

Treatment Recommendations for CKD in Cats (2015)

All treatments for chronic kidney disease (CKD) need to be tailored to the individual patient. The following recommendations are useful starting points for the majority of cats at each stage. Serial monitoring of these patients is ideal and treatment should be adapted according to the response to treatment. Note that staging of disease is undertaken **following diagnosis of CKD** – an increased blood creatinine concentration alone is not diagnostic of CKD.

Some of the treatment recommendations are not authorised for use in all geographical regions and some may not be authorised for use in cats. Such recommended dose rates are therefore empirical. It is the treating veterinarian's duty to make a risk:benefit assessment for each patient prior to administering any treatment.

IRIS is an independent non-profit organization, supported by an annual grant provided by Elanco Animal Health, a division of Eli Lilly and Company.





Treatment recommendations for Cats with Chronic Kidney Disease

Stage 1 Feline patients:

1. Discontinue all potentially nephrotoxic drugs if possible.
2. Identify and treat any pre-renal or post-renal abnormalities.
3. Rule out any treatable conditions like pyelonephritis (any urinary tract infection should be regarded as a potential pyelonephritis and treated appropriately) and renal urolithiasis with radiographs and/or ultrasonography.
4. Measure blood pressure and urine protein to creatinine ratio (UP/C).

Management of dehydration:

In these patients urine concentrating ability may be somewhat impaired and therefore

- correct clinical dehydration/hypovolemia with isotonic, polyionic replacement fluid solutions (e.g., lactated Ringer's) IV or SQ as needed.
- have fresh water available at all times for drinking.

Systemic hypertension:

The blood pressure above which progressive renal injury may be induced is unknown. Our goal is to reduce systolic blood pressure to <160 mm Hg and to minimize the risk of extra-renal target organ damage (CNS, retinal, cardiac problems/damage). If there is no evidence of such damage but systolic blood pressure persistently exceeds 160 mm Hg, increasing the risk of this occurring, treatment should be instituted.

'Persistence' of increased systolic blood pressure should be judged on multiple measurements made over the following time-scales in these blood pressure substages:

- Hypertensive (moderate risk of future target organ damage) – systolic blood pressure 160 to 179 mm Hg measured over 1 to 2 months
- Severely hypertensive (high risk of future target organ damage) – systolic blood pressure \geq 180 mm Hg over 1 to 2 weeks.

If evidence of target organ damage exists, cats should be treated without the need to demonstrate persistently increased systolic blood pressure. Reducing blood pressure is a long term aim in managing the patient with CKD and a gradual and sustained reduction should be the goal, avoiding any sudden or severe decreases leading to hypotension.

A logical stepwise approach to managing hypertension is as follows:

1. Dietary sodium (Na) reduction - there is no evidence that lowering dietary Na will reduce blood pressure. If dietary Na reduction is attempted, it should be accomplished gradually and in combination with pharmacological therapy.
2. Calcium channel blocker (CCB), such as amlodipine (0.125 to 0.25 mg/kg once daily).

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3. Double the dose of amlodipine (0.25 to 0.5 mg/kg once daily).
4. Combine an inhibitor of the renin-angiotensin-aldosterone system (RAAS; either an angiotensin converting enzyme inhibitor [ACEI, such as benazepril] or an angiotensin receptor blocker [ARB, such as telmisartan]) and CCB treatment.

Note: Take care not to introduce CCB/RAAS inhibitor treatment to unstable dehydrated cats as glomerular filtration rate may drop precipitously if these drugs are introduced before the patient is adequately hydrated.

Monitoring response to antihypertensive treatment:

Hypertensive cats normally require lifelong therapy and may require treatment adjustments. Serial monitoring is essential. After stabilization, monitoring should occur at least every 3 months.

Systolic blood pressure <120 mm Hg and/or clinical signs such as weakness or tachycardia indicate hypotension, which is to be avoided.

Blood creatinine concentration – reducing blood pressure may lead to small and persistent increases in creatinine concentration (<45 µmol/l or 0.5 mg/dl increase), but a marked increase suggests an adverse drug effect. Progressively increasing concentrations indicate progressive kidney damage/disease.

