

Acute Kidney Injury AKI UHL Childrens Hospital Guideline

Introduction and Who Guideline applies to:

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. This guideline is based on the NICE AKI guideline (August 2013)¹ which emphasises early intervention and stresses the importance of risk assessment and prevention, early recognition and treatment. In many milder cases, management by a general paediatrician is appropriate, but for more severe or rapidly deteriorating cases, the advice of a paediatrician with experience in treating renal failure should be sought early.

There are specific guidelines for the following common causes/types of acute kidney injury which should be referred to if relevant:

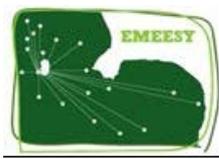
- Gastroenteritis - [Gastroenteritis UHL Childrens Guideline](#)
- Acute glomerulonephritis - [Glomerulonephritis UHL Childrens Hospital Guideline](#)
- Sepsis - [Sepsis UHL Childrens Hospital Guideline](#)

This guideline applies to Children and young people under 18 years of age with Acute Kidney Injury within the EMEESY Children's Kidney Network (East Midlands, East of England and South Yorkshire) being managed by the Leicester Children's Hospital and the Paediatric Emergency Department.

This EMEESY network guideline has been developed by clinicians from Nottingham Children's Renal Unit with consultation across the network including from the Leicester Royal Infirmary and has been ratified by the Leicester Children's Hospital guideline process.

Abbreviations:

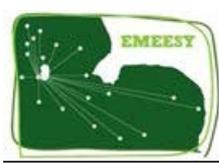
ACE – Angiotensin-Converting Enzyme
AKI – Acute Kidney Injury
AKIN – Acute Kidney Injury Network
ANA – Antinuclear Antibody
ANCA – Anti-Neutrophilic Cytoplasmic Autoantibody
ARBS - Angiotensin 2 Receptor Blockers
CKD – Chronic Kidney Disease
CXR – Chest X-ray
eGFR – estimated Glomerular Filtration Rate
EMEESY - Children's Kidney Network (East Midlands, East of England and South Yorkshire)
FE_{Na} - Fractional Excretion of Sodium
HSP - Henoch Schonlein Purpura
HUS – Haemolytic Uremic Syndrome
JVP – Jugular Venous Pressure
KDIGO – Kidney Disease: Improving Global Outcomes
LDH – Lactate Dehydrogenase



PEWS – Paediatric Early Warning System
pRIFLE – Risk Injury Failure Loss End-stage
SLE - Systemic Lupus Erythematosus

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1. Flow Chart for Management of Patients at Risk of Acute Kidney Injury

High index of suspicion for at risk patients e.g. (see section 2)

- Sepsis, hypovolaemia
- Underlying renal, cardiac or liver disease
- Bloody diarrhoea, purpuric rash, history of nephrotoxic drugs
- Young age, neurological or cognitive impairment or disability, which may mean limited access to fluids because of reliance on a parent or carer

- Monitor urine output and daily weight – optimise fluid balance (section 2)
- Measure electrolytes, urea, creatinine (calculate eGFR)
- Seek pharmacy advice to optimise drug dosing, and consider stopping or avoiding nephrotoxic agents (e.g. ACE inhibitors, NSAIDs, aminoglycosides, contrast agents)

If eGFR \geq 90 ml/min/1.73m²

- Continue regular monitoring

If reduced eGFR ($<$ 90 ml/min/1.73m²)

- Assess and manage fluid status (section 4)
- Manage any electrolyte disturbance (section 4)
- Manage hypertension (section 4)
- Attempt to identify cause of AKI (section 2)
- Watch for polyuria in recovery phase

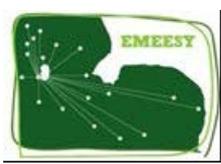
Criteria for discussion with Paediatric Nephrologist¹

- Hyperkalaemia or metabolic acidosis or fluid overload *failing to respond to medical management**
- Pulmonary oedema*
- AKI stage 1 with no clear underlying cause
- AKI stage 2 or 3
- Haemolytic Uraemic Syndrome
- Glomerulonephritis (including HSP) with \geq 1+ proteinuria
- Suspected vasculitis with renal involvement (e.g. SLE, Wegeners)
- Tubulointerstitial nephritis
- Some children with AKI will need urgent urology input. These will need to be discussed with a paediatric nephrologist first who will then liaise with a urologist to decide a joint management plan.
- Renal transplant
- CKD stage 3, 4 or 5
- Patient under care of Paediatric Nephrologist

