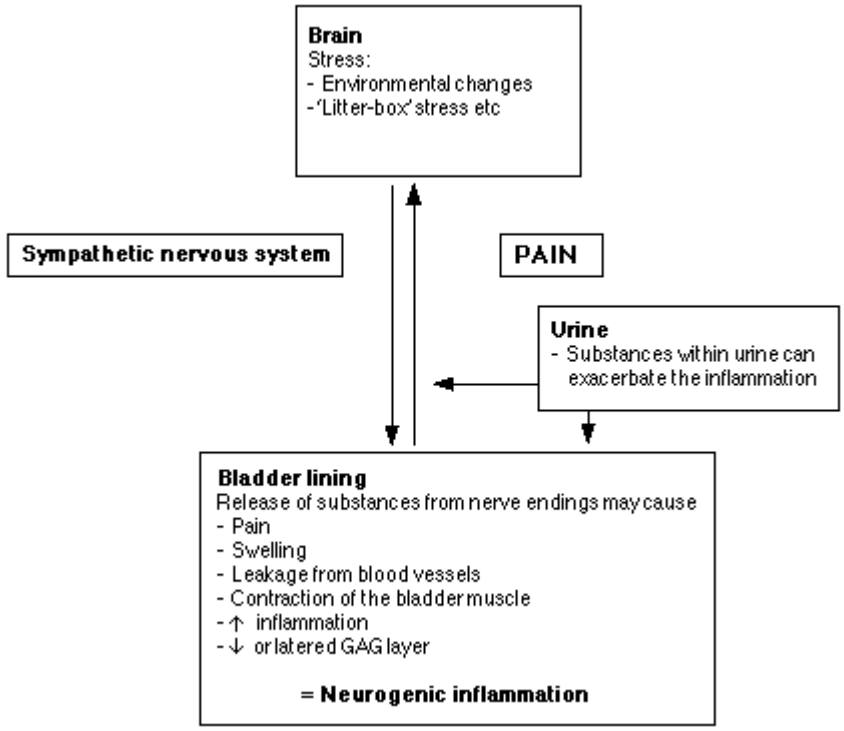
Which treatment should I use in cats with idiopathic cystitis?

Feline idiopathic cystitis (FIC) is the most common cause of lower urinary tract disease in cats, and is most frequent in middle aged, overweight cats that have sedentary lifestyle, restricted outdoor access, use indoor litter tray, eat dry diet and often in multicat household. It occurs with equal incidence in females and males but risk of obstruction greatest in males. Clinical signs are usually episodic, often associated with stress (e.g. change in routine or environment, addition of new family/pets to the household, a new cat in the neighbourhood), and are usually self limiting resolving within a few days

Theories of aetiopathogenesis

- Alteration between bladder neurons, protective glycosaminoglycan (GAG) layer lining bladder and compounds within urine (neurogenic inflammation)
- Uncoupling of hypothalamic-pituitary-adrenal axis resulting in inappropriate response to stress
    - FIC cats have increased tyrosine hydroxylase in locus coeruleus and hypothalamic nuclei
    - Chronic stress can increase tyrosine hydroxylase activity, increasing autonomic outflow – increased catecholamine concentration found in FIC cats
    - BUT FIC cats have reduced cortisol response & reduced adrenal volume so dissociation of SNS from HPA
- Obstruction may be result of inflammation, urethral plugs (= protein colloid matrix that ‘leaks’ from bladder wall as result of inflammation, +/- combined with crystals if get trapped in matrix) or urethral spasm

Figure 1 – Diagram showing how the nervous system may be able to induce/exacerbate inflammation in FIC (taken from Gunn-Moore DA 2001 UK Vet 6(5): 20- 26)



ie FIC results from complex interactions between the urinary tract, the endocrine system and nervous system

Management

If a diagnosis of FIC is made by excluding other signs, a range of management changes and treatments may be required. Like CKD management of FIC requires a multimodal approach and this is successful in the majority of cases (and backed up by the literature). Client education is vital to educate owners and emphasise the potential recurrent nature of the condition without appropriate management. Use of printed material can help motivated clients educate themselves about the condition (for example 'Caring for a cat with lower urinary tract disease' Caney and Gunn-Moore, see http://www.catprofessional.com/FLUTD.html).

1. **Identifying and reducing stressors**

The main aspect of managing FIC is taking time to discuss with owners their and their cats lifestyle and environment, and identifying particular stress factors that the cat could be facing, as more and more so, the evidence is that FIC is always associated with some sort of stress factor. Identifiying what these are, and managing those is therefore of fundamental importance, and this is often something done badly in practice, predominantly because it is a time consuming process.

