POSITION STATEMENT

Recognition and management of acute kidney injury in children

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Abstract

Acute kidney injury (AKI) is common among hospitalized neonates and children and is associated with adverse short- and long-term outcomes. Prevention and early detection of AKI are necessary to limit injury progression and avoid complications. However, paediatric AKI is currently under-recognized in Canada and follow-up care is highly variable. This Canadian Paediatric Society statement reviews epidemiology and risk factors for paediatric AKI, strategies for its diagnosis and management in hospitalized children, the potential long-term risks of childhood AKI, and recommendations for follow-up surveillance after AKI. A separate section focuses on the nuances of neonatal AKI detection and management, due to the unique considerations applicable to this patient population.

Keywords: *Acute kidney injury; Acute renal failure; Chronic kidney disease; Neonatal nephrology*

Background

Acute kidney injury (AKI) is an abrupt decline in kidney function that occurs in 5% to 10% of hospitalized children and ~30% of neonatal and paediatric intensive care unit (ICU) admissions[1]-[5]. AKI frequently occurs in children without pre-existing kidney disease due to systemic illnesses and nephrotoxic exposures[6]. Childhood AKI is associated with adverse hospital outcomes (i.e., prolonged length of stay and ventilation, and mortality)[1][7][8], long-term sequelae (i.e., chronic kidney disease [CKD] and hypertension)[9]-[11], and high health care costs[12][13]. Prevention and early AKI detection are needed to limit AKI progression. However, childhood AKI is often detected late, missed, and poorly documented[5][14][15]. Few AKI survivors receive follow-up care[13][16][17]. Improving AKI care is a priority in international nephrology and critical care communities. This need led to the development of consensus recommendations on adult AKI care[18] and responses from multidisciplinary teams of childhood and neonatal AKI experts[6][19]. Consequently, standards and quality improvement targets were set for paediatric AKI that have been used to develop this position statement. Significant paediatric AKI research is based in Canada and emphasized here, along with recommendations applicable to the Canadian health care context.

AKI epidemiology

Childhood AKI frequently begins in the community and is detectable at admission[1][8]. Community-acquired AKI is commonly caused by volume depletion, whereas hospital-acquired AKI is often multifactorial. There are several patient, disease, and procedural risk factors for AKI in hospitalized children (**Table 1**)[6]. Several AKI risk factors are associated with intravascular volume depletion or decreased kidney blood flow, which can cause serum creatinine rise without kidney tissue damage (now called "functional" AKI and previously known as "pre-renal" AKI). When decreased kidney blood flow becomes severe or prolonged, kidney tubular damage occurs (now called "structural" AKI and previously known as "renal" AKI). Urinary tract obstruction affecting a solitary or both kidneys can also cause AKI.

All paediatric health care providers (HCPs) should be aware of AKI risk factors (**Table 1**) which should be assessed for in hospitalized children[6][19]. Children identified to be at high risk should have close monitoring of their kidney function, blood pressure (BP), urine output, and volume status (by clinical examination and cumulative fluid balance calculation)[20]. The methods of screening should be adapted to suit local health care system resources and specific AKI risk factors.

Table 1. Paediatric acute kidney injury (AKI) risk factors

High-risk diseases	High-risk states
• Congenital heart disease • Malignancy • Stem cell or solid organ transplant • Kidney disease • Urinary tract obstruction • Systemic infections • Liver disease • Diabetes and diabetic ketoacidosis • Sickle cell disease • Anorexia nervosa	• Intensive care unit (ICU) admission • Neonates • Major surgery • Trauma • Nephrotoxic medications • Iodine-based radiocontrast exposure • Dehydration • Diarrheal illnesses • Hypotension and shock • Rhabdomyolysis • Severe hemolysis