Proteinuria:

Cats in Stage 1 with UP/C >0.4 should be investigated for disease processes leading to proteinuria (see 1 and 2 below) and treated with anti-proteinuric measures (see 3 and 4 below).

Those with borderline proteinuria (UP/C 0.2 to 0.4) require close monitoring (see 1 and 4 below).

1. Look for any concurrent associated disease process that may be treated/corrected.
2. Consider kidney biopsy as a means of identifying underlying disease (see Appendix and consult experts if unsure of indications for kidney biopsy).
3. Administer an RAAS inhibitor (ACEI or ARB) and feed a clinical renal diet.
4. Monitor response to treatment / progression of disease:
 - stable blood creatinine concentration and decreasing UP/C = good response.
 - serially increasing blood creatinine concentrations and/or increasing UP/C = disease is progressing.

Ordinarily therapy will be maintained lifelong unless the underlying disease has been resolved in which case dose reduction whilst monitoring UP/C might be considered.

Note:

- a. The use of an RAAS inhibitor is contraindicated in any animal that is clinically dehydrated and/or is showing signs of hypovolaemia. Correct dehydration before using these drugs otherwise glomerular filtration rate may drop precipitously.
- b. Cats with proteinuria and hypoalbuminemia likely share the same thromboembolic

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risk as dogs, but aspirin is difficult to use in cats to achieve a selective antiplatelet effect. A suggested dose rate if plasma albumin is below 20 g/l (2 g/dl) is 1 mg/kg every 72 hours.

Stage 2 Feline patients:

All of the above listed for Stage 1 plus any additional steps indicated.

1. Discontinue all potentially nephrotoxic drugs if possible.
2. Identify and treat any pre-renal or post-renal abnormalities.
3. Rule out any treatable conditions like pyelonephritis (any urinary tract infection should be regarded as a potential pyelonephritis and treated appropriately) and renal urolithiasis with radiographs and/or ultrasonography.
4. Measure blood pressure and urine protein to creatinine ratio (UP/C).
5. Consider feeding a clinical renal diet: this may be accomplished more easily early in the course of CKD, before inappetence develops.

Management of dehydration:

These patients have decreased urine concentrating ability and therefore

- correct clinical dehydration/hypovolemia with isotonic, polyionic replacement fluid solutions (e.g., lactated Ringer's) IV or SQ as needed.
- have fresh water available at all times for drinking.

Systemic hypertension:

The blood pressure above which progressive renal injury may be induced is unknown. Our goal is to reduce systolic blood pressure to <160 mm Hg and to minimize the risk of extra-renal target organ damage (CNS, retinal, cardiac problems/damage). If there is no evidence of this but systolic blood pressure persistently exceeds 160 mm Hg, increasing the risk of such damage, treatment should be instituted.

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If evidence of target organ damage exists, cats should be treated without the need to demonstrate persistently increased systolic blood pressure. Reducing blood pressure is a long term aim in managing the patient with CKD and a gradual and sustained reduction should be the goal, avoiding any sudden or severe decreases leading to hypotension.

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2. Calcium channel blocker (CCB) such as amlodipine (0.125 to 0.25 mg/kg once daily).
3. Double the dose of amlodipine (0.25 to 0.5 mg/kg once daily).
4. Combine an inhibitor of the renin-angiotensin-aldosterone system (RAAS; either an angiotensin converting enzyme inhibitor [ACEI, such as benazepril] or an angiotensin receptor blocker [ARB, such as telmisartan]) with the CCB

Take care not to introduce CCB/RAAS inhibitor treatment to unstable dehydrated cats as glomerular filtration rate may drop precipitously if these drugs are introduced before the patient is adequately hydrated.

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Hypertensive cats normally require lifelong therapy and may require treatment adjustments. Serial monitoring is essential. After stabilization, monitoring should occur at least every 3 months.