***These children may require dialysis and should be discussed urgently with the paediatric nephrologist on call to ensure that therapy is started as soon as needed.**

Follow up (section 8)

- Any child who has had stage 3 AKI (whether or not dialysis was required)
- Persistent (i.e. more than 3 months post AKI)
 - Hypertension
 - Proteinuria (early morning urine albumin: creatinine ratio $>$ 30 mg/mmol)
 - Reduced renal function i.e. estimated GFR $<$ 90 ml/min/1.73m²



2. At Risk Patients¹

Certain children and young people are particularly at risk of developing AKI. Measurement of serum creatinine and comparison with baseline should be undertaken in children and young people with acute illness if any of the following are likely or present:

- chronic kidney disease / transplant
- heart failure
- liver disease
- past history of acute kidney injury
- oliguria (urine output less than 0.5 ml/kg/hour)
- young age, neurological or cognitive impairment or disability, which may mean limited access to fluids because of reliance on a parent or carer with history or examination suggestive of dehydration
- hypovolaemia
- use of drugs with nephrotoxic potential (such as Nonsteroidal Anti-inflammatory Drugs, aminoglycosides, Angiotensin Converting Enzyme Inhibitors, Angiotensin 2 Receptor Blockers and diuretics) within the past week, especially if hypovolaemic
- symptoms or history of urological obstruction, or conditions that may lead to obstruction
- sepsis
- a deteriorating paediatric early warning score
- severe diarrhoea (children and young people with bloody diarrhoea are at particular risk)
- symptoms or signs of nephritis (such as oedema or haematuria)
- haematological malignancy
- hypotension.

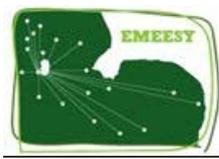
Be aware that in children and young people with chronic kidney disease and no obvious acute illness, a rise in serum creatinine may indicate acute kidney injury rather than a worsening of their chronic disease – what used to be called “acute on chronic renal failure”

Ensure that acute kidney injury is considered when child or young person presents with an illness with no clear acute component and has any of the following:

- chronic kidney disease, stage 2 or above
- new onset or significant worsening of urological symptoms
- symptoms or signs of a multi-system disease affecting the kidneys and other organ systems (for example rash, joint pains).

When children and young people are at risk of acute kidney injury because of risk factors above, the following measures should be undertaken:

- measure **urine output**
- record **weight daily** to determine fluid balance
- measure **urea, creatinine and electrolytes**
- consider measuring lactate, blood glucose and blood gases (e.g. PEWS 6 or greater)
- seek pharmacy advice (this including out of hours the BNFC may not be the most appropriate source of advice about renal dosing) about **optimising medicines and drug dosing**
 - consider temporarily stopping **ACE inhibitors and ARBs** in children and young people with diarrhoea, vomiting or sepsis until their clinical condition has improved and stabilised
 - avoid use of **nephrotoxic agents** e.g. NSAIDS, aminoglycosides, iodinated contrast agents



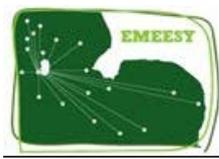
3. Detecting Acute Kidney Injury^{2,3,4,5}

AKI can be staged using a modification of KDIGO, AKIN and pRIFLE definitions.

	Serum Creatinine	Urine Output
Stage 1	eGFR < 75 ml/min/1.73m ² Or decrease in eGFR by 25% Or increase creatinine 1.5 – 2 x baseline	<0.5 ml/kg/hr for 6 hours
Stage 2	eGFR < 50 ml/min/1.73m ² Or decrease in eGFR by 50% Or increase creatinine 2 – 3 x baseline	<0.5 ml/kg/hr for 12 hours
Stage 3	eGFR <35 ml/min/1.73m ² Or decrease in eGFR by 75% Or increase in creatinine > 3 x baseline Or requirement for renal replacement therapy	<0.3 ml/kg/hr for 24 hours Or anuria for 12 hours