AKI definitions

The Kidney Disease Improving Global Outcomes (KDIGO) criteria are internationally accepted for paediatric AKI classification (**Table 2**)[21] and have been adapted for neonates[22]. Their definition is based on changes in kidney function markers (serum creatinine or urine output). Baseline creatinine (defined as a measurement within 3 to 6

months *before* hospitalization) is used to classify AKI severity (i.e., the magnitude of hospital creatinine elevation), although such measures are often missing for children[1]. When a baseline creatinine is missing, it can be estimated (see **Table 2**)[22]-[24]. There are limitations to using creatinine-based AKI definitions. Creatinine is a late marker of kidney damage (25% to 50% of kidney function is lost before creatinine rises[25]) and changes are delayed by 24 to 48 hours in AKI[26]. Creatinine concentration is affected by volume status, muscle mass, and certain medications (e.g., trimethoprim, cimetidine).

Table 2. Paediatric and neonatal acute kidney injury (AKI) classification

AKI stage	Paediatric (KDIGO) criteria[21]		Neonatal (modified KDIGO) criteria[22]	
	Serum creatinine	Urine output	Serum creatinine	Urine output
1	Increase 1.5 to 1.9 x baseline[a] within 7 days prior ***or*** increase of ≥26.5 µmol/L within 48 hours	<0.5mL/kg/h for 6 to 12 hours	Increase 1.5 to 1.9 x from baseline[a] (lowest value within 7 days prior) ***or*** increase ≥26.5 µmol/L within 48 hours	<0.5mL/kg/h for 6 to 12 hours
2	Increase 2 to 2.9 x baseline	<0.5mL/kg/h for ≥12 hours	Increase 2 to 2.9 x baseline	<0.5mL/kg/h for ≥12 hours
3	Increase ≥3 x baseline ***or*** ≥353.6 µmol/L ***or*** dialysis initiation ***or*** eGFR <35mL/minute/1.73m^2	<0.3mL/kg/h for ≥24 hours ***or*** anuric for ≥12 hours	Increase ≥3x baseline ***or*** ≥221 µmol/L ***or*** dialysis initiation	≤0.3mL/kg/h for ≥24 hours ***or*** anuric for ≥12 hours

[a] Missing baseline creatinine can be back-calculated using height-dependent (CKiD) or height-independent (Hoste) equations, assuming a normal estimated GFR (eGFR) of 120 mL/minute/1.73m^2 (or median age-specific eGFR normative values for infants ≤2 years)[23][24].

CKiD equation: Baseline SCr (umol/L) = (36.5 x height [cm]) / eGFR (mL/minute/1.73m^2)

Hoste equation: Baseline SCr (umol/L) = (9485.3 x Q) / eGFR (mL/minute/1.73m^2)

-- Q (boys; age in years) = 0.21 + (0.057 x age) – (0.0075 x age^2) + (0.00064 x age^3) – (0.000016 x age^4)

-- Q (girls; age in years) = 0.23 + (0.034 x age) – (0.0018 x age^2) + (0.00017 x age^3) – (0.0000051 x age^4)

CKiD Chronic Kidney Disease in Children Study; eGFR Estimated glomerular filtration rate; H hours; KDIGO Kidney Disease Improving Global Outcomes; Q Serum creatinine normalization factor; SCr Serum creatinine

AKI recognition

Less than one-half of AKI episodes are recognized among hospitalized children[5][14][15][27]. Creatinine is measured infrequently in children at risk for AKI[28]. When AKI goes unrecognized, children may continue to receive nephrotoxic medications or inappropriate intravenous (IV) fluids causing AKI progression. Further, AKI under-recognition is a barrier to implementing follow-up. Less than one-quarter of discharge summaries document AKI occurrence[5][29]-[31] or key information regarding AKI severity, medication changes, and follow-up recommendations[3]. Strategies to increase paediatric AKI recognition include: provider education, increased serum creatinine and urine output monitoring among high-risk patients, and electronic health record (EHR) systems to estimate missing baseline creatinine, display age-specific creatinine reference ranges, and alert providers to AKI occurrence.

