

Guidelines for management of

Acute Renal Failure (Acute Kidney Injury)

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DISCLAIMER: These guidelines were produced in good faith by the author(s) reviewing available evidence/opinion. They were designed for use by paediatric nephrologists at the University Hospital of Wales, Cardiff for children under their care. They are neither policies nor protocols but are intended to serve only as guidelines. They are not intended to replace clinical judgment or dictate care of individual patients. Responsibility and decision-making (including checking drug doses) for a specific patient lie with the physician and staff caring for that particular patient.

Guidelines on management of Acute Renal Failure (Acute Kidney Injury)

Definition of ARF (now referred to as AKI)

- Acute renal failure is a sudden decline in glomerular filtration rate (usually marked by rise in serum creatinine & urea) which is potentially reversible with or without oliguria.
- Oliguria defined as urine output $<300\text{ml/m}^2/\text{day}$ or $<0.5\text{ ml/kg/h}$ ($<1\text{ ml/kg/h}$ in neonates).
- Acute on chronic renal failure suggested by poor growth, history of polyuria and polydipsia, and evidence of renal osteodystrophy

However, immediately after a kidney injury, serum creatinine & urea levels may be normal, and the only sign of a kidney injury may be decreased urine production. A rise in the creatinine level can result from medications (e.g., cimetidine, trimethoprim) that inhibit the kidney's tubular secretion. A rise in the serum urea level can occur without renal injury, such as in GI or mucosal bleeding, steroid use, or protein loading.

Aetiology

AKI can be oliguric or nonoliguric, have a rapid or slow rise in creatinine levels, and may have qualitative differences in urine solute concentrations and cellular content. The reason for this lack of a uniform clinical presentation is a reflection of the variable nature of the injury.

Pre-renal	Renal (intrinsic)	Post-renal
Hypovolaemia Peripheral vasodilatation Low cardiac output Renal vessel occlusion Drugs Hepatorenal syndrome Increased intra-abdominal pressure	Circulatory insufficiency Nephrotoxins Renal diseases (GN, IN) Myo/haemoglobinuria Tumour infiltrate/lysis Intratubular obstruction Iatrogenic Vasculitis/ vascular lesions	Posterior urethral valves Blocked catheters/ureters Obstructions by other factors Trauma Stones Neurogenic bladder

History

Current urinary output/colour, urinary stream, history of: hypovolaemia, recent drugs (prescribed or recreational), fever, diarrhoea, rash, arthropathy, URTI, previous UTIs, urinary symptoms, recent wt, wt loss, H/S/O above aetiology, features S/O acute on chronic renal failure & family history of renal problems

Examination

Vitals	Systems
Airway Breathing - respiratory rate, resp. distress (colour & oxygen saturation), temperature Circulation - HR, BP *, dehydration hypo/hypervolemia, Chart Weight and height	Abdomen - mass, palpable kidneys/distended bladder, bruit CVS - HR, signs of heart failure, raised JVP RS - tachypnoea, signs of pulmonary oedema Skin - rash, petechiae, purpura Eyes - uveitis, optic fundi, hypertensive changes CNS -drowsiness, confusion, focal deficits, ?hypocalcaemia Joints - Arthropathy

*Blood pressure should not be viewed in isolation - hypertension in the presence of cool peripheries suggests intravascular depletion whilst hypertension with warm peripheries suggests fluid overload. A low BP implies shock.

Investigations-Use clinical judgement

Attend to life-threatening features first

- Any features of pulmonary oedema - PICU referral, blood gas, CXR
- Urgent U&E for hyperkalaemia

All patients should have the following investigations:

Blood	Urine	Other
U&E, creatinine, bicarbonate, Ca, PO ₄ , Mg, alkaline phosphatase, albumin, LFTs FBC, clotting screen, osmolality Blood culture, throat swab and CRP Urate, LDH, CK, ESR, (PTH) Complement (C3, C4, C3 nephritic factor), Immunoglobulins, ASOT, anti-DNAse B, ANA, dsDNA, anti-GBM abs, ANCA, Auto-immune profile, lipid profile	Gross examination Urinalysis M,C&S Osmolality Urea and electrolytes Prot:creat ratio Calculate FeNa	Renal US scan -urgent or within 24 hours (large bright kidneys suggests an acute process; small kidneys suggest CRF) Chest X-ray

Other additional investigations are dictated by clinical presentation:

For suspected HUS:

- Blood film, Group and save or crossmatch, VTEC serology
- Stool culture

If D+ve HUS, consider other aetiology (T antigen *Strep. pneumoniae*, SLE, Factor H deficiency)

Others:

- eNA, anti-cardiolipin antibodies, Hepatitis screen, urine for eosinophils (haematology)
- Doppler, MCUG, MAG3, DMSA, wrist X-ray, CT scan
- **A renal biopsy is indicated as soon as possible where renal function is deteriorating and the aetiology is not certain.**

