Acute kidney injury in cats: part 2 – management

The approach to management of acute kidney injury (AKI) in cats is to maintain renal perfusion and oxygen delivery, maintain urine output and address any secondary complications. Underlying causes of AKI should be identified and specific treatment instigated. General management may include appropriate fluid therapy, diuretic administration, correction of electrolyte and acid-base abnormalities and management of anaemia, hypertension and nausea. Response to treatment can be monitored through measuring urine output and assessing hydration status. The prognosis for AKI is guarded and approximately 50% of cats that survive will have persistent chronic kidney disease post-recovery.

Key words: Cats, acute kidney injury, management

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Natalie is passionate about everything feline but her research interests include feline geriatric medicine and in particular feline CKD and methods of evaluating renal function. Natalie was awarded the International Renal Interest Society (IRIS) award in 2013 for recognition of her outstanding contribution to clinical research in veterinary nephrology.

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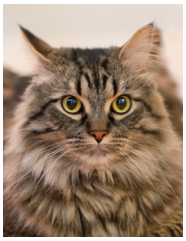
Introduction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Part one of this two-part series on acute kidney injury (AKI) discussed the aetiology and diagnosis of this condition in cats. In this article the management is covered in detail, along with the prognosis for affected cats.

General management

The management goal of acute kidney injury (AKI) is firstly, to prevent further renal damage by identifying and treating any primary underlying cause appropriately, and secondly, to enhance renal cellular recovery. Box 1 summarises the general management of AKI in cats.

Potentially treatable underlying diseases include pyelonephritis, urinary tract obstruction or lymphoma. If the cat has a known very recent history of toxin ingestion, then inducing emesis or the administration of activated charcoal as an absorbent may be beneficial. The Veterinary Poisons Information Service (VPIS) provides up to date information regarding management and are contactable 24 hours a day (www.vpisglobal.com), telephone number 0207 3055055. Box 2 outlines the management of ethylene glycol toxicity.

The key management strategies for AKI patients include:

- Maintaining renal perfusion and oxygen delivery
- Maintaining urine output
- Addressing secondary complications of AKI.

Standard treatment

Fluid therapy

Fluid volume status should be addressed first and foremost. AKI patients can range from dehydrated or hypovolaemic to fluid volume overloaded. Dehydrated or hypovolaemic patients should receive appropriate fluid administration to correct any deficit and become volume replete. Dehydration or hypovolaemia can result in reduced renal blood flow (RBF) and consequently decreased glomerular filtration rate (GFR) and urine formation, and contribute to further renal injury. In patients with pre-renal AKI, the changes are potentially reversible if the haemodynamic abnormalities are identified and corrected quickly. Care must be taken with fluid administration as overzealous administration can result in fluid volume overload particularly in oliguric or anuric patients. In human patients, studies have shown increased mortality rates in patients with liberal fluid administration compared to those with more restrictive plans (Prowle *et al.* 2010). Drip pumps and syringe drivers are invaluable for delivering accurate fluid volumes to cats (Figure 1). Fluid therapy is also important in correcting electrolyte imbalance and acid-base disorders.

Compound sodium lactate (Hartmann's, lactated Ringer's) would be an appropriate fluid to administer in most cases. Physiologic saline (0.9% NaCl) may be more suitable for patients with hypochloraemia, hyponatraemia or severe secondary hyperkalaemia. Fluid resuscitation in hypovolaemic or hypotensive cats should involve a fluid bolus of 10-15ml/kg administered over 10–20 mins. The cat should then be