AKI prevention

AKI can be prevented if risk factors are recognized early, and prompt action is taken to mitigate injury (**Table 3**[32]-[41]). Baseline kidney function should be ascertained, and other AKI risk factors (**Table 1**) addressed before planned major surgery or nephrotoxic medication use. For ICU patients, the renal-angina index (a clinical prediction tool for paediatric AKI) is validated for the prediction of severe AKI within 72 hours of ICU admission[42]-[44]. The KDIGO AKI care bundles help prevent AKI development and progression[45]-[47]. These bundles focus on maintaining effective circulating volume by restoring intravascular volume and optimizing hemodynamic status, avoiding nephrotoxins, and closely monitoring urine output and fluid balance. Nephrotoxin stewardship programs, such as Nephrotoxic Injury Negated by Just-in-Time-Action (NINJA)[40][41] reduce nephrotoxin use and AKI incidence among hospitalized children and neonates[40][41][48]. However, targeted therapies to prevent AKI among hospitalized children are lacking, which is an important unmet need.

In childhood Shiga toxin-producing *Escherichia coli* infection, the risk of hemolytic uremic syndrome is 15%[49]. Antibiotics and antimotility medications should be avoided to limit Shiga toxin release. Volume expansion with IV fluids during the diarrheal phase is associated with a lower risk for AKI progression[49][50]. Close monitoring of kidney and hematological parameters is warranted because hemolytic uremic syndrome typically develops within 1 to 2 weeks of infection.

Table 3. Evidence-based strategies to prevent acute kidney injury (AKI)

Restore and maintain effective circulating volume	*Intravenous fluids* • If hypovolemic, fluid administration to restore intravascular volume. Use caution for signs of fluid overload or heart failure[32][33] • Hyperhydration is often used in rhabdomyolysis, tumor lysis syndrome, or high-risk nephrotoxin exposures (e.g., intravenous acyclovir) • Among adults, balanced crystalloids are associated with better kidney outcomes versus 0.9% saline[34][35]. In children, balanced crystalloids have not been proven to be superior[36][37], but randomized controlled trials are being conducted[38] *Inotropes/vasopressors* • In fluid-refractory shock, inotropes or vasopressors should be considered as indicated • There is insufficient evidence supporting 'kidney dose' (i.e., low-dose) dopamine or fenoldopam to prevent paediatric AKI[39]
Avoid unnecessary nephrotoxins	• Avoid unnecessary nephrotoxins in children at-risk for AKI or with AKI[40] • If nephrotoxins are necessary, carefully monitor kidney function and therapeutic drug levels • Common nephrotoxins[41]: non-steroidal anti-inflammatory drugs (NSAIDs), specific antibiotics (gentamicin, vancomycin, piperacillin-tazobactam), antivirals (acyclovir), chemotherapy, immunosuppression (tacrolimus), and radiocontrast
Kidney function monitoring	• All children at risk for AKI should have serial kidney function, blood pressure, urine output, and cumulative fluid balance monitoring • Early AKI detection provides opportunities to intervene and prevent AKI progression and complications

AKI management

Effective AKI management can be delivered in community or tertiary care hospitals. Focused history-taking, physical examination, laboratory, and imaging investigations (**Table 4**) can identify AKI causes[32]. Although AKI is often multifactorial, causes should be identified and addressed. The goal of supportive AKI care is to identify kidney dysfunction early, mitigate progression, and minimize complications. Paediatric nephrologist consultation is recommended for children with: severe (KDIGO stage 2-3) or worsening AKI, pre-existing chronic kidney disease (CKD), or suspected glomerular or tubulointerstitial disease (see **Table 4** for signs). Transfer to a tertiary care hospital may be needed for ongoing care.