Systolic blood pressure <120 mm Hg and/or clinical signs such as weakness or tachycardia indicate hypotension, which is to be avoided.

Blood creatinine concentration – reducing blood pressure may lead to small and persistent increases in creatinine (<45 $\mu\text{mol/l}$ or 0.5 mg/dl increase), but a marked increase suggests an adverse drug effect. Progressively increasing creatinine concentrations indicate progressive kidney damage/disease.

Proteinuria:

Cats in Stage 2 with UP/C >0.4 should be investigated for disease processes leading to proteinuria (see 1 and 2 below) and treated with anti-proteinuric measures (see 3 and 4 below).

Those with borderline proteinuria (0.2 to 0.4) require close monitoring (see 1 and 4 below).

1. Look for any concurrent associated disease process that may be treated/corrected.
2. Consider kidney biopsy as a means of identifying underlying disease (see Appendix and/or consult experts if unsure of indications for kidney biopsy).
3. Administer an RAAS inhibitor (ACEI or ARB) and feed a clinical renal diet.
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Ordinarily therapy will be maintained lifelong unless the underlying disease has been resolved in which case dose reduction whilst monitoring UP/C might be considered.

Note:

- a. Use of an RAAS inhibitor is contraindicated in any animal that is clinically dehydrated and/or showing signs of hypovolaemia. Correct dehydration before using these drugs otherwise glomerular filtration rate may drop precipitously.
- b. Cats with proteinuria and hypoalbuminemia likely share the same thromboembolic risk as dogs, but aspirin is difficult to use in cats to achieve a selective antiplatelet effect. A suggested dose rate if plasma albumin is below 20 g/l (2 g/dl) is 1 mg/kg every 72 hours.

Reduction of phosphate intake:

Many cats in Stage 2 will have normal plasma phosphate concentrations but will have increased plasma PTH concentration. Evidence suggests that chronic reduction of phosphate intake to maintain a plasma phosphate concentration below 1.5 mmol/l (but not less than 0.9 mmol/l; <4.6 mg/dl but >2.7 mg/dl) is beneficial to patients with CKD.

The following measures can be introduced sequentially in an attempt to achieve this:

1. Dietary phosphate restriction (i.e., clinical renal diet therapy).
2. If plasma phosphate concentration remains above 1.5 mmol/l (4.6 mg/dl) after dietary restriction, give enteric phosphate binders (such as aluminium hydroxide, aluminium carbonate, calcium carbonate, calcium acetate, lanthanum carbonate) to effect, starting at 30-60 mg/kg/day in divided doses to be mixed with each meal (mixed with the food). The dose required will vary according to the amount of phosphate being fed and the stage of kidney disease. Treatment with phosphate binders should be to effect (as outlined above), with signs of toxicity limiting the upper dose rate possible in a given patient. Monitor serum calcium and phosphate concentrations every 4-6 weeks until stable and then every 12 weeks. Microcytosis and/or generalized muscle weakness suggests aluminium toxicity if using an aluminium containing binder – switch to another form of phosphate binder should this occur. Hypercalcaemia should be avoided – combinations of aluminium and calcium containing phosphate binders may be necessary in some cases.

Metabolic acidosis:

If metabolic acidosis exists (blood bicarbonate or total CO₂ <16 mmol/l) once the patient is stabilized on the diet of choice, supplement with oral sodium bicarbonate (or potassium citrate if hypokalaemic) to effect to maintain blood bicarbonate / total CO₂ in the range of 16-24 mmol/l.

Additional recommendation for Stage 2 patients:

If the patient is hypokalemic, then potassium gluconate or potassium citrate should be supplemented to effect (typically 1-2 mmol/kg/day).



Stage 3 Feline patients:

All of the steps listed for Stage 1 and 2 plus any additional steps indicated.

1. Discontinue all potentially nephrotoxic drugs if possible.
2. Identify and treat any pre-renal or post-renal abnormalities.
3. Rule out any treatable conditions like pyelonephritis (any urinary tract infection should be regarded as a potential pyelonephritis and treated appropriately) and renal urolithiasis with radiographs and/or ultrasonography.
4. Measure blood pressure and urine protein to creatinine ratio (UP/C).
5. Feed a clinical renal diet.

