THE GERIATRIC CAT: COMPLEX MANAGEMENT WITH MULTIPLE DISORDERS

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Elderly cats often present with multiple concurrent conditions. "Age-associated" or "age-appropriate" illnesses" we expect to see in older cats include problems related to the urinary tract (chronic kidney disease (CKD), pyelonephritis, calcium oxalate ureteronephroliths, bacterial cystitis), endocrine system (hyperthyroidism, diabetes mellitus, hyperaldosteronemia), arthritis, dental diseases and neoplasia. Constipation may become an ongoing concern. Certain infectious diseases become more likely in the older individual (e.g., FIP). A decline in functioning of the special senses occurs frequently and behaviour changes suggestive of cognitive dysfunction may be seen in some individuals.

Making management recommendations may be challenging as treatments, at first glance, may appear to be in conflict. Thankfully, the body is complex and all systems relate to one another. The most important therapeutic tools that must be incorporated in caring for every patient, especially those who are older, are optimizing comfort through analgesia, hydration and nutrition.

Chronic kidney disease Stage II-III with hyperthyroidism and degenerative joint disease

Concurrent renal dysfunction is fairly common in untreated hyperthyroid cats. It may be masked due to increased cardiac output and renal blood flow making it essential to monitor renal parameters during therapy. Similarly, hypertension may become evident only during the course of therapy. It is well recognized that amelioration of the hyperthyroid state by any method (i.e. medical therapy, 131I treatment or surgery) can result in decreased GFR, elevations in BUN and creatinine, and, in some cases, overt azotemia. The decline in GFR stabilizes by approximately four weeks (Adams, Becker, Boag, DiBartola, Graves).

Numerous studies have attempted to identify predictive parameters. GFR can be measured using plasma clearance of exogenous creatinine exo-iohexol or endo-iohexol; N-acetyl-beta-D-glucosaminidase index and retinol-binding protein have been assessed as possible biomarkers (Lapointe, Riensche, Slater). Symmetric dimethylarginine (SDMA) is a promising biomarker for estimating GFR (Hall). What role it, or any other early predictor, will play in the management of CKD in cats is unclear. Of the common clinical measures, cats with hypertension and/or an increase urine protein: creatinine ratio are more likely to develop problems while cats with elevated plasma globulins, a high usg and PCV are less likely to.

A practical approach is to treat cats with methimazole until the serum T4 is adequately controlled when the effect of permanent therapy can be assessed. If renal decline becomes overt after achieving euthyroidism, exogenous thyroid hormone can be supplemented to support the kidneys. A balance must then be struck between creating iatrogenic hyperthyroidism and maintaining renal function as iatrogenic hypothyroidism appears to contribute to azotemia and decreased survival.

As always, the treatment plan hinges on hydration, nutrition, and analgesia. In this scenario, maintaining euvolemia will be reflected in coat and stool character as well as subjective measures of well-being including grooming, interaction and posture. Daily subcutaneous fluids may be appropriate. Appetite may be negatively affected by renal disease from uremic toxins or, less likely, uremic gastritis. Cats with CKD are more likely to have gastric fibrosis rather than uremic gastropathy as has been reported in humans and in dogs. Appetite stimulation with mirtazapine (1.88 mg PO q48h) has been

shown to benefit cats with CKD. (Quimby) Interestingly, maropitant, while effective in palliating vomiting, did not significantly improve appetite or support weight gain in cats with Stage II and III CKD. (Quimby, Advanced Renal Therapies Symposium, March 2014)

Cats with untreated hyperthyroidism may have an increased appetite yet, due to increased metabolism, lose weight and muscle. Once euthyroid, their appetites generally normalize. Muscle catabolism results in reduced serum creatinine levels. The renal effects of endogenous protein breakdown have not been studied but may potentially be no different than those of dietary protein. Additionally, sarcopenia is associated with increased morbidity and mortality in cats. (Cupp, Freeman) This is of clinical relevance when we try to design the optimal nutritional regime for our older feline patients: protein and fat restriction may well be contraindicated. Especially if underweight, older cats will benefit from a more energy-dense, highly digestible diet to help offset these age-related digestive and metabolic changes. While it appears that some loss of weight is part of normal aging (Armstrong, Dora-Rose), is not caused by illness but that we may be able to address it nutritionally to some degree, potentially improving longevity as well as quality of life.