4. Identifying the Cause(s) of Acute Kidney Injury^{1,5,6}

- **detailed history and examination** (see appendix 2)
- **urine dipstick testing** and further testing as below;
 - Nitrites or leucocytes → microscopy, culture and sensitivities
 - Blood ≥+ consider nephritis screen
 - Protein ≥+ → early morning urine albumin: creatinine ratio
- **urea and electrolytes**
- **creatinine** - calculate eGFR if child over 2, consider abnormal if child under 2 years of age has creatinine >35 umol/l (see appendix 1)
- **calcium, phosphate, bicarbonate, chloride, liver function tests (inc. albumin)**
- **glucose**
- **full blood count**
- consider, if clinically appropriate (see appendix 2 for possible underlying causes of AKI and discuss with paediatric nephrologist if unsure):
 - blood film and LDH (especially if bloody diarrhoea, low platelets ?HUS)
 - coagulation screen
 - blood culture, CRP, lactate
 - complement levels, ANCA, ANA
 - anti-GBM antibody titres
 - creatine kinase



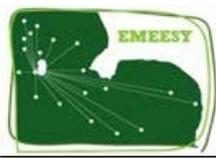
- calculation of fractional excretion of sodium to differentiate between pre-renal and established renal failure (see appendix 3)
- CXR if respiratory or cardiac signs
- renal biopsy
- ultrasound
 - if **pyonephrosis** or **renal vein thrombosis** suspected - ideally within 6 hours of assessment
 - if no identified cause of acute kidney injury and **at risk of urinary tract obstruction** e.g. stones - ideally within 24 hours of assessment
 - if **undiagnosed chronic kidney failure suspected** e.g. Potential renal dysplasia - within 2 – 3 days of assessment
 - do not routinely offer ultrasound when the cause of the acute kidney injury has been identified e.g. HUS, drug / toxin injury

5. Criteria for Discussion with Paediatric Nephrologist¹

- Hyperkalaemia or metabolic acidosis or fluid overload **failing to respond to medical management***
- Pulmonary oedema*
***These children may require dialysis and should be discussed urgently with the paediatric nephrologist on call to ensure that the therapy can be started promptly. (0115 924 9924 - ask switch to page on call Paediatric Nephrologist)**

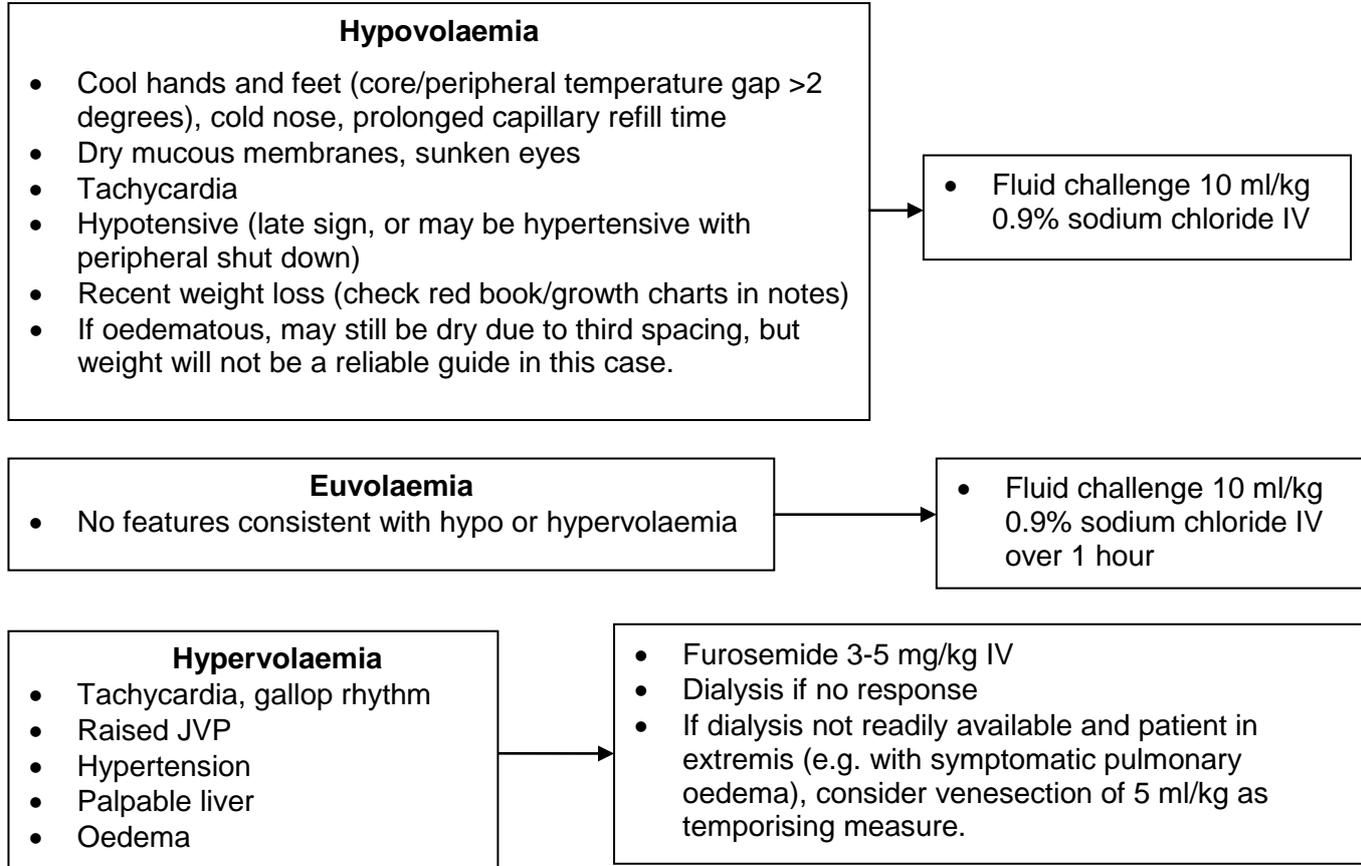
Otherwise contact the person on service for Paediatric Nephrology for children as below