Management of dehydration:

These patients have decreased urine concentrating ability and therefore

- correct clinical dehydration/hypovolemia with isotonic, polyionic replacement fluid solutions (e.g., lactated Ringer's) IV or SQ as needed.
- have fresh water available at all times for drinking.

Systemic hypertension:

The blood pressure above which progressive renal injury may be induced is unknown. Our goal is to reduce systolic blood pressure to <160 mm Hg and to minimize the risk of extra-renal target organ damage (CNS, retinal, cardiac problems/damage). If there is no evidence of this but systolic blood pressure persistently exceeds 160 mm Hg, increasing the risk of extra-renal target organ damage, treatment should be instituted.

'Persistence' of increase in systolic blood pressure should be judged on multiple measurements made over the following time-scales in these blood pressure substages:

- Hypertensive (moderate risk of future target organ damage) – systolic blood pressure 160 to 179 mm Hg measured over 1 to 2 months.
- Severely hypertensive (high risk of future target organ damage) – systolic blood pressure \geq 180 mm Hg over 1 to 2 weeks.

If evidence of target organ damage exists, cats should be treated without the need to demonstrate persistently increased systolic blood pressure. Reducing blood pressure is a long term aim in managing the patient with CKD and a gradual and sustained reduction should be the goal, avoiding any sudden or severe decreases leading to hypotension.

A logical stepwise approach to managing hypertension is as follows:

1. Dietary sodium (Na) reduction – there is no evidence that lowering dietary Na will reduce blood pressure. If dietary Na reduction is attempted, it should be accomplished gradually and in combination with pharmacological therapy.
2. Calcium channel blocker (CCB) such as amlodipine (0.125 to 0.25 mg/kg once daily).

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3. Double the dose of amlodipine (0.25 to 0.5 mg/kg once daily).
4. Combine an inhibitor of the renin-angiotensin-aldosterone system (RAAS; either an angiotensin converting enzyme inhibitor [ACEI, such as benazepril] or an angiotensin receptor blocker [ARB, such as telmisartan]) with the CCB.

Note: Take care not to introduce CCB/RAAS inhibitor treatment to unstable dehydrated cats as glomerular filtration rate may drop precipitously if these drugs are introduced before the patient is adequately hydrated

Monitoring response to antihypertensive treatment:

Hypertensive cats normally require lifelong therapy and may require treatment adjustments. Serial monitoring is essential. After stabilization, monitoring should occur at least every 3 months.

Systolic blood pressure <120 mm Hg and/or clinical signs such as weakness or tachycardia indicate hypotension, which is to be avoided.

Blood creatinine concentration – reducing blood pressure may lead to small and persistent increases in creatinine concentration (<45 µmol/l or 0.5 mg/dl increase), but a marked increase suggests an adverse drug effect. Progressively increasing concentrations indicate progressive kidney damage/disease.

Proteinuria:

Cats in Stage 3 with urine protein to creatinine ratio (UP/C) >0.4 should be investigated for disease processes leading to proteinuria (see 1 and 2 below) and treated with anti-proteinuric measures (see 3 and 4 below).

Those with borderline proteinuria (0.2 to 0.4) require close monitoring (see 1 and 4 below).

1. Look for any concurrent associated disease process that may be treated/corrected.
2. Consider kidney biopsy as a means of identifying underlying disease (see Appendix and/or consult experts if unsure of indications for kidney biopsy).
3. Administer an RAAS inhibitor (ACEI or ARB) and feed a clinical renal diet.
4. Monitor response to treatment / progression of disease:
 - stable blood creatinine concentration and decreasing UP/C = good response.
 - serially increasing creatinine concentrations and/or increasing UP/C = disease is progressing.

Ordinarily therapy will be maintained lifelong unless the underlying disease has been resolved in which case dose reduction whilst monitoring UP/C might be considered.

