SPRUN

SCOTTISH PAEDIATRIC RENAL AND UROLOGY NETWORK

Neonatal Acute Kidney Injury

NOTE

This guidance is not intended to be interpreted or used as a standard of care. Standards of care are determined based on the full range of clinical information available for an individual case and may change as scientific knowledge, technology, and clinical practice evolve. Following the recommendations in this guidance does not guarantee a successful outcome in every situation, nor does it encompass all appropriate methods of care or exclude other acceptable approaches that may achieve the same results. Clinical judgement remains the responsibility of the healthcare professional(s) involved in the patient's care. Clinicians must also be mindful of the requirements of patient autonomy and patient-centred care, which may involve a patient opting (after discussion of the available treatment options) for a treatment which is not the treatment recommended in the guidance. Where treatment involves deviation from national guidance, or local guidance derived from them, this should be fully documented in the patient's records at the time the decision is made.

This document has been prepared by NHS Public Services Delivery Scotland (PSDS) on behalf of the Scottish Paediatric Renal Urology network (SPRUN). Accountable to Scottish Government, PSDS works at the heart of the health service providing national strategic services to the rest of NHS Scotland and other public sector organisations to help them deliver their services more efficiently and effectively. SPRUN is a collaboration of stakeholders involved in the care of patients with renal and urological conditions, who are supported by an NSS Programme Team to drive improvement across the care pathway.

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Rationale	The network was established to support and develop Nephrology services throughout Scotland in improving standards of clinical care for patients. The network supports the delivery of evidence based, patient centred care through the development and implementation of clinical guidance, care pathways and information resources utilising a once for Scotland approach. Neonatal Acute Kidney Injury (AKI) is recognised as a risk factor for chronic kidney disease (CKD) and end stage renal disease (ESRD) in later childhood and beyond. An increasing evidence base demonstrates the long term renal consequences of neonatal AKI, underscoring the need for guidance to support consistent monitoring and follow up of affected patients. The provision of this guidance will promote equitable, evidence based care and ensure a standardised approach to review and surveillance across the network.
Scope	Neonatology, General Paediatric and Paediatric Nephrology teams throughout network, looking after neonates with acute kidney injury.
Approval process	This document was approved by the SPRUN Steering Group on 27 March 2026. Membership available in Appendix 2.

Contents

1. Background 4
2. Risk factors 4
3. Definition of acute kidney injury (AKI) 5
4. Classification 5
5. Aetiology 6
6. Evaluation of AKI 6
7. Management of AKI 7
   Aims: 7
   Established kidney injury: 7
   Anticipated problems: 8
8. Management after AKI 10
9. Follow up 111
10. References 122

Appendix 1 – Creatinine reference ranges 15
Appendix 2 – Steering group membership 16

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1. Background

Acute kidney injury (AKI) in the neonate or infant is independently associated with increased mortality and increased length of stay. Preterm infants are at increased risk of chronic kidney disease (CKD) with infants with low birthweight (LBW) and small for gestational age being novel risk factors. Emerging data studies in adults and children have demonstrated that a single episode of AKI is associated with an increased risk of CKD and ESKD, with more severe AKI conferring greater risk.

60% of Nephrogenesis occurs in 3rd Trimester
Glomerular Filtration Rate (GFR) is not at equivalent adult levels until 6-12 months of age
Nutrition and growth key to optimising nephrogenesis and renal function

2. Risk factors

- Preterm <34 weeks
- Very low birthweight and Intra-uterine growth restriction.
- Hypoxia secondary to respiratory failure or asphyxia, Apgar score <5 at 10 minutes of age and therapeutic cooling.
- Congenital diaphragmatic hernia – peri-operatively
- Critical cardiac defects – requiring neonatal cardiac surgery
- Infants treated with Extracorporeal Membrane Oxygenation (ECMO).
- Hypo-perfusion secondary to sepsis, patent ductus arteriosus (PDA), necrotising enterocolitis (NEC) cardiac disease, and high mean airway pressure impairing venous return.
- Dehydration from decreased fluid intake or increased fluid losses.
- Prenatal and postnatal exposure to nephrotoxic medication.
- Anomalies of the kidney and urinary tract.

3. Definition of Acute Kidney Injury

Acute decline in kidney function resulting in fluid and electrolyte imbalances/abnormalities and accumulation of waste products.