- AKI stage 1 with no clear underlying cause
- AKI stage 2 or 3
- Haemolytic Uraemic Syndrome
- Glomerulonephritis (including HSP) with $\geq 1+$ proteinuria
- Suspected vasculitis with renal involvement (e.g. SLE)
- Tubulointerstitial nephritis
- Some children with AKI will need urgent urology input. These will need to be discussed with a paediatric nephrologist first who will then liaise with a urologist to decide a joint management plan.
- Renal transplant
- Chronic Kidney Disease (CKD) stage 3, 4 or 5
- Patient under care of Paediatric Nephrologist



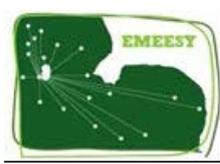
6. Management

6.1 Initial Fluid Management



6.2 Ongoing Fluid Management (see [Fluid Electrolyte Management UHL Childrens Hospital Guideline](#))

- If tolerated give oral fluids / feeds
- Children with AKI stage 1 responding well to initial management can be managed as per the gastroenteritis guideline (maintenance + deficit over 12-24 hr)
- Children with AKI stage 2-3 require a more complex regimen. Start with:
 - insensible losses (400 ml/m²/day) plus:
 - ml for ml replacement of urine output plus:
 - replacement of any significant ongoing losses plus:
 - correction for hydration status i.e.
 - replacement of deficit if dehydrated
 - reduction in fluid intake if fluid overloaded
- Include all fluid i.e. oral, IV, medication
- Review fluid balance initially after 4-6 hours and continue to reassess regularly
- If at review it is clear that AKI and fluid & electrolyte status is improving, consider step down to a simpler fluid regimen



- Generally 0.9% saline with 5% dextrose is used, but this will depend on the diagnosis and recent blood results. Discuss with paediatric nephrologist and review regularly with blood results

6.3 Management of Complications

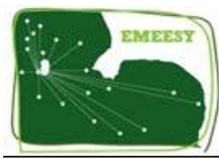
- Guidelines are available for:
 - Hypertension ([Hypertension UHL Childrens Medical Guideline](#))
 - Hypo or Hypernatremia ([Fluid Electrolyte Management UHL Childrens Hospital Guideline](#))
 - Hypokalaemia ([Fluid Electrolyte Management UHL Childrens Hospital Guideline](#))
 - Hyperkalaemia ([Fluid Electrolyte Management UHL Childrens Hospital Guideline](#))
 - Hypocalcaemia ([Calcium Disorders UHL Childrens Hospital Guideline](#))
- Metabolic acidosis (may be severe, especially in critically ill children with shock, sepsis or impaired respiratory compensation)
 - May resolve on treating underlying cause
 - Can consider bicarbonate *once calcium corrected* (see hypocalcaemia guideline)
 - See high risk drug monograph for further information on administration of bicarbonate.

