Acute Kidney Injury (AKI)

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Purpose
To inform staff about the risk factors for, and recognition, investigation and management of Acute Kidney Injury (AKI) in children.

Intended Audience
For use by all staff within the Trust who care for children.
Acute Kidney Injury

1. Background

Acute kidney injury (AKI), previously known as acute renal failure encompasses a wide spectrum of injury to the kidneys, not just kidney failure. It is a sudden, potentially reversible inability of the kidney to maintain normal body chemistry and fluid balance.

The incidence of AKI in children is unknown. It is likely that early AKI goes unrecognised currently and a number of AKI cases in at risk groups/high risk clinical scenarios are preventable. For this reason it is important to have a high index of suspicion when reviewing a child who is in this situation. Early steps to address physiological disturbances and to reduce exposure to drugs that may adversely affect renal function may well prevent progress to more severe renal dysfunction and so reduce morbidity and mortality.

AKI can occur without symptoms and is detected through measurement of serum creatinine and/or a decrease in urine output. In many milder cases, management by a general paediatrician is appropriate, but where there are specific risk factors or more severe or rapidly deteriorating cases, the advice of a paediatrician with experience in treating renal failure should be sought early.

2. How to recognise AKI and interpret AKI warning scores

A creatinine above the acceptable range may indicate chronic kidney disease (CKD) or AKI. The hallmark of AKI is a recent increase in creatinine greater than 1.5 x previous "reference" result or a value greater then 1.5 x upper limit of the reference interval for age (ULRI). It is usually associated with a fall in urine output (< 0.5mls/kg/hr for 8 hours).

i. Interpretation of AKI warning scores

Hospitals are required to issue electronic AKI warning scores based on the measurements of serum creatinine and these appear on the ICE report. These alerts need to be coupled together with an appropriate clinical management plan.

AKI Stage 1 – creatinine > 1.5 - 2x reference creatinine or ULRI
AKI Stage 2 – creatinine 2 - 3x reference creatinine or ULRI
AKI Stage 3 – creatinine > 3x reference creatinine or ULRI

The causes of AKI may be pre-renal, intra-renal (intrinsic renal disease) or post renal (obstruction). It is important to recognise all stages of AKI and take appropriate action to manage and investigate the cause of AKI. AKI 1 may respond to simple measures eg appropriate fluid management but may herald a more significant situation or disease and progress to AKI 2 or 3.

3. At Risk Patients

Certain children are at greater risk of developing AKI either because of pre-existing disease/risk factors or because they fall into an acute high risk clinical scenario. Children in these groups should have their serum creatinine measured as part of their clinical assessment.

i. Children at high risk of AKI include those with:

- Nephro-urological, cardiac or liver disease
- Malignancy and/or a bone marrow transplant
- Dependence on others for access to fluids
- History of taking medication that may adversely affect renal function (most commonly ACE Inhibitors eg Enalapril/Angiotensin II blockers eg Losartan, Non Steroidal Anti-
inflammatory drugs eg Ibuprofen (including topical NSAIDs), aminoglycosides, calcineurin inhibitors eg tacrolimus

ii. Clinical scenarios in which children can be at high risk of AKI include:

- History of reduced urine output
- Sepsis
- Hypoperfusion or dehydration
- History of exposure to drugs or toxins that may adversely affect renal function
- Renal disease or urinary tract obstruction
- Major surgery

4. What steps should be taken to prevent AKI?

The following steps should be undertaken to prevent AKI in high risk groups/scenarios or where there are other concerns:

Monitor, Maintain, Minimise – 3Ms

i. Monitor

Check creatinine and repeat as needed if there are any concerns. Assess and record state of hydration, fluid balance including urine output, weight, urinalysis and PEWS. Review all of this on at least a daily basis. Urgently investigate and manage any signs of sepsis.

ii. Maintain

Ensure that there is an adequate circulatory volume and perfusion pressure. Hypo-perfusion should be addressed urgently with fluid boluses and inotropic support once the child is fluid replete as needed.

iii. Minimise

Further harm should be reduced by reviewing, adjusting and monitoring medication that may affect renal function adversely eg NSAIDs, ACEI, ARB, aminoglycosides and calcineurin inhibitors. Intravenous contrast should be avoided if possible.

5. How should AKI be managed to prevent permanent damage?

After recognition, the underlying cause should be assessed and treated where possible.

AKI should be evaluated and reviewed according to the following cycle:
To establish the cause of AKI, assessment by a senior clinician should be undertaken urgently. The majority of AKI will be pre-renal due to hypovolaemia and will be corrected with adequate fluid repletion and/or steps to improve perfusion.

Further investigations allow identification of established intra-renal disease and obstruction. Early identification of these situations such as nephritis, vasculitis, HUS may help ameliorate the course of the disease and reduce progression to CKD.

i. Investigations recommended for all children with AKI:

FBC, U&Es, creatinine, bone profile, albumin
Urinalysis (dipstick), urine microscopy (and culture where indicated)
Urinary tract ultrasound scan – unless mild AKI and responding promptly and fully to management – within 24 hours

ii. Further management

a. AKI 1

Undertake further investigations as clinically relevant – C3, C4, ASOT, antiDNAse B titres, immunoglobulins, autoimmune profile, ANCA, anti-GBM antibodies, CK, LDH, blood film.