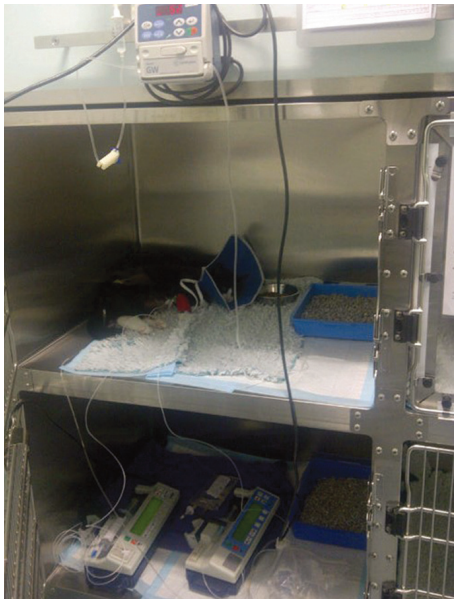


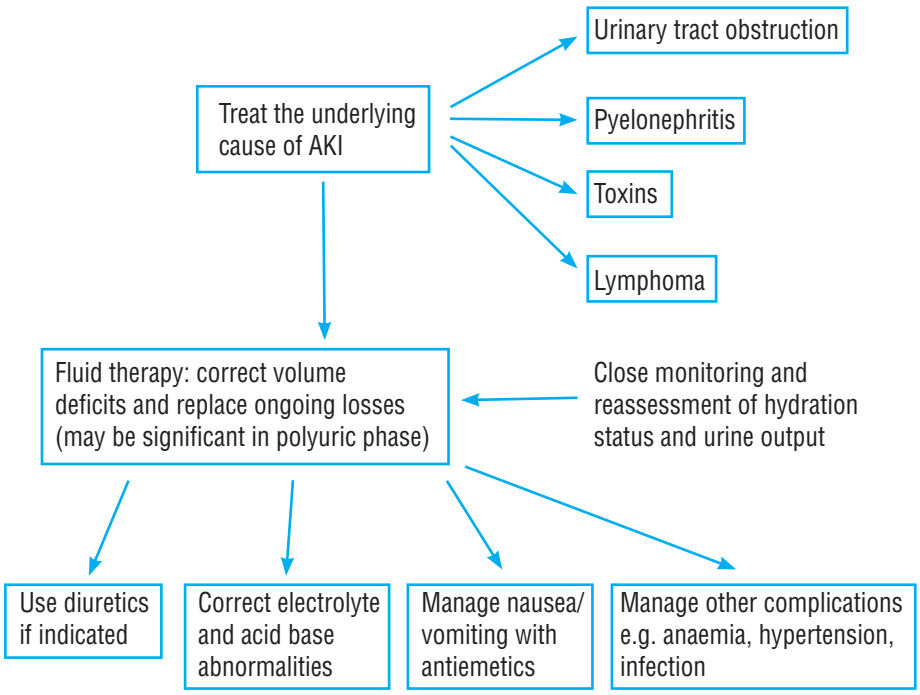
Figure 1: Drip pumps and syringe drivers are invaluable for ensuring accurate fluid delivery

reassessed and boluses repeated until the cat becomes haemodynamically stable. Ongoing fluid rates should take into account fluid losses such as urine output, vomiting or diarrhoea. In anuric patients, fluid rates should be carefully considered and it is recommended to replace insensible losses (respiration and faeces) only, which are approximately 22ml/kg/day.

Compromised renal tubules are unable to respond to any initial increase in GFR and tubular cell damage can also lead to solute loss. Therefore, there is an initial period of polyuria and solute diuresis in the early recovery stages of AKI. This can be challenging for clinicians to manage as large volumes of IV fluids are required and there may be significant electrolyte losses resulting particularly in hypokalaemia.

Once the patient becomes stable and the azotaemia has either resolved or stabilised, fluid therapy can be gradually tapered ensuring there is a corresponding decrease in urine output and no increase in creatinine concentration.

Box 1: Flow chart showing general approach to AKI in cats



Box 2: Treatment protocols for ethylene glycol toxicity

Ethanol is one of the traditional treatment options for ethylene glycol toxicity in cats. Its mode of action is to compete with ethylene glycol for metabolism by alcohol dehydrogenase which has a greater affinity for ethanol than ethylene glycol. It is only considered to be effective if administered within 12 hours of ingestion. It is important to monitor mentation of cats which should be sedated but not comatose. The following protocol can be used for administering ethanol to exposed cats:

Dilute ethanol to 20% solution (200mg/ml) in 0.9% NaCl

Day 1: Give 5ml/kg IV at 0, 6, 12, 18 and 24 hours

Day 2: Give 5ml/kg IV at 8, 16 and 24 hours

Day 3: Give 5ml/kg IV at 8 hours (final treatment)

Fomipazole (or 4-methylpyrazole) is an alcohol dehydrogenase inhibitor which prevents metabolisation of ethylene glycol. Unfortunately, to be effective, it must be administered within 3hours of ethylene glycol toxicity (Connally *et al.* 2010). It is also very expensive and not widely available within the UK. Its use has been reported in a case series of 3 cats in USA using the following dosage (Tart & Powell 2011):

Initial dose - 125mg/kg IV

12, 24, 36hrs following initial dose – 31.25mg/kg IV

Monitoring urine output

Urine output should be monitored as accurately as possible, particularly in response to fluid therapy. This should ideally be achieved by placement of a urinary catheter and a closed collection system. Urinary catheters should be placed once the cat is stabilised under a short general anaesthetic. General anaesthesia will facilitate catheter placement by inducing appropriate muscle relaxation. It may be possible to quickly pass a urinary catheter in a collapsed cat following opioid analgesia. Urinary catheter placement without a collection system should be avoided due to the risk of development of urinary tract infection. There is also a risk of iatrogenic urinary tract infection associated with catheter placement and management of the catheter should be performed as aseptically as possible. If placement of a urinary catheter is not possible, then collecting and weighing naturally voided urine can provide some estimate (1g = 1ml), as can measurement of urine in the bladder using ultrasound. Normal urine output in a healthy animal not on fluid therapy would be 1-2ml/kg/hr. Oliguria is defined as urine production <1ml/kg/hr and anuria as zero urine production. Once normal urine output is achieved and further monitoring is not required, the urinary catheter should be removed as its presence will increase the risk of urinary tract infection.

Urinary tract obstruction

If a urinary tract obstruction or rupture is identified then placement of a urinary catheter or surgical intervention may need to be considered. Ureteral obstructions can be managed with placement of ureteral stents although there are limited centres offering this currently.