Examples of common stressors include conflict with other cats either in the household, or in the neighbourhood, inadequate litter tray provision, inappropriate location of litter tray, changes in environment or diet or owner stress. Use of feline pheromone (Feliway™) may help in some cases but mainly as an adjunctive therapy. It is essential for underlying stressors to be identified and specifically managed. This aspect will be discussed in more detail within the lecture.

2. Increase water intake

<u>This together with reducing stress is the most important</u> aspect of treatment; ***increasing water intake*** to encourage production of ***more dilute urine***. The easiest way of doing this is to ***feed a wet diet*****.** Some recent dry diets have added sodium to encourage drinking but this is not as effective as feeding a wet diet. Other ways of encouraging water intake may also need to be instituted such:

- Add small amounts of water to the food
- Increase access to fresh water – think about bowls – multiple bowls with wide brims, filled to the top, ceramic or metal
- Have a bowl outside for rainwater
- Change water reguarly
- Try different types of bowls
- Offer distilled water
- Use a pet water fountain
- Leave some water in the bottom of the bath or sink
- Put a shallow bowl under a slowly dripping tap to provide constant fresh water
- Offer 'broths' – tuna in spring water with added water is often lapped up
- You can freeze cubes of chicken broth and pop one in the water bowl to flavour the water

Again, talking through all this with owners, and trialling different ways to suit the individual cat can be a time consuming process, and one which vets are not always very good at! It isn't enough to simply say to an owner, 'you need to increase your cats water intake', because they won't know how to do that successfully, and therefore the likelihood is the cats water intake won't increase. So, this is another area where nurses could make a huge difference to the management of these cases given some time to interact with the clients.

You can also check the effectiveness of the owners changes by measuring USG which should be less than 1.040.

3. Encourage normal urination

- Increase outdoor access
- Increase access to litter trays
- Frequently clean litter trays

- Try different types of litter
- Try covered litter trays
- Think about litter tray location

4. Weight reduction for overweight cats

- Reduce food intake – a wet diet helps here as it has fewer calories per gram
- Encourage exercise

See http://www.catprofessional.com/overweight.html for a great book for owners!

5. Medication

Note that multiple medications have been trialled and no drugs have been statistically proven to help prevent recurrence or reduce severity.

- ***GAG (glycosaminoglycan supplements).*** e.g. Cystease™. Various oral and parenteral GAG supplements are available with the assumption that they will help to replace the GAG layer lining the bladder. However, evidence for their use is lacking. They may also have anti-inflammatory/analgesic properties.
- ***Analgesia and anti-inflammatory drugs*** (e.g. meloxicam) are usually used in an attempt to reduce the discomfort associated with FIC. They are unlikely to fully relieve clinical signs in severe cases however can reduce the severity of signs if initiated at the first signs of development of FIC. NB Avoid NSAIDs if reduced renal function. Buprenorphine – given buccally – can be incredibly helpful in some cases and can be administered at home.
- ***Anti-spasmodics.*** Treatment for urethral spasm may reduce signs in some cats, particularly if urethral obstruction is occurring without evidence of physical obstruction. Treat with smooth muscle antispasmodics (Prazosin; Hypovase™ 0.25-1mg/cat PO q8-12hrs or phenoxybenzamine; Dibenyline™ 0.5-1 mg/kg PO q 12 hrs) or a combination of a smooth and skeletal anti-spasmodic (dantrolene; Dantrium™ 0.5-2mg/kg PO q 12hrs) may be most effective.

NB Drugs described above are not licensed for use in cats, refer to BSAVA formulary for further information and potential side effects

- Treatment of urethral plugs. Main aim is to reduce protein matrix (ie **treatment for FIC as above**). Crystalluria is not usually a problem on its own unless associated with development of urolithiasis. If continued clinical signs and significant struvite crystalluria together with persistent alkaline urine (NB post prandial alkaline tide occurs so urine tests should be on morning samples before feeding), then use of an **acidifying diet** may be considered. However, excessive acidification of urine can exacerbate FIC and long term use of acidifying diets can increase the risk of calcium oxalate urolithiasis, and result in metabolic acidosis, renal damage and hypokalaemia, so care needs to be taken. As always increasing water intake is the best thing!

- Remember :

IMPORTANT POINT:

Many NORMAL HEALTHY cats will have crystals in their urine. Cats have concentrated, often supersaturated urine.

When you identify crystalluria ask yourself the following questions:

- Is it a fresh or stored sample – the study above shows stored samples contain crystals commonly
- Does the cat have clinical signs/test results to suggest the crystalluria is significant? i.e. urolith identified on radiography, urethral plug that was gritty
- What other urinalysis results do I have? Check that pH, SG, bacterial culture and full sediment examination have been performed