Table 4. Diagnostic evaluation for paediatric acute kidney injury (AKI)

AKI cause	History	Physical exam
Functional (reduced kidney blood flow – may cause tissue damage)	• Volume depletion (e.g., vomiting, diarrhea) • Reduced fluid intake • Cardiac disease • Liver disease	• Weight loss • Delayed capillary refill • Tachycardia • Hypotension • Signs of heart or liver failure
Structural (kidney tissue damage)	Immune-mediated glomerular disease: • Gross hematuria, edema • Joint swelling, arthralgia, rash, uveitis • Constitutional symptoms • Cough, hemoptysis, epistaxis • Antecedent illness (e.g., pharyngitis) • Bloody diarrhea (i.e., hemolytic-uremic syndrome) Tubulointerstitial disease: • Shock or prolonged hypotension (i.e., acute tubular necrosis) • Trauma or myalgia (i.e., rhabdomyolysis) • Nephrotoxin exposures	• Hypertension • Peripheral edema • Pulmonary edema • Rash or mucosal ulcers
Post-kidney obstruction (solitary or both kidneys)	• Reduced urine output • Poor urinary stream • Suprapubic or flank pain	• Bladder distention • Abdominal mass

Investigations (for all AKI patients):

- Creatinine, urea, sodium, potassium, bicarbonate, calcium, phosphate, magnesium, albumin
- Urinalysis, urine microscopy, spot urine protein:creatinine or albumin:creatinine ratio
- Consider renal doppler ultrasound to exclude obstruction and thrombosis

Investigations (for suspected glomerulonephritis):

- C3, C4, ANA, dsDNA, ASOT, ANCA, and anti-GBM antibodies

Investigations (for suspected hemolytic-uremic syndrome):

- LDH, haptoglobin, bilirubin, peripheral blood smear
- Stool culture or Shiga toxin PCR

Other investigations:

- Complete blood count, differential, reticulocyte count (infection, hemolysis)
- Creatinine kinase, uric acid (rhabdomyolysis, tumour lysis)
- Urine sodium and creatinine (for fractional excretion of sodium [FENa], to help differentiate functional [typically <1%] versus structural AKI [typically >2%])
- Urine eosinophils and beta-2-microglobulin (tubulointerstitial nephritis)
- Urine culture (urinary tract infection)
- Kidney biopsy (considered for severe or prolonged AKI with suspected structural etiology after paediatric nephrologist consultation)

ANA Antinuclear antibody; ANCA Antineutrophil cytoplasmic antibodies; ASOT Antistreptolysin O titer; C3 and C4 Complement component 3 and 4; dsDNA Anti-double stranded DNA antibodies; Anti-GBM Anti-glomerular basement membrane antibodies; LDH Lactate dehydrogenase; PCR Polymerase chain reaction

Fluids and electrolytes

During AKI, the goal is to restore and maintain euvolemia (**Table 5**)[51]. Conventional paediatric fluid prescribing rules (e.g., the ‘4-2-1’ Holliday-Segar equation) should not be used in AKI due to dysregulated fluid homeostasis. Daily weights and urine output should be monitored. Urinary catheterization can be considered to accurately measure urine

output or relieve lower urinary tract obstruction. Cumulative fluid balance calculations evaluate the appropriateness of urinary output relative to fluid intake. Fluid overload is a pathological state of positive cumulative fluid balance that often accompanies AKI[52]. Cumulative fluid balance >10% to 20% is associated with mortality and delayed AKI recovery[32]. Cumulative fluid balance can be calculated using fluids ins and outs or change in body weight (**Table 5**) [32].

Table 5. Fluid management in paediatric acute kidney injury (AKI)

Volume status	Goals	Fluid prescription
Hypovolemic	Prompt restoration of circulating volume	• Fluid resuscitation to restore intravascular volume • Consider inotropes or vasopressors for critically ill children with fluid-refractory hypotension or shock • Once effective circulating volume is restored, fluid management should maintain euvolemia
Hypervolemic	Fluid restriction ± removal	• Calculate cumulative fluid balance[a] • Fluid restriction • Loop diuretics (e.g., furosemide) for fluid overload or symptomatic edema when fluid restriction alone is insufficient • Severe fluid overload (>10% to 20%) unresponsive to diuretics may require kidney replacement therapy (intermittent dialysis or continuous kidney replacement therapy). Consult paediatric nephrology
Euvolemic	Maintain euvolemia	• Total daily fluid intake should = urine output + insensible losses[b] ± other major losses (e.g., stool, emesis) • Example: Intravenous or oral intake = insensible losses[b] + urine output replacement (± other losses) 1mL:1mL using a crystalloid solution with a sodium concentration similar to what is being replaced (e.g., urine) • Strict intake/output monitoring, daily weights, and cumulative fluid balance calculation