7. Ongoing Monitoring

- Continue daily weights whilst an in-patient
- Once stable, re-assessment of fluid balance and bloods can be decreased in frequency.
- Polyuria may develop – consider returning to hourly input/output measurements, BP and pulse to avoid development of hypovolaemia
- Patients can be discharged once renal function is improving, providing fluid and electrolyte balance and any hypertension can be managed as a frequent outpatient or regular ward attender. Monitor creatinine and consider referral to a paediatric nephrologist for long term follow up.

8. Long Term Follow Up

- Many children recover normal renal function. Paediatric nephrology follow up, with a plan to measure **formal GFR at 1 and 5 years** following AKI and **post puberty** is recommended for⁷:
 - Any child who has required dialysis
 - Persistent (i.e. more than 3 months post AKI)
 - Hypertension



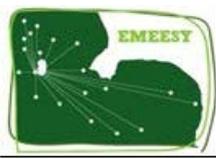
- Proteinuria (albumin: creatinine ratio > 30 mg/mmol on early morning specimen of urine)
 - Reduced renal function i.e. estimated GFR < 90 ml/min/1.73m²
- Children who have sustained a significant kidney injury (**AKI Stage 3**) but did not require dialysis should have **lifelong annual review** looking for signs predisposing to late onset renal dysfunction:
 - Blood pressure (refer if hypertensive)
 - Urinalysis for proteinuria (refer if albumin: creatinine ratio > 30 mg/mmol on early morning specimen of urine)
 - Creatinine at 1 and 5 years post AKI (refer if estimated GFR < 90 ml/min/1.73m²)
 - If eGFR at 5 years is >90 ml/min/1.73m² and there is no evidence of hypertension or proteinuria, further follow up (annual review as above) can be undertaken by the GP.

9. Audit Points

1. Are investigations undertaken as per guidelines?
2. Are referrals made to paediatric nephrology appropriate and at the correct time?
3. Is appropriate long term follow up organised?

10. References

1. Acute kidney injury: Prevention, detection and management of acute kidney injury up to the point of renal replacement therapy – NICE guideline CG169 August 2013
2. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, Levin A. Acute Kidney Injury Network: Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Crit Care*. 2007;11:R31
3. Akcan-Arikan A, Zappitelli M, Loftis LL, Washburn KK, Jefferson LS, Goldstein SL (2007) Modified RIFLE criteria in critically ill children with acute kidney injury. *Kidney Int* 71:1028-1035
4. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int Suppl*. 2012;2:1-138
5. NUH Acute Kidney Injury (AKI) Guideline – Early Identification and Management (Jan 2011) NUH Intranet
6. Rees L, Brogan PA, Bockenhauer D, Webb N Pediatric Nephrology 2nd ed Oxford Specialist Handbooks in Paediatrics (Oxford University Press) 2012
7. Goldstein SL, Devarajan P Acute kidney injury in childhood: should we be worried about progression to CKD? *Pediatr Nephrol* 2011:26-509



11. Training & Education:

None

12. Key words:

Acute kidney injury, nephritic syndrome, glomerulonephritis, haematuria, Henoch Schoenlein Purpura, HSP, Haemolytic Uraemic Syndrome, HUS, Renal

The Trust recognises the diversity of the local community it serves. Our aim therefore is to provide a safe environment free from discrimination and treat all individuals fairly with dignity and appropriately according to their needs. As part of its development, this policy and its impact on equality have been reviewed and no detriment was identified.

Document Control

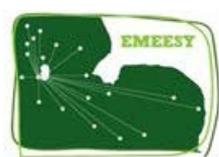
Document Amendment Record

Version	Issue Date	Author
V1	July 2014	Dr Corinne Langstaff
V2	Sept 2017	Dr Andrew Lunn

Summary of changes for new version:

Removal of reference to Haemolytic uraemic syndrome guideline

CONTACT AND REVIEW DETAILS	
Guideline Lead (Name and Title) Angela Hall – Associate Specialist	Executive Lead Chief Medical Officer
Details of Changes made during review: 24/07/2020 Added examples of nephrotoxic drugs - use of drugs with nephrotoxic potential (such as Nonsteroidal Anti-inflammatory Drugs , aminoglycosides , Angiotensin Converting Enzyme Inhibitors , Angiotensin 2 Receptor Blockers and diuretics Added caution - seek pharmacy advice (this including out of hours the BNFc may not be the most appropriate source of advice about renal dosing) Added local information - Appendix 4: UHL Clinical review	

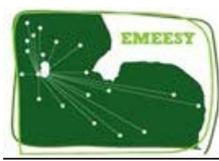

UHL local renal specialists are:

Dr Angela Hall Associate Specialist

Dr Sudarsana De Consultant Paed Nephrologist (shared post with Nottingham)

They can be contacted via UHL switchboard.