- Serum creatinine is traditionally used to classify AKI. This has limitations as Serum creatinine at birth reflects maternal value.
- Urea is less reliable than creatinine as an index of GFR. It is more dependent on protein catabolism than a reflection of hydration.
- After 14 days creatinine should fall to within normal reference range.

Rises in serum creatinine are delayed by 48-72 hours after insult

A significant amount of function has been lost before serum creatinine rise is seen

4. Classification

This is a classification not a diagnosis but should be used to trigger review.

Stage	Serum Creatinine (SCr)	Urine Output	pRIFLE
1	SCr rise ≥1.5-1.9 x reference SCr within 7day	<0.5ml/kg/hr for 6-12 hours	RISK OF AKI
2	SCr rise ≥ 2.0-2.9 x reference SCr	<0.5ml/kg/hr for >12hours	INJURY
3	Rise SCr ≥3 x reference SCr OR Receipt of dialysis	<0.3ml/kg/hr for ≥ 24 hours Or anuria for ≥12 hours	FAILURE
Reference SCr = lowest previous serum creatinine			

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5. Aetiology

In clinical practice, these causes frequently overlap; however, this framework can be helpful when evaluating the underlying cause.

Pre-renal	Renal	Post-renal
Hypovolemia, (dehydration)	Severe hypoxic insult eg Hypoxic Ischaemic Encephalopathy, critical cardiac surgery	Obstructive Uropathy - eg Posterior urethral Valve, urethral stricture,
Congenital cardiac disease PDA, hypoxic cardiomyopathy, obstructive cardiac lesion eg Co-arctation	Nephrotoxins eg NSAIDs, Aciclovir, Gentamicin, Vancomycin	Urate nephropathy
Reduced renal perfusion eg Sepsis, NEC	Renal vascular thrombosis	
Haemorrhage Twin to twin transfusion, feto maternal haemorrhage, coagulopathy	Congenital renal disease Multi-cystic dysplastic kidney disease Polycystic kidney disease	

6. Evaluation of AKI

History and examination

- Detailed medical history, including consideration of risk factors – as noted above, including current drug history.
- Review maternal medications for nephrotoxins.
- Review antenatal scans for suggestion of congenital anomaly and any reference to oligohydramnios.
- Thorough examination to assess fluid status which includes urine output and fluid balance.
- Review any weight change (must have up to date weight).
- Observations including blood pressure.

Investigation

- Urea and electrolytes, including Bicarbonate, Bone profile - Phosphate, Calcium, Magnesium, LFTs, Albumin, FBC.
- Renal Ultrasound - Any structural abnormality, evidence of dysplasia or evidence of obstruction.
- Renal Doppler Ultrasound (only if available, to exclude thrombosis).

7. Management of AKI

Aims:

1. **Identify and correct treatable causes**
   - Treat hypoxia, hypotension, hypovolemia.

2. **Minimise additional insult**
   - Review drug chart for nephrotoxic medications, stop, adjust dose / frequency of renal excreted medications (refer to local health board monographs, BNFc or Guys and St Thomas' Formulary).
   - If unable to stop gentamicin / vancomycin, doses should not be given until trough level demonstrates that the drug has been sufficiently cleared. Correct subsequent dose for renal function and adjust according to levels.

3. **Investigate and treat possible causes**
   - Collect urine for culture and microscopy – most commonly will be a catheter specimen or if not catheterised a clean catch specimen.
   - Renal USS (see notes above).

Failure to produce diuresis in response to fluid challenge and furosemide should be treated as having established acute kidney injury.

Established Kidney Injury:

1. **Fluid restriction**
   - Use pre-AKI weight or estimated weight, adjusted for fluid overload.
   - Adjust fluids carefully to fluid status.
   - Replace total fluids as urine output and be mindful of insensible losses (30-60ml/kg/day; consider gestation, age and humidity when calculating insensible losses).
   - Review fluid balance 6-8 hourly with replacement of losses every 6-8 hours.
   - Evaluate all fluids going in. Attempt to concentrate nutritional fluids (including TPN) and any other IV infusions. Need to ensure normoglycaemia and minimise catabolism.
   - This will likely require central IV access.

Other measures to consider in contact of fluid overload and oliguria

- Furosemide ** 1-2mg/kg (dose can be repeated 6 hourly if needed). (Refer to local health board monographs, BNFc or Guys and St Thomas' Formulary)

Next steps if no improvement

- Discuss with nephrology.
- Consider Furosemide Infusion.

- If Albumin <20g/L - consider Albumin (20%) and Furosemide infusion (Refer to local health board monograph).