We would like to feed first cat, an elderly individual with Stage 3 renal disease, a protein-restricted diet suitable for renal insufficiency. Do all cats with renal disease have the same etiologic cause for their decline in renal function? Are they all at the same stage? Do they have identical nutritional requirements? Could this cat, perhaps, benefit from being fed a protein enhanced diet, a recuperative diet, a growth diet, a senior diet or a maintenance diet?

Protein: calorie malnutrition occurs when a cat is getting enough calories but not enough of them come from protein. As a result, there may or may not be weight loss, but there will be muscle wasting as well as a deterioration in the hair coat quality. Because protein is component in antibodies, immune function may be compromised; anemia may be exacerbated due to the lack of building components for hemoglobin; albumin levels may decrease and tissue healing may be affected. Protein is a preferred flavour, so if a cat is already inappetant, restricting protein may result in inadequate intake of all nutrients, and the protein intake may fall below that required for normal function.

As an *obligate* carnivore, if a cat doesn't get enough dietary protein to meet metabolic requirements, he must draw on endogenous, stored protein sources to meet those needs. Over months, cats can down-regulate their protein needs and switch to use other pathways, but in the short and intermediate term, muscle will be catabolized. The resulting muscle wasting and decreased mass reduces the serum level of creatinine (Cr) measured. This makes it difficult to know how much of a Cr decrease seen in a cat fed a restricted protein diet is from improvement in renal function and how much is because there is less functional muscle producing Cr.

Despite numerous experimental studies and clinical trials having been performed, questions about feeding protein to the cat with renal disease still remain. These include the following:

1. What is optimal amount of protein for a cat with CKD? How much restriction is necessary?
2. Do different types of kidney disease require different dietary therapies?
3. At what point in disease progression should protein restriction be implemented?
4. Does the type of protein fed make a difference?
5. Does every meal have to be restricted?
6. Is phosphorus restriction as, or more, beneficial than protein restriction in Stages 2 and 3?
7. Might some cats with advanced disease benefit from increased protein levels?
8. Should the diets of cats with proteinuria be protein restricted or enhanced?

Protein levels in “restricted” and “high” protein diets fall within the nutritional guidelines, merely at the low or at the high end of the range. Protein-restricted therapeutic diets are not all the same; there are some marked differences in their composition, not just in protein sources and quantities, but also in the calorie source, in their phosphorus, potassium, and sodium content. Table 2 compares reduced protein and phosphorus foods as of December 2012.

Dietary protein is not, in and unto itself, toxic to kidneys. Because of inherent progression of chronic renal insufficiency, IRIS staging focuses on factors which, when managed, are known to slow progression. These are: azotemia, metabolic acidosis, hyperphosphatemia, proteinuria and hypertension.

The effects of renal diets go beyond protein restriction, however. Different diets are variably supplemented with potassium and fatty acids, restricted in phosphorus. Every patient’s response to a given diet may differ and each cat should be rechecked to assess the effects and suitability of the recommended diet. (Table 1)

Addressing the **DJD** in a patient with CKD requires thought but is not insurmountable. The most common concern regarding drug therapy are side effects of using NSAIDs in a dehydrated patient and their effects on gastric mucosal health or on renal function. Opioids while safe are not a first drug of choice for cats with arthritic pain as they are not very effective for DJD. Pharmacokinetic data is lacking for safe, long-term use of many NSAIDs in cats. Carprofen half-life varies from nine to over 40 hours in cats. As most NSAIDs have long half-lives in cats when compared to other species, one precaution to avoid toxicity is to reduce the frequency of administration.

Metacam 0.5 mg/ml oral suspension is licenced specifically for the alleviation of inflammation and pain in chronic musculoskeletal disorders in cats in the EU. Pharmacokinetic studies as well as safety and efficacy studies have been performed to the satisfaction of the regulatory bodies. The registered dose is 0.1 mg/kg on the first day followed by 0.05 mg/kg orally once daily. This is the first NSAID licensed for long-term use in cats.