	Title of Guideline (must include the word "Guideline" (not protocol, policy, procedure etc)	Guideline for the assessment and management of acute kidney injury in children and young people
	Contact Name and Job Title (author)	Dr A Lunn Consultant Paediatric Nephrologist Andrew.lunn@nuh.nhs.uk
	Directorate & Speciality	Directorate: Family Health – Children Speciality: Renal
	Date of submission	Sept 2017
	Date when guideline reviewed	Sept 2022
	Guideline Number	2245
	Explicit definition of patient group to which it applies (e.g. inclusion and exclusion criteria, diagnosis)	Children and young people under 18 years of age with or at risk of acute renal failure
	Abstract	This guideline describes the assessment and initial management of children and young people presenting with or at risk of acute kidney injury including criteria for referral to specialist services.
	Key Words	Paediatrics. Children. Renal failure, acute kidney injury, nephritic syndrome, glomerulonephritis, haematuria, Henoch Schoenlein Purpura, HSP, Haemolytic Uraemic Syndrome, HUS, renal
	Statement of the evidence base of the guideline – has the guideline been peer reviewed by colleagues?	
1a	meta analysis of randomised controlled trials	
2a	at least one well-designed controlled study without randomisation	
2b	at least one other type of well-designed quasi-experimental study	
3	well –designed non-experimental descriptive studies (ie comparative / correlation and case studies)	
4	expert committee reports or opinions and / or clinical experiences of respected authorities	X British Association for Paediatric Nephrology AKI working group guideline available on thinkkidneys.nhs.uk
5	recommended best practise based on the clinical experience of the guideline developer	
	Consultation Process	Paediatric Nephrologists, Child Health Guidelines SOP
	Target audience	Clinicians and healthcare professionals within Nottingham Children's Hospital and throughout the East Midlands, East of England and South Yorkshire caring for children and young people with or at risk of acute kidney failure.
This guideline has been registered with the trust. However, clinical guidelines are guidelines only. The interpretation and application of clinical guidelines will remain the responsibility of the individual clinician. If in doubt contact a senior colleague or expert. Caution is advised when using guidelines after the review date.		

**Appendix 1: Calculation of estimated GFR (eGFR)**

A height based eGFR can be calculated in children over the age of 2 years using the formula:

$$\frac{K \times ht \text{ (cm)}}{\text{Creatinine (umol/l)}} = \text{eGFR (ml/min/1.73m}^2\text{)}$$

K will vary depending on the method used to measure creatinine. This should be established in consultation with the local laboratory.

In Nottingham, K = 30 except for males 13 or over when K = 36
Outside Nottingham K=40.

e.g.

- 5 year old girl:
height 110cm
creatinine 70 umol/l from UHL
K= 40

$$\frac{40 \times 110}{70} = \text{eGFR } 62 \text{ ml/min/1.73m}^2 - \text{REFER as under } 90$$

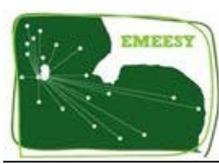
- 15 year old boy:
height 175cm
creatinine 70umol/l from UHL
K=40

$$\frac{40 \times 175}{70} = \text{eGFR } 100 \text{ ml/min/1.73m}^2 - \text{NORMAL (over } 90\text{)}$$

This calculation is **not reliable** for children **under 2 years of age**, for whom a normal **upper limit 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.