Urinary abnormalities in ARF

	Pre-renal	Intrinsic Renal	Post-renal
Urine output	Oliguria	Oliguria-polyuria	Variable
U Osmolality	>500(>350 newborn)	< 300 (<300 newborn)	< 350(<300 newborn)
U Sodium	<10 (<20 newborn)	> 40	> 40
FeNa (%)	<1 (< 2.5 newborn)	>2 (>3 newborn)	<2
Blood	Marked urea increase compared to creatinine	Low calcium, elevated PO ₄ ± potassium, Creatinine increases by 45-130µmol/L/day	Hyponatraemia, Hyperkalaemia, hyperchloraemic acidosis

Urinary sediments in Intrinsic (Renal) ARF

RBCs & casts	Bland/scant	Epithelial/WBCs	Crystalluria
GNs- proliferative GN/ small vessel vasculitis, rarely interstitial nephritis or ATN	Pre-glomerular vasculitis, HUS	Interstitial nephritis, acute tubular necrosis, pyelonephritis	Uric acid-tumour lysis syndrome Ca oxalate- B6 deficiency, hyperoxaluria

Ongoing investigations

U&E, creatinine and bicarbonate (frequency determined by clinical picture)

Ca, PO₄, Mg, albumin, ALP, FBC, Urinalysis (daily), Urine U&E daily (unless on diuretics)

Supportive Management

Initial Fluid management

Hydration status	clinical features	initial management *
Dehydrated	tachycardia, cool peripheries, prolonged CRT, dry mucous membranes, sunken eyes, U _{Na} <10 (<20 in neonates)	fluid resuscitation 10-20 ml/kg normal saline over 1 hour then re-assess
Euvolaemic		fluid challenge 10-20 ml/kg normal saline over 1 hour, consider furosemide up to 5 mg/kg if no urine response
Overloaded	tachycardia, gallop rhythm, elevated JVP, oedema, hypertension	furosemide 4 mg/kg if fluid overload is severe; dialysis if no response to furosemide

* Further boluses of crystalloid or colloid and/or frusemide as indicated by clinical state of hydration and urine output. If nephrotic, consider albumin

Monitoring - daily or twice daily weights, accurate input-output recording, at least hourly BP initially, at least 4 hourly monitoring of toe-core temperature gradient, 6 hourly BMs

On going fluid management –

Aim is to maintain isovolaemia erring on the side of minimal fluid overload. As a rule of thumb fluid therapy should equal insensible fluid losses (400 ml/m²/d) plus output (urine, vomiting, drain losses, diarrhoea etc).

Insensible fluid losses:

Normal (ml/Kg)	Increase	Decrease
1-10 Kg 25 10-20 Kg 12.5 >20 Kg 5	Abnormal fluid losses, 10-25% if sweating By 12% for (temp) every 1°C >37.5 25-50% if hypermetabolic/hyperventilating 25% for radiant heater/phototherapy	Oedema or antidiuretic state, Humidified atmosphere, Ventilated, By 40% if paralysed

- Give 100 % urine output if euvolaemic but restrict to 50 - 75 % urine output if overloaded
- This can be modified to fluid restriction if patient is on dialysis or urine output established
- In polyuric recovery phase, replace urine output and insensible losses for 24 h, then set fluid target if renal function continuing to improve
- maintain normoglycaemia, especially in infants

Hyperkalaemia

- $K > 6.5$ mmol/l is an indication for treatment until dialysis or urine output has been established
- Monitor for signs of toxicity on ECG
- Manage as per **Hyperkalaemia guidelines**

Hyponatraemia

- Mild hyponatraemia often dilutional secondary to fluid overload
- If $Na > 120$ mmol/l, will usually correct with fluid restriction, RRT and fluid replaced as normal saline
- If $Na < 120$ mmol/l, risk of seizures, should raise Na to around 125 mmol/l with hypertonic saline (3 %) according to formula: (***Details see hyponatraemia guidelines***)
$$Na \text{ dose (mmol)} = (125 - \text{measured } P_{Na} \times \text{weight} \times 0.6) \text{ and given over 2 h}$$

Give 50% of above and then reassess before administering more
- Severe hyponatraemia with oliguria is an indication for dialysis

Hypernatraemia

- Less common than hyponatraemia, may be caused by sodium retention or water depletion so careful assessment of fluid status is mandatory.
- consider frusemide 4 mg/kg iv or replacing insensible losses as hypotonic fluid
- Severe hypernatraemia with oliguria is an indication for dialysis

Hyperphosphataemia

- Start treatment if plasma $PO_4 > 1.7$ mmol/l (> 2.0 in a neonate)
- Prevent tissue catabolism by providing adequate calories
- Treatment includes dietary phosphorus restriction and PO_4 binders which are given with meals.
- Calcium carbonates starting dose:
 - Up to 2 years: 250 mg qds
 - 2 - 5 years: 420 mg tds
 - 5 - 10 years: 840 mg tds
 - over 10s 1250 mg tds