[a] Cumulative fluid balance can be calculated using:
1) fluids ins and outs
% cumulative fluid balance = 100 x (sum of daily fluid ins [L] – sum of daily fluid outs [L]) / admission weight [kg]
2) change in body weight
% cumulative fluid balance = 100 x (current weight [kg] – admission weight [kg]) / admission weight [kg]

[b] Insensible losses are ~400 mL/m^2/day (or 20 mL/kg/day) for children and ~600 mL/m^2/day (or 30 mL/kg/day to 40 mL/kg/day) for neonates. Insensible losses should be replaced with oral intake or an isotonic intravenous crystalloid solution (e.g., balanced crystalloid or 0.9% saline).

Fluid overload is addressed by restricting fluid intake and/or administering diuretics. Repeated diuretic administration is not recommended if the patient is unresponsive. To manage fluid overload, the aim is to achieve a negative daily fluid balance.

Serum electrolytes should be monitored daily during AKI episodes because hyperkalemia, hyperphosphatemia, hypocalcemia, and metabolic acidosis are common. If serum potassium (non-hemolyzed sample) or phosphate are high, dietician consultation, dietary restriction, and/or ‘binder’ medications (e.g., calcium carbonate for hyperphosphatemia or sodium polystyrene sulfonate for hyperkalemia) may be used. Bicarbonate supplementation may be used for severe metabolic acidosis. In severe AKI, kidney replacement therapy (KRT) may be needed to correct fluid or electrolyte derangements. Discussion of KRT[53][54] is beyond this statement’s scope. A paediatric nephrologist should be consulted early in cases of severe, progressive AKI because decisions regarding KRT initiation and modality are patient- and centre-specific[55].

Blood pressure

We recommend regular BP monitoring during AKI. Hypertension is common due to fluid and sodium retention. Hypertensive patients should receive low-sodium diets. If fluid overload is present, hypertension may improve with fluid restriction and diuretics. Short- or long-acting antihypertensive medications may be needed, and medication choice is case-dependent.

Nutrition

Significant catabolism occurs during AKI. Nutritional management is complex, particularly for infants with dietary and fluid restrictions. Dietitian consultation is recommended. Specialized formulas or parenteral nutrition may be needed to meet caloric and protein requirements during AKI.

Medications

Unnecessary nephrotoxic medications[41] or radiocontrast should be avoided during AKI[56]. Care providers should be aware of kidney-excreted medications that may require adjustment based on estimated glomerular filtration rate (eGFR). Consultation with local formularies and clinical pharmacists is recommended during paediatric AKI. Therapeutic drug monitoring may be warranted.

Long-term risks of childhood AKI

Recent studies have shown strong associations between childhood AKI and development of recurrent AKI, CKD, hypertension, adverse neurodevelopmental outcomes, increased health care expenditure, and mortality[9][13][57]-[59]. Among 4173 paediatric AKI survivors in Ontario, 16% developed CKD, 2% kidney failure, 17% hypertension, and 6% recurrent AKI over median 10-year follow-up[60]. The risks of developing kidney failure, CKD, and hypertension were 2 to 12-times higher among AKI survivors (versus hospitalized children without AKI). In a systematic review of ten paediatric AKI studies, the pooled long-term incidence of abnormal glomerular filtration rate (GFR) (<90 mL/minute/1.73m^2) was 28% and kidney failure was 0.4%[61]. This level of risk underlines the need for kidney health surveillance after childhood AKI. Children with stage 2-3 AKI, incomplete functional recovery (serum creatinine persisting ≥1.5 x baseline[62][63]), or other CKD risk factors (cancer treatment, heart disease) are at highest risk, warranting closer post-AKI monitoring[9]-[11][64]-[66].