Note:

- a. Use of an RAAS inhibitor is contraindicated in any animal that is clinically dehydrated and/or is showing signs of hypovolaemia. Correct dehydration before using these drugs otherwise glomerular filtration rate may drop precipitously.

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- b. Cats with proteinuria and hypoalbuminemia likely share the same thromboembolic risk as dogs, however, aspirin is difficult to use in cats to achieve a selective antiplatelet effect. A suggested dose rate if plasma albumin is below 20 g/l (2 g/dl) is 1 mg/kg every 72 hours.

Reduction of phosphate intake:

Evidence suggests that chronic reduction of phosphate intake to maintain a plasma phosphate concentration below 1.5 mmol/l (but not less than 0.9 mmol/l; <4.6 mg/dl but >2.7 mg/dl) is beneficial to patients with CKD.

A more realistic post-treatment target plasma phosphate concentration for cats at Stage 3 is <1.6 mmol/l (5.0 mg/dl).

The following measures can be introduced sequentially in an attempt to achieve this:

1. Dietary phosphate restriction (i.e., clinical renal diet therapy).
2. If plasma phosphate concentration remains above 1.6 mmol/l (4.6 mg/dl) after dietary restriction, give enteric phosphate binders (such as aluminium hydroxide, aluminium carbonate, calcium carbonate, calcium acetate, lanthanum carbonate) to effect, starting at 30-60 mg/kg/day in divided doses to be mixed with each meal (mixed with the food). The dose required will vary according to the amount of phosphate being fed and the IRIS stage. Treatment with phosphate binders should be to effect (as outlined above), with signs of toxicity limiting the upper dose rate possible in a given patient. Monitor serum calcium and phosphate concentrations every 4-6 weeks until stable and then every 12 weeks. Microcytosis and/or generalized muscle weakness suggests aluminium toxicity if using an aluminium containing binder – switch to another form of phosphate binder should this occur. Hypercalcaemia should be avoided – combinations of aluminium and calcium containing phosphate binders may be necessary in some cases.
3. Although there is evidence that judicious use of calcitriol prolongs survival in dogs at IRIS Stage 3, beneficial effects of ultra-low dose calcitriol have not yet been established in cats.

Metabolic acidosis:

If metabolic acidosis exists (blood bicarbonate or total CO₂ <16 mmol/l) once the patient is stabilized on the diet of choice, supplement with oral sodium bicarbonate, (or potassium citrate if hypokalaemic) to effect to maintain blood bicarbonate / total CO₂ in the range of 16-24 mmol/l.

Additional recommendations for Stage 3 patients:

1. If the patient is hypokalemic, then potassium gluconate or potassium citrate should be supplemented to effect (typically 1-2 mmol/kg/day).

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2. Consider treatment for anemia if it is affecting the patient's quality of life: typically this occurs when the PCV is <0.20 l/l (20%). Human recombinant erythropoietin is the most effective treatment but it is not approved for veterinary use: darbepoetin is preferable as it is less antigenic than epoetin alfa. Anabolic steroids are of no proven benefit and may be detrimental.
3. Treat vomiting / decreased appetite / nausea with a proton pump inhibitor (such as omeprazole) and an antiemetic (such as maropitant or ondansetron). However, further investigations are needed on the use of these and other drugs to determine whether they are useful for managing gastrointestinal disturbances in cats with CKD and uraemia.
4. Give appropriate maintenance fluids parenterally as necessary to maintain hydration (see Footnote).

Drugs that rely predominantly on renal function for their clearance from the body should be used with caution in patients in Stage 3 CKD. It may be necessary to adjust the dose of these drugs (depending on their therapeutic indices) to avoid accumulation.



Stage 4 Feline patients:

All of the steps listed for Stages 1, 2 and 3 plus any additional steps indicated.

1. Discontinue all potentially nephrotoxic drugs if possible.
2. Identify and treat any pre-renal or post-renal abnormalities.
3. Rule out any treatable conditions like pyelonephritis (any urinary tract infection should be regarded as a potential pyelonephritis and treated appropriately) and renal urolithiasis with radiographs and/or ultrasonography.
4. Measure blood pressure and urine protein to creatinine ratio (UP/C).
5. Feed a clinical renal diet.