Appendix 2: Causes of Acute Kidney Injury

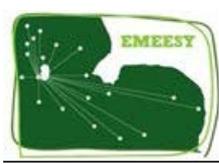
Note that these groups are not mutually exclusive and the same insult may cause pre and intrinsic renal failure (e.g. drugs, shock) or post and intrinsic renal failure (e.g. untreated vesico-ureteric junction obstruction)

Pre renal injury (reduced blood flow to the kidney)

- Hypovolaemia
 - Haemorrhage
 - Severe dehydration
 - gastrointestinal losses
 - salt wasting
 - diabetes (insipidus and ketoacidosis)
 - third space losses
 - burns
 - sepsis
 - nephrotic syndrome
 - other factors which impair renal perfusion
 - reduced cardiac output
 - vasodilatation (warm shock)
- Bilateral renal arterial or venous thrombosis
- Drugs
 - Prostaglandin synthetase inhibitors (ibuprofen)
 - Angiotensin converting enzyme inhibitors (e.g. Lisinopril, enalapril) and angiotensin receptor blockers (e.g. Losartan)
 - Diuretics
 - Calcineurin inhibitors (cyclosporine A, tacrolimus)
- Hepato-renal syndrome

Intrinsic renal injury (cause within the renal parenchyma)

- Disease of the kidney parenchyma
 - Acute glomerulonephritis
 - Bilateral acute pyelonephritis
 - Acute on chronic (decompensation of CKD due to intercurrent illness)
- Hypoxic/ischaemic injury
 - Prolonged pre-renal injury of any cause
 - Severe hypoxic insult (e.g. HIE in neonates)
- Toxins
 - Medication (e.g. Aminoglycosides, NSAIDs, aciclovir, tacrolimus, ciclosporin A, amphotericin)
 - Exogenous toxins (e.g. Ethylene glycol, methanol, insecticides, contrast media, heavy metals)
 - Endogenous toxins (e.g. Myoglobinuria from severe trauma, prolonged convulsions, viral myositis)
 - Tumour lysis syndrome (precipitation of uric acid crystals)
- Tumour infiltrate
- Vascular insults
 - Renal vein thrombosis
 - Cortical necrosis
 - Haemolytic uraemic syndrome
 - Vasculitis, polyarteritis

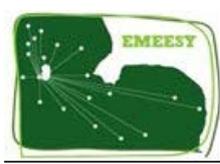
**Post renal injury (obstruction to the outflow of urine)**

- Urethral obstruction inc posterior urethral valve, ureterocele
- Obstruction in a solitary kidney
- Bilateral ureteric obstruction
- Neuropathic bladder
- Calculi
- Tumour
- Trauma
- Blocked urinary catheter
- Iatrogenic (e.g. Blocked vesico-ureteric orifices post deflux injection)

Given the multiplicity of causes of acute kidney injury, a structured approach to history and examination is important. Often, the cause is readily apparent from the presentation (e.g. acute haemorrhage, bloody diarrhoea of HUS, suspected posterior urethral valves on antenatal scan), but if not, often a careful history can indicate further appropriate investigations.

A 1.1 History (nb. Attend to life-threatening features first e.g. Volume depletion, oxygenation, extreme hypertension)

- **History of any prodromal illness:**
 - Diarrhea +/- blood (?dehydration / HUS)
 - Other events likely to result in volume depletion (see table 2.2.1)
 - History of acute pharyngitis / other infection (?acute glomerulonephritis)
 - Fever
 - Rash (legs ?HSP, face?SLE), arthropathy, weight loss (vasculitis)
- **Urinary symptoms:**
 - Polydipsia and polyuria (diabetes, renal tubular disease)
 - Macroscopic haematuria (?acute nephritis)
 - Poor urinary stream (?posterior urethral valves)
 - Dysuria, frequency, loin pain (?pyelonephritis)
- **Other systemic involvement:**
 - Diarrhoea (esp if bloody ?HUS), vomiting (dehydration or uraemia), acute weight loss
 - Recent throat or skin infection (see post infectious glomerulonephritis guidelines)
 - History of cardiac impairment
 - Prolonged convulsions (?rhabdomyolysis)
 - Birth asphyxia,
 - Small / syndromic, long history of malaise (suggests acute on chronic condition)
- **Recent fluid balance**
 - Fluid input and output (urine, gastrointestinal losses, blood loss)
 - Recent weights (see red book if possible)
- **Antenatal history including details of ultrasound scans**

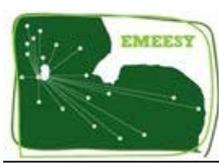


- **Neonatal history:**
 - Birth asphyxia
 - presence or history of umbilical catheters (renal vein thrombosis)
- **Past Medical History:**
 - Recurrent urinary tract infections
 - Calculi
 - Antenatally diagnosed urinary tract abnormality
 - Previous significant illness e.g. Cardiac or liver disease or previous surgery
- **Drug/toxin history (see 2.1, 2.2)**
 - Drugs causing reduced renal blood flow or renal injury
 - Non-prescribed drugs (e.g. herbal remedies, ibuprofen)
 - Toxin exposure (e.g. Insecticides, heavy metals)
- **Family history of renal disease including calculi**

A 1.2 Examination

This sometimes helps to determine the **cause** of the renal failure (rash, arthropathy, palpable bladder) but is also important in determining **fluid status** and other **complications** arising from the acute kidney injury.