AKI follow-up care

AKI follow-up care in Canada is inadequate. Low rates of AKI detection, poor AKI documentation, lacking provider and patient awareness of AKI long-term effects, limited post-AKI follow-up guidelines, and poor access to paediatric nephrology clinics are contributors. Strategies to improve post-AKI follow-up include:

- increasing AKI recognition (through EHR alerts and provider education)
- improving AKI documentation in discharge summaries (including information on AKI occurrence, severity, duration, recovery, suspected cause, complications, and follow-up recommendations)
- improving communication with primary care providers, and
- educating patients and caregivers about AKI, its long-term risks, and the need for kidney health surveillance (**Table 6**).

Finally, increasing paediatric and nephrology workforce capacity through additional physician/nurse training, telemedicine services, and government funding is needed to implement post-AKI follow-up.

In Canada, most children with AKI do not have nephrologist follow-up or creatinine measured after hospital discharge[12][13][16][17][62]. Among 277 PICU admissions in Quebec, only half of AKI survivors had creatinine measured within the first year post-discharge[16]. Among childhood survivors of severe AKI in Ontario and Quebec, nephrologist follow-up is uncommon (<20% by 1 year) but most (>95%) have follow-up with other health care providers[13][17]. Thus, paediatric AKI follow-up strategies must include general paediatricians and primary care providers.

Optimal post-AKI follow-up strategies remain uncertain. The 2012 KDIGO guidelines suggest evaluation “3 months after AKI for resolution, new onset, or worsening of pre-existing CKD”[21]. More current evidence suggests that:

- The timing of post-AKI follow-up should reflect AKI severity, duration, extent of recovery at time of discharge, and CKD or hypertension risk factors (i.e., diabetes, obesity, heart disease, cancer, prematurity, or low birthweight).
- All children with severe AKI (stage 2-3 or KRT), acute kidney disease (AKD, defined as an AKI episode lasting 7 to 90 days), incomplete recovery, pre-existing CKD, or recurrent AKI should undergo a kidney health

assessment (**Table 6**[67][68]) within 1 to 3 months post-discharge. Referral to a nephrologist or paediatrician with nephrology experience may benefit these high-risk children.

- Children with transient stage 1 AKI with complete functional recovery should receive a kidney health assessment by a community paediatrician or primary care provider within 6 to 12 months post-discharge. After the first follow-up visit, the frequency and duration of subsequent kidney function and BP surveillance should be based on the individual child's CKD risk and local resources.

The risk of adverse outcomes is highest within the first 5 years following paediatric AKI, with approximately one-half of new CKD diagnoses occurring within the first year[9][60]. While supporting data are limited and consensus has not been established, children with AKI should be followed for at least one year after an episode to confirm complete kidney function recovery and normal BP. Annual (or more frequent) follow-up for up to 3 to 5 years should be considered for children who experienced severe AKI.

Table 6. Kidney health assessment and response

"ABCD" kidney health assessment	"4 M's" kidney health response
A: AKI history • Previous AKI occurrence, severity, duration, and recovery	**M:** Medication adjustment • Avoid unnecessary nephrotoxic medications • Acetaminophen is a preferred analgesic and antipyretic. Cautious short-term use of NSAIDs is reasonable for children with prior AKI and complete kidney function recovery
B: Blood pressure measurement • Use an appropriate technique, device, and cuff size[67]	**M:** Minimize high-risk exposures for at least 2 years following AKI • Avoid dehydration • Monitor kidney function before and after high-risk states • Adhere to "sick day" rules for future illnesses (i.e., hydration and withholding nephrotoxic medications)
C: Creatinine (and kidney function) measurement • Estimate glomerular filtration rate using the CKiD U25 creatinine or cystatin C[68] equation • Consider other labs (urea, electrolytes, bicarbonate) if kidney function is abnormal	**M:** Message patient and care providers • Inform patients of AKI • Educate patient/family about long-term kidney health • Inform primary care and other providers regarding AKI follow-up care recommendations
D: Drug list review • For nephrotoxic medications ± **D:** Determine proteinuria • First morning urine for urinalysis and protein:creatinine or albumin:creatinine ratio	**M:** Monitor for kidney disease and AKI recurrence • Repeat kidney health assessment at regular intervals *and* before and after high-risk states

