Anatomy and Physiology

The role of the kidneys is to filter the blood through the glomerulus to form filtrate. The filtrate is then reabsorbed along the nephron until the remainder comprises of compounds (electrolytes) that are surplus to the requirements of the animal. The kidney tubules are able to respond to these high levels of electrolytes/compounds by excreting them in greater amounts. This is how the kidneys play a major role in the homeostasis. The kidneys are also responsible for the water balance of the animal and they will vary the excretion of water in relation to the hydration status of the animal.

The functions of the kidneys are to maintain the volume and composition of plasma, regulate water, ion and pH levels, retain nutrients and excrete waste, toxins and excess electrolytes. The kidneys achieve these functions via; glomerular filtration, solute reabsorption, tubular secretion, water balance and acid-base regulation.
When there is insufficient filtering of the blood by the kidneys then there becomes a build-up of nitrogen containing products (urea and creatinine). This is known as azotaemia. It may occur as a result of acute or chronic kidney disease. There are 3 categories of azotaemia depending on the cause:

- **Pre-renal causes:**
  - Reduced renal blood flow
  - Excessive vasoconstriction
  - Pre-renal azotaemia resolves when the inciting cause is removed

- **Intrinsic renal causes:**
  - Extension of pre-renal
  - Infectious causes
  - Toxins
  - Systemic disease

- **Post renal causes:**
  - Obstruction or diversion of urine
  - Restoration of urine flow resolves azotaemia unless prolonged obstruction

The RIFLE system

In 2002 the Acute Dialysis Quality Initiative was formed with an aim of developing guidelines for the treatment and prevention of AKI in humans. They created the RIFLE system as a criteria for defining AKI. The difficulty that we have in the veterinary world is that we often don’t know baseline numbers for our patients so it can make it difficult to select an appropriate category and thus stage.

<table>
<thead>
<tr>
<th>Category</th>
<th>Biochemistry</th>
<th>UOP</th>
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<tbody>
<tr>
<td>Risk</td>
<td>Cr ↑ x 1.5</td>
<td>&lt; 0.5 ml/kg/hr x 6 hours</td>
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<tr>
<td></td>
<td>GFR ↓ 25%</td>
<td></td>
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<tr>
<td>Injury</td>
<td>Cr ↑ x 2</td>
<td>&lt; 0.5 ml/kg/hr x 12 hours</td>
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<tr>
<td></td>
<td>GFR ↓ 50%</td>
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<tr>
<td>Failure</td>
<td>Cr ↑ x 3</td>
<td>&lt; 0.3 ml/kg/hr x 24 hours</td>
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<tr>
<td></td>
<td>GFR ↓ 75%</td>
<td>Anuria for 12 hours</td>
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<tr>
<td>Loss</td>
<td>Persistent AKI</td>
<td>Complete loss of function &gt;4 weeks</td>
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<tr>
<td>End Stage</td>
<td>➢ 3 months</td>
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What is AKI?

Acute kidney injury (AKI) is characterised by a rapid loss of nephron function. (Previously known as acute renal failure – ARF). AKI results in azotaemia, fluid retention, uraemic toxin retention, electrolyte imbalances and acid base abnormalities.

There are 4 phases to AKI:
- Initiation phase – there is a period of renal injury. Intervention may prevent progression to further injury. Changes in USG and GFR are not evident at this stage.
- Extension phase – cellular injury progresses to cellular death.
- Maintenance phase – irreversible renal damage has occurred.
- Recovery phase – This takes weeks to months.

History

There is often no gender, age or sex predisposition. Signs may develop over hours to days. Owners may report:
- Depression, lethargy
- Anorexia, vomiting, diarrhoea
- Change in urine production
- Exposure to toxins
- Recent or concurrent disease

Clinical Examination

In cases of AKI body condition will often be normal. On abdominal palpation kidneys may feel enlarged. Patients are likely to present with signs of dehydration, hypovolaemia, depression/lethargy, ureamic breath and oral ulceration. Blood pressure should be assessed on presentation.

Laboratory findings

SERUM BIOCHEMISTRY
- Azotaemia – characterised by an increased BUN and increased creatinine
- Normal to increased K+
- Hyperphosphataemia
- Hypocalcaemia

HAEMATOLOGY
- PCV normal to elevated
- Leukogram
- Serology for leptospirosis

URINALYSIS
- Dilute urine
  - USG <1.030 in dogs
  - USG <1.035 in cats
  - Isosthenuria 1.007 – 1.015
- Renal tubular necrosis
  - Glucosuria
  - Haematuria
  - Proteinuria
- Calcium oxalate crystals
- Casts
- Urine sample collection for culture and sensitivity

Imaging

- Radiographs – you may see normal to large kidneys. If uroliths are present then then they may show up on the radiograph.
- Ultrasound – again normal to large kidneys may be seen. You may also have loss of corticomedullary distinction and a hyperechoic band at the corticomedullary junction is visible in some ethylene glycol toxicity patients.
- CT – Care should be taken with angiography as the drugs can be nephrotoxic.
- Depending on the suspect cause of the AKI then a decision may be made to biopsy the kidney(s).

Treatment

Once a diagnosis of AKI is made then it is essential that treatment is started promptly.