Management of dehydration:

These patients have decreased urine concentrating ability and therefore

- correct clinical dehydration/hypovolemia with isotonic, polyionic replacement fluid solutions (e.g., lactated Ringer's) IV or SQ as needed.
- have fresh water available at all times for drinking.

Systemic hypertension:

The blood pressure above which progressive renal injury may be induced is unknown. Our goal is to reduce systolic blood pressure to <160 mm Hg and to minimize the risk of extra-renal target organ damage (CNS, retinal, cardiac problems/damage). If there is no evidence of this but systolic blood pressure persistently exceeds 160 mm Hg, increasing the risk of such damage, treatment should be instituted.

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If evidence of target organ damage exists, cats should be treated without the need to demonstrate persistently increased systolic blood pressure. Reducing blood pressure is a long term aim in managing the patient with CKD and a gradual and sustained reduction should be the goal, avoiding any sudden or severe decreases leading to hypotension.

A logical stepwise approach to managing hypertension is as follows:

1. Dietary sodium (Na) reduction - there is no evidence that lowering dietary Na will reduce blood pressure. If dietary Na reduction is attempted, it should be accomplished gradually and in combination with pharmacological therapy.
2. Calcium channel blocker (CCB) such as amlodipine (0.125 to 0.25 mg/kg once daily).
3. Double the dose of amlodipine (0.25 to 0.5 mg/kg once daily).

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4. Combine an inhibitor of the renin-angiotensin-aldosterone system (RAAS; either an angiotensin converting enzyme inhibitor [ACEI, such as benazepril] or an angiotensin receptor blocker [ARB, such as telmisartan]) with the CCB.

Note: Take care not to introduce CCB/RAAS inhibitor treatment to unstable dehydrated cats as glomerular filtration rate may drop precipitously if these drugs are introduced before the patient is adequately hydrated.

Monitoring response to antihypertensive treatment:

Hypertensive cats normally require lifelong therapy and may require treatment adjustments. Serial monitoring is essential. After stabilization, monitoring should occur at least every 3 months.

Systolic blood pressure <120 mm Hg and/or clinical signs such as weakness or tachycardia indicate hypotension, which is to be avoided.

Blood creatinine concentration – reducing blood pressure may lead to small and persistent increases in creatinine (<45 $\mu\text{mol/l}$ or 0.5 mg/dl increase), but a marked increase suggests an adverse drug effect. Progressively increasing concentrations indicate progressive kidney damage/disease.

Proteinuria:

Cats in Stage 4 with urine protein to creatinine ratio (UP/C) >0.4 should be investigated for disease processes leading to proteinuria (see 1 and 2 below) and treated with anti-proteinuric measures (see 3 and 4 below).

Those with borderline proteinuria (0.2 to 0.4) require close monitoring (see 1 and 4 below).

1. Look for any concurrent associated disease process that may be treated/corrected.
2. Consider kidney biopsy as a means of identifying underlying disease (see Appendix and/or consult experts if unsure of indications for kidney biopsy).
3. Administer an RAAS inhibitor (ACEI or ARB) and feed a clinical renal diet.
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Ordinarily therapy will be maintained lifelong unless the underlying disease has been resolved, in which case dose reduction whilst monitoring UP/C might be considered.

Note:

- a. Use of an RAAS inhibitor is contraindicated in any animal that is clinically dehydrated and/or is showing signs of hypovolaemia. Correct dehydration before using these drugs otherwise glomerular filtration rate may drop precipitously.
- b. Cats with proteinuria and hypoalbuminemia likely share the same thromboembolic risk as dogs, however, aspirin is difficult to use in cats to achieve a selective antiplatelet effect. A suggested dose rate if plasma albumin is below 20 g/l (2 g/dl) is 1 mg/kg every 72 hours.