** Furosemide acts on the tubule. Therefore, if the GFR is reduced larger doses are sometimes required. Response should be reviewed before giving further doses. Toxicity can occur with a total daily dose of 10mg/kg.

2. Nutrition

- Acute kidney injury is a hypercatabolic state.
- Many babies with AKI are premature and will have higher nutritional requirements than term infants.
- Nutritional intake may be restricted due to overall fluid limitation; however adequate glucose delivery should be maintained to prevent hypoglycaemia.
- Consultation with neonatal pharmacist if on parenteral nutrition (PN), neonatal dietitian if on feeds, should be undertaken to ensure requirements are being met and in longer term, growth is achieved.

3. Monitor urine output

- Consider inserting urinary catheter to monitor urine output.
- Calculate urine output in ml/kg/hr.
- Review of fluid balance 6-8 hourly.
- At least daily weights if possible.
- Review total input and total output for each day aiming for even balances and avoid cumulative positive balances.

4. Monitor electrolytes

- Daily U+E with bicarbonate, bone profile for phosphate, calcium, magnesium.
- (Venous sample preferable but recognise capillary sample more practical).

Anticipated problems:

Hyperkalaemia

- For all infants with AKI, evaluate the need for fluids containing potassium. **It is harder to remove once it is given**. (eg PN, blood products).
- Neonates can cope with higher potassium levels.
- If on oral feeds, consider introducing renal feed for persistent K > 6.5mmol/l
- Consider sodium bicarbonate.

Hypocalcaemia

- Correct hypocalcaemia as per local guidance.

Hyponatraemia

- Most likely to be due to fluid overload.
- Review fluid status and restrict fluid intake.

Metabolic acidosis

- Consider sodium bicarbonate if HCO_3<19mmol/l.
- Oral supplements preferred – 1-2mmol/kg/day in divided doses.
- IV Sodium Bicarbonate correction can be given; however, consider the volume of infusions.
- Maximise acetate in total parenteral nutrition.

Contact nephrology (Local Renal link or on call Renal consultant at RHC Glasgow)

- Renal function not improving despite intervention.
- Oedematous state needing IV Albumin or furosemide infusion.
- Significant hypertension - as per local neonatal hypertension guidance.
- Underlying congenital renal anomalies with AKI.

Kidney replacement therapy (KRT)

KRT is challenging in neonates and is often limited by weight, access and co-morbidities and requires discussion with nephrology.

Indications
Failure of conservative management

- **Uraemia**
- **Sustained hyperkalaemia**
- **Hyponatraemia and fluid overload, in particular where nutrition is likely to be compromised**
- **Refractory metabolic acidosis**

For some neonates, aggressive intervention may not be justified, or feasible. Decision to treat should be made on an individual basis. KRT in neonates is challenging and brings a high risk of complication; however, it is possible in cases. The decision to commence KRT and feasibility in undertaking is individualised to each patient and requires a wider discussion with neonatology and nephrology.

8. Management after AKI

- Watch for polyuria and replace losses as needed to avoid hypovolaemia.
- Once stable re-assessment of fluid balance and bloods can be decreased as necessary.
- Monitor blood pressure.
- For all patients, creatinine should be checked shortly before discharge.

9. Follow up

Why is this important?
Detecting CKD in those at risk is important. Early stage disease is often subtle and asymptomatic. As these infants grow into adulthood, early identification allows interventions that can slow progression and ideally prevent the need for significant renal support, such as dialysis or transplantation. Early detection helps optimise long-term health outcomes, and several emerging therapies offer additional opportunities to limit CKD progression.

Recommendations:

AKI stage 1 with normalisation of creatinine and no other risk factors do not require follow-up.

Risk Factor for CKD AKI 2 or 3 – Creatinine now normalised No evidence of kidney disease at discharge	**Kidney Surveillance** Review at 6months - Serum Creatinine / Cystatin C - Urine Protein Creatinine Ratio - AKI Parent/Carer Leaflet Interim assessment if felt necessary Review at 2 years - Serum Creatinine - Urine Protein Creatinine Ratio - Renal USS to review renal growth - Blood pressure
AKI 3	Review at 5 years - Serum Creatinine - Urine Protein Creatinine Ratio - Renal USS to review growth - AKI Parent/Carer Leaflet
Evidence of kidney disease at discharge Any of the following - BP >95th centile* - Elevated Creatinine - CAKUT (Congenital Anomalies of Kidney and Urinary Tract) - Nephrocalcinosis - Proteinuria * Refer to local Neonatal Hypertension Guidance	

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Discharging from follow-up **AKI 2** Normal eGFR >90ml/min/1.73m^2 No evidence of kidney dysfunction at 2 years **AKI 3** Normal eGFR >90/ml/min 1.73m^2 Good growth on renal USS No evidence of kidney dysfunction at 5 years	 AKI Parent/Carer Leaflet AKI Parent/Carer Leaflet Consider further review at 10 years and discharge then if no evidence of dysfunction.