Intravenous fluid therapy – dehydration should be corrected. Patients presenting with hypovolaemia should have fluids boluses given to address the volume depletion. Fluid choice will be made based on the electrolytes and acid base status of the patient. Any ongoing losses should be met.

Management of electrolytes – the most common derangement in electrolytes that will be seen is potassium. In some cases of AKI potassium can rise quickly and become life threatening. This electrolyte should be monitored closely and efforts should be made to lower it with the use of fluids, insulin and glucose. Sodium and calcium should also be monitored closely and addressed as necessary.

Management of acid base – the most common acid base derangement seen is a metabolic acidosis. With appropriate fluid therapy and treatment this will often correct itself. In severe cases when the pH is <7.2 then sodium bicarbonate should be considered.

Monitoring urine output – a urinary catheter with a closed collection system should be placed so that urine output can be managed and fluid rates can be adjusted as necessary. Ideally patients should be producing 1-2ml/kg/hr. The definition of oliguria is 0.27 – 0.5 ml/kg/hr and anuria < 0.27 ml/kg/hr.
Treatment of vomiting – the case vet may wish to administer gastrointestinal protectants and anti-emetics. Patients can develop GI ulceration from the mouth through to the intestines. The build-up of toxins and azotaemia can lead to nausea and vomiting.

Treatment of hypertension – blood pressure should be monitored closely and it may be necessary to administer hypertensive drug therapy to help prevent the complications associated with hypertension such as retinal detachment and organ damage.

**Specific treatment:**

**Ethylene Glycol Toxicity**
- Emesis if know toxicity within 3 hours
- Administration of ethanol
- Diuresis with fluids
- Haemodialysis

**Leptospirosis**
- Appropriate antibiotic therapy
- Barrier nurse precautions
- Any animal that presents with elevated liver enzymes and azotaemia should be barrier nursed as precaution until proven leptospirosis negative
  - Zoonosis
  - Care with urine in particular

**Pyelonephritis**
- Appropriate antibiotic therapy
- Select non-nephrotoxic antibiotics
- Prolonged course antibiotics

**Nursing Considerations**

Management of IV catheter and central line – These should be checked twice daily for signs of inflammation or infection. Central lines should be managed wearing sterile gloves.

CVP – We can use readings to monitor trends. A normal reading would be between 0-10 cmH20. <0 = hypovolaemia and >10 = hypervolaemia. CVP can be useful in guiding our decision with regards to fluid therapy.

BP- We can select NIBP vs IBP depending on how sick the patient is and whether we can get arterial access. We should aim for a normal reading of 120/80 (MAP 100). If cases of hypertension we may need to consider antihypertensive medications.

Management of urinary catheter- We can use a closed collection system to measure UOP. We should aim for a UOP of 1-2ml/kg/hour. We can use the UOP to help us adjust our fluid therapy.

Monitoring of acid base - Every 4 hours – daily depending on the patients status and how deranged the parameters are. If we are administering any therapy then we need to ensure that we are monitoring it to avoid over-supplementation.
Adjusting fluid therapy – We should try to match ins with our outs (see urinary catheter management above) We need to be monitoring for fluid overload in oliguric and anuric patients.

Administration of medications – The IV route is preferred due to slow absorption in dehydrated patients.

Monitoring of pain – We can use pain scoring systems such as the modified Glasgow composite pain score (dogs) or the Colorado cat pain score system (cats). Nephrotoxic drugs such as meloxicam should be avoided.

Blood sampling - Serial measurements of biochemistry should be taken every 24-48 hours to assess azotaemia. If there is a central line present then samples can be taken from here.

Nutrition – Patients with AKI may require assisted feeding. We could consider appetite stimulants or naso-oesophageal feeding tube placement. We should aim to feed the patient’s RER in the format of ideally a low protein diet. We can start with tempting the patient to eat anything.

**Oliguria and Anuria**

This occurs when the patient is adequately hydrated and their UOP is:

- <0.27ml/kg/hour – Anuria
- 0.27 – 0.5ml/kg/hour – Oliguria

UOP should be closely monitored using a closed fluid system and the patient can be frequently weighed. Some veterinary surgeons will choose to administer either furosemide or mannitol to increase UOP. If measures are not successful in restoring urine output within 6 – 12 hours the next step would be dialysis.

There are two types of dialysis that can be considered. Peritoneal dialysis or haemodialysis. Continuous renal replacement therapy (CRRT) is the gold standard treatment. It is expensive and is only carried out in specialist centres. Patients may require more than one round and the underlying cause of the AKI must be treated.

**Home Care**

Animals must not be discharged until renal function is normal. The ongoing management at home will depend upon the underlying cause. If there is an infectious agent involved then the patient is likely to require a long course of antibiotics. Future exposure to nephrotoxins should be avoided. Patients should have free access to water at all times. Animals with residual renal dysfunction should be managed as chronic renal failure.
Prognosis

AKI may take days to weeks to months to resolve

- Some recent literature documents mortality rates in dogs as 60% and cats 50%.
- Patient’s with polyuria have a better outcome than those with oliguria and anuria
- Recovery is characterised by:
  - decreasing BUN
  - normalisation of electrolytes and acid-base
  - improvement in urine concentration