Reduction of phosphate intake:

Evidence suggests that chronic reduction of phosphate intake to maintain a plasma phosphate concentration below 1.5 mmol/l (but not less than 0.9 mmol/l; <4.6 mg/dl but >2.7 mg/dl) is beneficial to patients with CKD.

A more realistic post-treatment target plasma phosphate concentration for cats at Stage 4 is <1.9 mmol/l (6.0 mg/dl).

The following measures can be introduced sequentially in an attempt to achieve this:

1. Dietary phosphate restriction (i.e., clinical renal diet therapy).
2. If plasma phosphate concentration remains above 1.9 mmol/l (6.0 mg/dl) after dietary restriction, give enteric phosphate binders (such as aluminium hydroxide, aluminium carbonate, calcium carbonate, calcium acetate, lanthanum carbonate) to effect, starting at 30-60 mg/kg/day in divided doses to be mixed with each meal (mixed with the food). The dose required will vary according to the amount of phosphate being fed and the stage of CKD. Treatment with phosphate binders should be to effect (as outlined above), with signs of toxicity limiting the upper dose rate possible in a given patient. Monitor serum calcium and phosphate concentrations every 4-6 weeks until stable and then every 12 weeks. Microcytosis and/or generalized muscle weakness suggests aluminium toxicity if using an aluminium containing binder – switch to another form of phosphate binder should this occur. Hypercalcaemia should be avoided – combinations of aluminium and calcium containing phosphate binders may be necessary in some cases.
3. Although there is evidence that judicious use of calcitriol prolongs survival in dogs at IRIS Stage 4, beneficial effects of ultra-low dose calcitriol have not yet been established in cats.

Metabolic acidosis:

If metabolic acidosis exists (blood bicarbonate or total CO₂ <16 mmol/l) once the patient is stabilized on the diet of choice, supplement with oral sodium bicarbonate (or potassium citrate if hypokalaemic) to effect to maintain blood bicarbonate / total CO₂ in the range of 16-24 mmol/l.

Additional recommendations for Stage 4 patients:

1. If the patient is hypokalemic, then potassium gluconate or potassium citrate should be supplemented to effect (typically 1-2 mmol/kg/day).
2. Consider treatment for anemia if it is affecting the patient's quality of life: typically this occurs when the PCV is <0.20 l/l (20%). Human recombinant erythropoietin is the most effective treatment but is not approved for veterinary use: darbepoetin is preferable as it is less antigenic than epoetin alfa. Anabolic steroids are of no proven benefit and may be detrimental.
3. Treat vomiting / decreased appetite / nausea with a proton pump inhibitor (such as omeprazole) and an antiemetic (such as maropitant or ondansetron). However, further investigations are needed on the use of these and other drugs to determine whether they are useful for managing gastrointestinal disturbances in cats with CKD and uraemia.

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4. Give appropriate maintenance fluids parenterally as necessary to maintain hydration (see Footnote).
5. Intensify efforts to prevent protein / calorie malnutrition. Consider feeding tube intervention (e.g., percutaneous gastrostomy tube).
6. Intensify efforts to prevent dehydration. Feeding tubes can be used to administer fluids as well as food.
7. Consider dialysis and/or renal transplantation.

Drugs that rely predominantly on renal function for their clearance from the body should be used with caution in patients in Stage 4 CKD. It may be necessary to adjust the dose of these drugs (depending on their therapeutic indices) to avoid accumulation.

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Appendix

Reasons for Undertaking Renal Biopsy

1. Renomegaly
2. CKD in a young patient
3. Persistent and severe proteinuria (UP/C>2.0) in a non-azotaemic patient
4. Worsening proteinuria in a CKD patient
5. Acute kidney injury, where renal biopsy may provide a prognostic indicator



Footnote

Maintenance fluids to maintain hydration status are low in sodium (30-40 mmol/l) and ideally have added potassium (about 13 mmol/l) to ensure daily requirements for fluid and electrolytes are met (e.g. Normosol-M® or 5% Dextrose plus 0.18% NaCl with added KCl)



Disclaimer

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