Discussion with Nephrology (local renal link or on-call renal consultant available via switchboard at RHC, Glasgow) at any point

- Hypertension
- Persistent proteinuria
- Renal USS showing reduced growth of kidneys
- At age 2 year eGFR <90mls/min/1.73m^2

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Appendix 1 - Creatinine reference ranges

Introduction
Differing methods of creatinine measurement, varying between enzymatic and Jaffe methodology, as well as different manufactures (Abbott, Roche) are employed by biochemistry departments throughout Scotland. Creatinine reference ranges are region specific and not currently standardised due partly to differing measuring techniques.

Discussions are ongoing within biochemistry departments across Scotland to seek agreement on standardising reference ranges for the same analysers.

This is a nationwide issue with biochemistry departments seeking similar methods of analysis and then standardising reference ranges. Once reference ranges are chosen, these have to be calibrated with local populations, so standardising reference ranges is not a straightforward process.

In NHS Greater Glasgow and Clyde (NHS GGC) the intention is to have agreement to amend reference ranges in keeping with Think Kidney ranges with the intention is for NHS GGC to move over to these soon.

At present, NHS GGC reference ranges remain in place.

Please refer to local preterm reference ranges if available.

Think Kidney reference ranges below

Age Group	Male (Creatinine µmol/l)		Female (Creatinine µmol/l)	
	Lower (LLRI)	Upper (ULRI)	Lower (LLRI)	Upper (ULRI)
0 - <14days	27	81	27	81
14d - <1yr	14	34	14	34
1 - <3yr	15	31	15	31
3 - <5yr	23	37	23	37
5 - <7yr	25	42	25	42
7 - <9yr	30	48	30	48
9 - <11yr	28	57	28	57
11yr	36	64	36	64
12yr	36	67	36	67
13yr	38	76	38	74
14yr	40	83	43	75
15yr	47	98	44	79
16yr	54	99	48	81
>16yr	Adult Range		Adult Range	
	59	104	45	84

Figure 1: Think Kidney Reference Ranges

At present, as reference ranges vary considerably throughout Health Boards in Scotland, we recommend reviewing creatinine values with Think Kidneys reference range in line with local ranges.

Appendix 2 – SPRUN Steering Group Membership

Name	Designation	Role	Area representing
Deepa Athavale	Consultant Paediatric Nephrologist	Lead Clinician/Chair	NHS Greater Glasgow & Clyde
Rozi Ardill	Consultant Paediatrician	Member	NHS Lothian
Susan Burns	Renal Data Manager	Member	NHS Greater Glasgow & Clyde
Claire Cowe	Renal Nurse Specialist	Member	NHS Grampian
Sheena Dunsmore	Trustee	Member	Third Sector (Kidney Kids Scotland)
Karen Elrick	Paediatric Renal Dietician	Member	NHS Greater Glasgow & Clyde
Fiona Graham	Paediatric Renal Dietician	Member	NHS Greater Glasgow & Clyde
Claire Haggerty	Renal Nurse Specialist	Member	NHS Greater Glasgow & Clyde
Liz Hunter	Consultant Clinical Psychologist	Member	NHS Greater Glasgow & Clyde
Angela Lamb	Paediatric Renal Pharmacist	Member	NHS Greater Glasgow & Clyde
Boma Lee	Consultant Surgeon / Urologist	Member	NHS Greater Glasgow & Clyde
Karen McFarlane	Manager	Member	Third Sector (Kidney Kids Scotland)
Ursula Monachan	Advanced Renal Nurse Practitioner	Member	NHS Greater Glasgow & Clyde
Robin Oswald	Consultant Paediatrician	Member	NHS Tayside
Peter Schulga	Consultant Paediatric Nephrologist	Member	NHS Greater Glasgow & Clyde
Jan Vedarajan	Specialty Doctor/ Link Paediatrician	Member	NHS Highland