In one study, cats were treated for one month with meloxicam, clients felt that their cats were more willing to jump and the height of jumping increased during the study. Evaluation of the cats by the veterinarian at the end of the month showed a significant reduction of gait stiffness. Two studies have evaluated long-term safety of this agent in older cats; one concluded that this agent is safe, efficacious and palatable for OA pain at 0.01-0.03 mg/kg PO q24h for a mean treatment duration of 5.8 months; no deleterious effect on renal function was detected in cats studied. Gastrointestinal upset in 4% of cats was the only adverse effect noted (Gunew). The second, reviewed the medical records of cats over seven years of age treated for a minimum of 6 months with a daily maintenance dose of 0.02 mg/kg meloxicam and concluded that this dose does not hasten progression of renal disease in aged cats or aged cats with pre-existent stable IRIS stage 1-3 renal disease (Gowan).

A recently published study has shown that after oral administration, the major route of excretion of meloxicam in the cat is fecal and that the main pathway of biotransformation of meloxicam in the cat is oxidation. This is of importance because in cats, glucuronidation is limited as a metabolic pathway, whereas oxidation is not. Additionally, 21% of the recovered drug was eliminated in urine (2% as unchanged meloxicam, 19% as metabolites) and 79% in the feces (49% as unchanged meloxicam, 30% as metabolites). This is significant because it means that the product was already metabolized and inactivated by the time it reached the kidneys (Grude).

After reviewing 108 papers on NSAIDs including all of the studies to date on their use in cats the International Society for Feline Medicine (ISFM) and the American Association of Feline Practitioners (AAFP) made the following summary point statement in their document providing guidelines on the long-term use of NSAIDs in cats: "*to date, published studies of the medium-to long-term use of the COX-1 sparing drug meloxicam in older cats and cats with chronic kidney disease provides encouraging data that these drugs can be used safely and should be used to relieve pain when needed*"(Sparkes).

This document can be found at: www.catvets.com/professionals/guidelines/publications as well as at: www.isfm.net/toolbox/info_sheets/NSAIDs_guidelines.pdf with a client brochure regarding the safe use of NSAIDs in cats at: www.isfm.net/toolbox/info_sheets/NSAIDs_client%20leaflet.pdf.

A suitable protocol for a cat with pain from DJD might be baseline NSAID (such as meloxicam or robenacoxib) with intermittent use of an opioid (such as burprenorphine) when break-through pain is evidenced by a decrease in appetite or mobility or social withdrawal. A recent study by King et al has shown that robenacoxib (1.0–2.4 mg/kg PO q24h) is safe in cats with, or without, CKD when administered for DJD for 30 days.

Disease-Modifying Osteoarthritis Agents: Over the past two decades, research has been aimed at attempts to slow the progression of cartilage degradation as well as to promote rebuilding of healthy matrix. Products have been developed that, in research on humans and dogs, have been shown to be beneficial in enhancing hyaluronic acid production, inhibiting catabolic enzymes in osteoarthritic joints, and encouraging normalization of the synovial fluid and joint cartilage matrix. There is no evidence for efficacy in cats.

Studies using radio-labeled compounds have shown that 87% of orally administered glucosamine is absorbed and is incorporated into the cartilage matrix. Glucosamine provides raw materials for synthesis of glycosaminoglycans. Since chondrocytes obtain preformed glucosamine from the circulation (or synthesize it from glucose and amino acids), adequate glucosamine levels in the body are essential for synthesis of glycosaminoglycans in cartilage. Glucosamine is required for the production of hyaluronic acid by synoviocytes. *In vitro* studies indicate that administration of glucosamine may normalize cartilage metabolism and stimulates the synthesis of proteoglycans. In one study, glucosamine stimulated synthesis of glycosaminolgycans, prostaglandin and collagen by chondrocytes and fibroblasts, suggesting it may actually up-regulate their synthesis.