- **General**
 - weight and height
 - temperature
 - rash?
 - arthropathy?
- **Cardiovascular** examination considering particularly the fluid status
 - capillary refill time
 - heart rate
 - blood pressure
 - JVP
 - Oedema
- **Respiratory** examination
 - tachypnoea due to acidosis, fluid overload or pulmonary disease
- **Abdominal** examination
 - renal mass or palpable bladder
 - loin area tenderness
 - ascites
- **Neurological** examination
 - confusion, drowsiness
 - manifestations of hypocalcaemia
 - evidence of focal neurological abnormality



Appendix 3: Calculation of Fractional Excretion of Sodium

Paired urine and blood sodium and osmolality measurements can be used to differentiate between pre-renal and established renal failure by calculating the fractional of filtered sodium that is excreted in the urine (known as the fractional excretion of sodium (FE_{Na})). This calculation directly evaluates renal sodium handling and is therefore not affected by urine volume

Pre-renal failure is suggested by

- Urine osmolality >500 mOsm/kg (>400 mOsm/kg in neonates)
- Fractional Excretion of Sodium (FE_{Na}) <1% (<2.5% in neonates)

The calculation is:

$$\frac{\text{Urine}_{\text{Na}} \times \text{Serum creatinine}}{\text{Serum}_{\text{Na}} \times \text{Urine creatinine}} \times 100 = \text{FE}_{\text{Na}}$$

Note that in:

- congestive cardiac failure
- severe liver disease
- acute glomerulonephritis
- and post administration of diuretics

a low FE_{Na} does not correlate with pre-renal disease, so **FE_{Na} should always be interpreted in line with the clinical picture.**

Appendix 4: UHL Clinical review

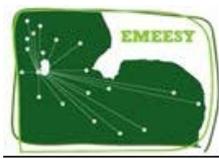
At the LRI we have a sticker to be used when children are thought to have AKI (stickers usually found with fluid balance sheets).

This is to prompt clinicians to think about possible causes and basic management.

It also acts as a reminder at discharge to include the occurrence of AKI on the discharge letter and arrange follow up if required.

Paediatric Acute Kidney Injury Alert Review Sticker						
Patient's Name:				Hospital No:		
Date:	Time:	Creatinine:	AKI Stage:	Signature:		
Investigate Potential Causes	Present? (tick)	Treatment / Comments	ACTIONS	Done or N/A	Comments	
Volume Depletion			MONITOR PEWS inc BP, strict fluid balance, unanalysis, daily weight, repeat creatinine and electrolytes			
Sepsis			MAINTAIN CIRCULATION Treat hypoperfusion and re-assess			
Medication / Contrast			MINIMISE KIDNEY INJURY Review, monitor and adjust medication eg NSAID, ACEi, ARB, diuretics, antibiotics			
Urinary Tract Obstruction		Consider USS	MANAGE Senior review Investigate as per AKI guideline USS if no clear cause or not improving			
Intrinsic Renal Disease		D/W Feed, Nephrology	DISCUSS WITH PAED. NEPHROLOGY Intrinsic renal disease, vasculitis, HUS Background of CKD or renal transplant Severe electrolyte abnormalities AKI stage 2 or 3			
Other			INFORM Patient / Parent / Carer = www.infokid.org.uk BP = discharge summary to include AKI stage, cause, treatment, and renal follow-up plan			

PAEDIATRIC AKI ALERT STICKER



Please note there are limitations with the AKI score which is calculated directly from the creat result by biochemistry. If in doubt please calculate an eGFR (height x40/ creat)

Our local renal specialists are:

Dr Angela Hall Associate Specialist

Dr Sudarsana De Consultant Paed Nephrologist (shared post with Nottingham)

They can be contacted via UHL switchboard.