AKI Acute kidney injury; CKiD Chronic Kidney Disease in Children Study; NSAIDs Non-steroidal anti-inflammatory drugs

Data drawn from references [6][18]

Neonatal AKI

AKI within the first month of life is common, occurring during up to 38% of NICU admissions, and is associated with increased mortality, hospital length of stay, and long-term CKD[2][69]-[72]. Unique neonatal kidney physiology increases AKI susceptibility, particularly among neonates with prematurity, low birth weight, perinatal asphyxia, or congenital heart disease[70][73]. Normal physiological changes in GFR and urine output after birth complicate neonatal AKI detection. A modified KDIGO definition is commonly used (**Table 2**)[22]. Urine output can be misleading due to impaired urinary concentrating ability and sodium wasting. Dietician consultation is recommended for neonates with fluid or electrolyte disorders because fluid restrictions, formula adjustment, or "pre-treatment" with binders may be needed. Breast milk is often a safe nutritional option during AKI. Care providers need to be aware of normal neonatal BP ranges to address hypo- or hypertension. All neonates with AKI should receive follow-up of their kidney function and BP (**Table 6**).

Care provider recommendations

1. Paediatric health care providers (HCPs) should be aware of acute kidney injury (AKI) risk factors (**Table 1**) and assess for these among hospitalized children and neonates. When risk is present, serum creatinine, blood pressure, and urine output should be monitored to detect AKI. AKI should be classified by Kidney Disease Improving Global Outcomes (KDIGO) criteria (**Table 2**).

2. AKI care involves treating reversible causes, preventing progression, and minimizing complications, including fluid overload. Consider nephrologist consultation for severe (KDIGO stage 2-3) or worsening AKI.

3. Children with stage 2-3 AKI, AKI duration ≥7 days, incomplete recovery, pre-existing chronic kidney disease (CKD), or recurrent AKI should have post-AKI follow-up. Decisions about the follow-up provider (nephrologist versus non-nephrologist) and timing should be individualized based on AKI severity, duration, recovery status, and other risk factors for CKD and hypertension (e.g., heart disease, diabetes, prematurity). The duration of post-AKI follow-up should also be individualized based on a child's risk factors and status at follow-up. A minimum post-AKI follow-up for 1 year (for less severe AKI with complete kidney function recovery and normal blood pressure [BP]) and up to 3 to 5 years in high-risk cases are recommended.

4. It is currently uncertain whether children with transient stage 1 AKI that completely recover need post-AKI follow-up. Until further evidence is available, it is recommended that they receive a kidney health assessment by a community paediatrician or primary care provider within 6 to 12 months post-discharge.

5. Post-AKI follow-up (**Table 6**) should include kidney health assessment, medication reconciliation, care provider communication, patient education, and implementation of strategies to prevent recurrence.

Policy recommendations

1. Hospitals should develop strategies to improve AKI recognition and care, including: 1) AKI risk assessment and detection protocols, 2) EHR-integrated AKI alerts, 3) AKI documentation standards, and 4) provider education (including risk assessment, volume management, and nephrotoxin avoidance).

2. Hospitals should monitor rates of nephrotoxin-associated AKI and consider implementing nephrotoxin stewardship programs. Hospitals should evaluate inpatient kidney function monitoring practices to identify whether high-risk patients are adequately screened.

3. Hospitals, community health organizations, and policy-makers should develop systems to facilitate community-based post-AKI follow-up, including improved access to laboratories with paediatric-trained personnel, validated paediatric-specific BP devices, and specialized post-AKI follow-up clinics, with sufficient nursing resources to implement education and follow-up.

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