Chondroitin Sulfate (CS) is a long chain polymer of repeating disaccharide units. It is the predominant glycosaminolycan found in articular cartilage and can be purified from bovine, whale, and shark cartilage sources. Bioavailibilty studies in rats, dogs and humans have shown that 70% of orally administered CS is absorbed, some of it intact. Studies in rats and humans using radiolabeled CS have shown that CS reaches synovial fluid and articular cartilage. Hyaluronate concentrations and viscosity were increased, and collagenolytic activity was decreased, in the synovial fluid of human osteoarthritis patients treated with CS for 10 days.

Both oral preparations (Cosequin) and parenterally injected preparations (Adequan) have been shown to have therapeutic benefit in *in vivo* studies. One factor of note is that a polysulfated glycosaminoglycan, such as Adequan, is a heparin analog, resulting in a transient prolonged partial thromboplastin time. Avoid using it in cats with bleeding disorders or pre-operatively and do not use it concurrently with NSAIDs that have potent anti-thromboxane activity.

Diet: Weight loss should be encouraged in the obese, arthritic cat to reduce the pressure on joints. The addition of omega 3 fatty acids may be beneficial by blocking the production of prostaglandins from arachadonic acid in the inflammatory cascade. A therapeutic prescription diet for joint health (Hill's j/d, 505.7 kcal/cup) is available in both a canned and a dry formulation; to date the author is not aware of peer-reviewed papers on this diet. Royal Canin has developed Mobility Support (391 kcal/cup) for cats. These diets may not have sufficient protein for some older patients. A recently published study comparing this diet to the identical diet without the added high levels of eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA) and supplemented with green-lipped mussel (GLM) extract and glucosamine/chondroitin sulfate. Forty cats with no detectable systemic disease but with radiographic evidence of at least one affected joint and who showed an aversive response to manipulation of that joint were fed one of the two diets (control or test diet) for nine weeks. Cats on the test diet showed significantly greater improvements including an increased ability to jump, increased time spent eating, less time sleeping, increased playing and interacting with other pets. In an earlier study that looked at efficacy and tolerance of an extract of GLM in dogs with DJD, a similar subjective improvement was noted by clients whether the dogs were receiving placebo extract or GLM extract. Thus, dietary modulation might be one modality to include in the therapy of cats with DJD-associated pain (Lascelles).

Therapeutic exercise - Moderate exercise has benefits in maintaining range of motion in the face of joint capsule fibrosis, to maintain/build muscle mass and maintain/build healthy articular cartilage. In acute flare-ups, restricting activity may be warranted.

Additional therapeutic considerations – Environmental changes in the home as well as empathic awareness and changes in handling in a clinic setting should be instituted. Regular nail trimming helps by maintaining proper joint relationships. Ramps and steps to favourite sleeping spots may be helpful. Warm, soft, padded sleeping places for stiff, painful, possibly bony joints should be considered. Adding a litter box to reduce the distance between boxes may reduce accidents as well as encourage regular voiding and defecation. The rim of the box mustn't be too high, nor the opening into the box too small. It should be scooped several times a day to encourage use.

Acupuncture may be considered and can safely be combined with pharmacologic approaches. While efficacy has been shown for acupuncture in a few conditions in humans, there is no solid scientific evidence at the time of writing that clearly supports its efficacy in cats. Peer-reviewed publications for efficacy of laser therapy and stem cell therapy are also lacking.

Chronic kidney disease Stage II with diabetes mellitus and Inflammatory Bowel Disease

The challenges with these comorbidities are nutritional (protein quantity and source) and immunomodulation in the face of glycemic instability. Treatment with corticosteroids takes priority over glycemic effects because inflammation is recognized as a predisposing factor for susceptible individuals to develop diabetes. Franchini has shown at a molecular level that the inflammation induced by bacterial or viral infection can, via molecules recognized by toll gate receptors, damage endocrine pancreatic tissue. In some individuals IBD can be managed using chlorambucil and metronidazole without corticosteroids.

Feeding strategies for diabetes include a high protein, low carbohydrate diet or a high fiber diet. However, a diabetic cat can be controlled with insulin as long as the diet and treats fed remain consistent from day to day.