Consider discussion with paediatrician with special interest in nephrology (SPIN) or tertiary nephrologist. This should occur if known CKD, renal transplant, features of intrinsic renal disease eg nephritis, HUS or evidence of multisystem disease.

b. AKI 2 & 3

Investigations as for AKI 1.
Discuss with SPIN or tertiary nephrologist.

c. Indications for immediate paediatric nephrology referral in any stage of AKI

Potassium > 6.5 mmol/l in non-haemolysed sample
Oligoanuria and plasma Na < 125 mmol/l
Pulmonary oedema or hypertension unresponsive to diuretics
Plasma urea > 40 mmol/l unresponsive to fluid challenge where this is appropriate management
AND discuss with PICU if the child is not already known to them.

6. Medicines optimisation in children with AKI

When AKI is identified a thorough review of medication is required:

- To eliminate as potential cause/risk/contributory factor for AKI
- To avoid inappropriate combinations of medications in the context of AKI
- To reduce adverse events
- To ensure that doses are appropriate for the patient’s level of renal function
- To ensure all medicines prescribed are clinically appropriate

Summary of drugs to be stopped, avoided, reduced or monitored in AKI:

Refer to BNFc for more information. Consider ALL medications including any “usual” long term medications.
Check medication history thoroughly including “Over the Counter” preparations and alternative therapies.


7. When to re-start Angiotensin Converting Enzyme Inhibitors (ACEI), Angiotensin II Receptor Blockers (ARB), diuretics, anti-hypertensives and non steroidal anti-inflammatory drugs (NSAIDs) after an episode of AKI
The original indication for the use of any suspended drugs should be reviewed and any new contra-indications (eg hyperkalaemia, identification of renal artery stenosis) identified.

For individuals previously stabilised on drugs for heart failure, these drugs should be re-started as soon as clinically reasonable and re-titrated to get the best control of fluid balance and blood pressure unless there is a specific contra-indication. It is advised to discuss with both cardiology and nephrology specialists.

Individuals previously stabilised on ACEI or ARB for albuminuria secondary to chronic kidney disease or diabetes mellitus should be re-started on these drugs with resolution of AKI and any associated hypovolaemia, hypotension or sepsis unless there is a new contra-indication to their use (eg hyperkalemia). If concerns discuss the timing with paediatric nephrology specialist.

Renal function and electrolytes should be re-checked within a week of re-starting treatment.

When individuals previously stabilised on anti-hypertensives have had medication discontinued or reduced, this should continue to be titrated according to clinical situation and blood pressure control.

Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided until AKI has resolved completely. They should be avoided in the future if it is felt that the NSAID was responsible for AKI or if there is continuing renal disease. Otherwise, they may be considered for occasional use with caution and with close monitoring if continued use is required. If any doubt consult with nephrology specialist.

Prompt follow up including review of any of the above drugs following discharge is required. Any changes to drug treatment, plans for treatment and arrangements for follow up and repeat blood tests where required should be made clear on the discharge summary.

8. Follow up after AKI

It is important to demonstrate complete resolution of AKI. That is renal function, blood pressure and urinalysis all return to normal.

All patients who required haemofiltration or haemodialysis or who have persisting proteinuria, reduced renal function or other concerns regarding their renal status 3 months post AKI should be followed up by either someone with a renal interest or a tertiary nephrologist.

Assessment of renal function is undertaken by calculation of estimated GFR (eGFR) from serum creatinine.

- In those **over 2 years** of age, the equation that is used currently to estimate the kidney function is:

  \[
  \text{height (in cm)} \times 40/\text{serum creatinine} = \text{GFR in mls/minute/1.73m}^2.
  \]

  Normal should be taken as > 90 mls/minute/1.73m².

  However, if the result is reduced or around this value then seek nephrology advice as eGFR may be inaccurate and overestimate renal function.

  In due course eGFRs will be reported on ICE.

- For those **< 2 years old**, seek nephrology advice if you are uncertain as to if renal function has returned to normal or not.
- Using a formula calculation is **not reliable** for children **under 2 years of age**, for whom a normal upper limit of serum creatinine of 35 umol/l can be used.

Staging of AKI can be as follows:
Stage 1 – creatinine over 50 umol/l
Stage 2 – creatinine over 75 umol/l
Stage 3 – creatinine over 100 umol/l or need for renal replacement therapy

- In the neonatal period, GFR matures rapidly. Creatinine in the first days of life will reflect that of the mother, but should fall quickly to below 35 umol/l. A stable or rising creatinine in this period is abnormal.

Blood pressure should be assessed for age and height – see guidelines for blood pressure and hypertension and review value using centile charts. The Fourth Task Force on Blood Pressure in Children is available online and contains the same blood pressure centile tables.

Porteinuria can be assessed on initial dipstick analysis of urine. If the dipstick is negative for protein, this can be taken as normal. However, if there is proteinuria evident on dipstick (trace or more in this situation) measure protein:creatinine (on early morning urine if possible) and seek nephrology advice if > 20 mg/mmol.

9. References and further information


- Think Kidneys website www.thinkkidneys.nhs.uk

- Nottingham University Hospital Clinical guideline on assessment and management of Acute Kidney Injury in Children and Young People. Revised September 2017

Additional comment

This guideline is based on “Guidance for clinicians managing children at risk of, or with, acute kidney injury” published May 2016 (https://www.thinkkidneys.nhs.uk/aki/guidance-clinicians-managing-children-risk-acute-kidney-injury/). This document was created by The AKI Working Group of The British Association for Paediatric Nephrology. It is in line with the NICE Guideline relating to the management and treatment of acute kidney “Acute Kidney Injury” issued December 2014.