Hypocalcaemia

- A combination of hyperkalaemia and hypocalcaemia can lead to cardiac arrest, therefore cardiac monitoring should be commenced for severe hypocalcaemia
- Measure ionised Ca and PTH before commencing treatment
- Can be treated with oral calcium salts and calcium carbonate (hypocalcaemia is related to hyperphosphataemia and requires aggressive treatment of the latter in the first instance)
- Need to consider possibility of acute on chronic which needs commencement of 1α -calcitriol
- if severe (< 1.9 mmol/l) or if bicarbonate therapy is required, treat with iv 10 % calcium gluconate 0.5 ml/kg over 30 min (***refer to hypocalcaemia guidelines***)

Acidosis

- May be severe if depression of consciousness as respiratory system unable to compensate
- Correct with sodium bicarbonate if acidosis severe or child has hyperkalaemia
- Calculate iv dose as: $\text{mmol } NaHCO_3 = (18 - \text{measured } HCO_3) \times 0.5 \times \text{weight}$
- Give half over 1 hour, then reassess before giving remainder
- Oral dose is 1-2 mmol/kg/d for infants and 70 mmol/m²/d for older children, in 2-4 divided doses.
- Check ionised calcium before treatment and correct severe hypocalcaemia first (iCa is affected by pH and raising pH will worsen hypocalcaemia, potentially leading to cardiac arrhythmias).

Hypertension

- May be related to fluid overload or alteration in vascular tone
- First treatment of fluid overload is diuretics, and failure to respond is an indication for dialysis
- Medical treatment (i.e. oral or intravenous therapy) depends on clinical condition or severity
- Pulmonary oedema with oliguria is an absolute indication for ventilation and dialysis.
- If dialysis is adequate, nifedipine alone is often sufficient. Best to use modified/slow release preparations to avoid sudden drop in case of acute on chronic hypertension. Starting dose is 250 µg/kg tds. Maximal daily dose is 4 mg/kg/d.
- See ***hypertension guidelines*** for more detailed advice on hypertension
- Choice of agent depends on degree of HT, presence of CNS symptoms & cause of ARF

Nutrition

- ARF is a hypercatabolic state and malnutrition can develop rapidly
- Dietetic review for all children with ARF, to prescribe low K, low PO₄ diet
- Aim for at least maintenance calorie intake
- Protein intake can be judged on plasma urea. When the urea is > 40, the diet should be protein-free; around 30, it should contain 0.5 g/kg/d and if < 20, the full recommended nutrient intake for protein can be given.
- Start nutritional feeds orally or via NG tube early to minimise catabolism and uraemia
- If enteral feeding not possible, TPN should be considered early

Drug therapy

- For the purposes of correcting drug doses according to GFR, estimate GFR as <20 in pre-recovery ART
- Change of GFR is important and doses may need to be revised regularly
- Many drugs require decreased doses or prolonged dosage interval in renal failure - consult formulary for advice before prescribing
- Best to avoid known nephrotoxic drugs in ARF where alternative available

Dialysis

Indications

1. Hyperkalaemia > 6.5 mmol/l
2. Severe fluid overload with pulmonary oedema which is resistant to diuretics
3. Uraemia > 40 mmol/l (> 30 mmol/l in a neonate) and rising
4. Multi-system failure
5. Anticipation of prolonged oliguria e.g. HUS
6. Uncontrolled acidosis
7. Severe hypo/hypernatraemia with oliguria

Choice of dialysis

- options are peritoneal dialysis, haemodialysis or haemofiltration
- Haemodialysis is the preferred option if plasma exchange is thought to be a possibility since vascular access is obtained
- Gold standard choice is haemodialysis (better solute clearance). Temporary vascular access lasts only for 2 weeks so if dialysis is likely to continue for longer, peritoneal dialysis may be better (e.g. HUS)
- In HUS, PD may also be better for removal of plasminogen-activator inhibitor type I (4)
- Tenckhoff catheters are associated with fewer problems of leakage and poor drainage than acute PD catheters (5)

Haemodialysis

- Put patient on emergency list
- Inform transplant surgeon (for those >16 years old) or paediatric surgeon (those <16 years) on-call to request placement of catheter. Provide catheter of correct size (available in store room in Heulwen ward/ HD room in CKC). Place a nasogastric tube in theatre.
- Chase pre-operative blood results (FBC and U&E, bone profile, coagulation, Gp& save)
- Inform nurse on-call for haemodialysis

Peritoneal dialysis

- Put patient on emergency list
- Inform transplant surgeon (for those >16 years old) or paediatric surgeon (those <16 years) on-call to request placement of catheter. Provide catheter of correct size (available in store room in Heulwen ward/ HD room in CKC). Place a nasogastric tube in theatre.
- Chase pre-operative blood results (FBC and U&E, bone profile, coagulation, Gp& save)
- HUS patients may require platelet transfusion peri-operatively
- Commence PD on return from theatre
- Bicarbonate dialysis is occasionally required in cases of abnormal liver function or lactic acidosis.

References

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Algorithm for the diagnosis and treatment of acute renal failure