Neither carbohydrates nor dry extruded diets are cause of diabetes or obesity. However, exchanging dietary carbohydrate for protein appears to be useful for weight loss treatment and management of non-insulin dependent diabetes in cats.

In a prospective, randomized, double blinded 10-week study (Hall et al), 12 cats (7/12 obese) of whom six were newly diagnosed and six were poorly controlled diabetics evaluated standard maintenance diet vs. lower carbohydrate, higher protein (LCHP) diets. The cats ate dry or canned based on their preference. All were treated with glargine and assessed at weeks 1, 2, 4, 6, and 10 with fructosamine, BG curve and clinical signs. One cat from each diet group achieved remission by week 10. All cats improved clinically, increased weight and achieved good glycemic control. Those fed the LCHP had a significantly greater decrease in fructosamine. The conclusion, based on this small study was that using insulin, "frequent monitoring is key to achieving glycemic control in diabetic cats; potential benefits of dietary modification require further evaluation". The author summarizes all of the preceding studies and approaches: high fiber & low fat, high insoluble fiber vs. low fiber, LCHP canned, low carbohydrate diet vs. low carbohydrate diet plus acarbose, low carbohydrate & low fiber diet vs. moderate carbohydrate & high fiber diet. None of these approaches appears to make a meaningful difference in the small numbers of cats in each study.

Diet recommendations for a cat with a sensitive gastrointestinal tract and a diagnosis of "IBD" are either a limited antigen, a "hypoallergenic" or a hydrolyzed protein diet. Some cats may tolerate a highly digestible, low residue intestinal diet. Omega-3 fatty acids may be beneficial in small quantities. Unless the GI disease is extremely mild, this condition has dietary priority as the diabetes can be regulated on any diet if used consistently and protein restriction may be unnecessary or contraindicated.

As already mentioned, when making a nutritional recommendation, we are performing a feeding trial with one subject in it (n=1). Reassessment is very important to determine suitability. How is his weight? How is his coat? Does he eat with enjoyment or vigour? What are his stools like (moist logs or dry pellets, cow patties or coloured water)? How energetic is he since he has been on this diet? Has there been a change in his PCV and proteins? Have biochemistries or usg changed? Is he proteinuric and potentially protein deficient? What about his blood pressure?

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Table 1: FELINE REDUCED PROTEIN AND PHOSPHORUS DIETS

Listed in order of decreasing protein content – all non-acidifying

		Nutrients of Concern (/100 kcal)		
Product	**kcals/can or cup**	**Protein (g)**	**PO4 (mg)**	**Na (mg)**
Hill's j/d (5.5 oz can)	152	9.3	195	102
Hill's j/d (dry)	506	8.6	161	79
Science Mature Hairball (dry)	326	8.5	182	101
Hill's y/d (dry)	519	8.2	150	58
Hill's y/d (5.5 oz can)	188	8.2	141	58
Hill's g/d (5.5 oz can)	165	8.2	123	76
Hill's g/d (dry)	297	7.9	135	72
Purina NF (5.5 oz can)	193	7.7	110	50
Purina NF (dry)	398	7.2	100	50
Iams renal plus (dry)	514	7.1	93	87
Hill's l/d (dry)	505	7.0	163	65
Iams renal plus (6 oz can)	199	6.8	128	82
Hill's l/d (5.5 oz can)	183	6.7	145	43
Hill's k/d (dry)	492	6.6	114	56
Hill's k/d chicken (5.5 oz can)	183	6.5	85	68
Royal Canin hepatic (dry)	439	6.3	150	80
Royal Canin renal LP modified-P(pork) (dry)	428	6.2	80	70
Royal Canin hypoallergenic hydrolyzed HP (dry)	344	6.0	170	140
Royal Canin renal LP Modified-C (chicken) (dry)	358	5.7	120	80
Royal Canin renal LP modified (**3.0** oz can)	**97**	5.6	**90**	**60**
Royal Canin renal LP modified (6 oz can)	212	5.5	80	50
AAFCO minimum	**---**	**6.5**	**125**	**50**

As of December 2012

Note: manufacturers change diet compositions from time to time and diets differ in different parts of the world.