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SYMPOSIUM ON PEDIATRIC NEPHROLOGY

Management of Acute Kidney Injury

Gurinder Kumar · Anil Vasudevan

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Abstract Acute kidney injury is common in hospitalized children and is associated with siginficant morbidity and mortality especially in critically ill children. A complete evaluation is necessary for all children with AKI as early recognition and treatment is paramount. Apart from clinical evaluation, urinalysis, biochemical investigations and imaging studies helps in the diagnosis of the specific cause of AKI and assessing its severity. Attention should be given to assessment of volume status and fluid administration because volume depletion is a common and modifiable risk factor for AKI. Prevention or prompt management of complications like fliud overload, hyperkalemia and metabolic acidosis improves outcomes. Immediate initiation of renal replacement therapy (RRT) is indicated in the presence of life threatening changes in fluid, electrolyte and acid-base balance. Other measures like treating the underlying cause of AKI, adapting dosage of drugs to renal function, treatment of infections and providing adequate nutrition is important. Children with AKI should be followed up as they are at risk for development of chronic kidney disease.

Keywords Acute kidney injury · Diagnosis · Evaluation · Management

Introduction

Acute kidney injury (AKI), formerly known as acute renal failure is characterized by an abrupt (<48 h) and sustained decline in glomerular filtration rate and an inability of kidneys to appropriately regulate fluid, electrolytes and acid-base homeostasis. Two similar definitions based on changes in serum creatinine and urine output for diagnosis and classification of AKI; The Pediatric Risk, Injury, Failure, Loss, End-Stage Renal Disease Criteria and the Acute Kidney Injury Network Staging System have been proposed [1, 2]. AKI is common in hospitalized children, especially in critically ill and is associated with adverse short and long term outcomes [3]. Even small increases in creatinine may be associated with morbidity and mortality. Early identification of children with AKI and also those who are at risk of AKI is crucial to provide appropriate and effective treatment to prevent or limit the disease intensity [4]. The clinical spectrum of AKI can range from asymptomatic with mild to moderate elevation in serum creatinine to anuric renal failure.

Etiology

Table 1 provides a list of the many different and diverse causes of AKI which has been traditionally divided into three categories: prerenal azotemia, renal injury, and post renal obstruction. The common causes of AKI in our country are volume loss leading to ischemic renal injury, infections and hemolytic uremic syndrome [5]. In many instances, such as in critically ill children, multiple factors are likely to be implicated [6].

Evaluation of Risk Factors

Identifying settings and factors that predisposes to AKI is an important part of evaluation. Children with sepsis, circulatory insufficiency, trauma, burns, major surgeries (cardiac surgery)

G. Kumar · A. Vasudevan (✉)
Department of Pediatric Nephrology,
St John's Medical College and Hospital,
John Nagar,
Bangalore, India
e-mail: anil.vasudevan@sjri.res.in

Table 1 Etiology of acute kidney injury

Prerenal	Renal	Postrenal
Decrease in true intravascular volume	Glomerular causes	Obstructive uropathy
Severe diarrhea	Post infectious glomerulonephritis	Ureteropelvic junction obstruction
Vomiting	Crescentic glomerulonephritis	Urethral obstruction
Burns	Hemolytic uremic syndrome	Posterior urethral valves
Hemorrhage	Chronic glomerular disease—lupus, membrano proliferative glomerulonephritis	Nephrolithiasis
Sepsis	Acute tubular necrosis	
Decrease in effective intravascular volume	Toxin mediated:	
Anaphylaxis	Endogenous toxins: Intravascular hemolysis, rhabdomyolysis, tumor lysis syndrome	
Septic shock	Exogenous toxins: Ethylene glycol, methanol	
Dengue hemorrhagic fever	Drugs induced:	
Cardiac failure	Nephrotoxic agents	
Medication	Hypoxic/ ischemic insult	
Indomethacin/angiotensin converting enzyme inhibitor/ angiotensinogen receptor blocker	Acute tubulointerstitial nephritis	
	Drug induced	
	Idiopathic	
	Vascular lesions	
	Renal vein thrombosis	
	Renal artery thrombosis	
	Hereditary renal disease	
	Autosomal recessive polycystic kidney disease	
	Alport syndrome	

and poisonings are at risk of developing AKI. Young age, dehydration, presence of co-morbidities including pre-existing chronic kidney disease, use of nephrotoxic medications or radiocontrast agents in the above setting further increases the risk of AKI.

Evaluation of the volume status by history, physical examination and laboratory tests is an important measure in the prevention of AKI.

Evaluation after Diagnosis of AKI

Prompt evaluation is necessary to determine the cause with special attention to reversible causes (*e.g.*, dehydration, obstruction), and assess the severity and complications of AKI.

History and Examination

Common clinical features of AKI are decreased or no urine output, fluid overload (edema, tachypnea, abdominal pain), hypertension (headache, vomiting, blurred vision, seizures, hypertensive left ventricular failure) and symptoms of uremia and dyselectrolytemias. Non-oliguric AKI is commonly seen in toxin/medication mediated acute tubular necrosis while anuric renal failure is seen in children with urinary tract obstruction, renal cortical necrosis, bilateral renal vein thrombosis and severe dehydration. Clinical features of volume depletion suggest ischemic acute tubular necrosis. Edema, cola-colored urine and hypertension suggest a glomerular cause. History of pyoderma or acute pharyngitis preceding the onset of hematuria, edema and oliguria by few weeks suggests post infectious glomerulonephritis. A critically ill child with fever, jaundice, pallor and hepatosplenomegaly may have AKI secondary to malaria or leptospirosis. Rash or arthritis suggests vasculitis; ascites or jaundice suggests hepatorenal syndrome. A child with history of dysentery, and pallor, petechiae and oliguria is likely to have hemolytic uremic syndrome. A history of interrupted or poor urinary stream with palpable bladder suggests obstructive uropathy. A detailed medication history, including use of over the counter pain killers, herbal medications, aminoglycosides and chemotherapeutic agents provides a clue to possible cause of AKI.

Laboratory Evaluation

Laboratory investigations help define the cause of AKI and evaluate its severity (Table 2). Analysis of urine sediment and supernatant in a fresh sample for presence of cells, casts, crystals, protein and pigments provides important clues.

Nephrotic range proteinuria (urine albumin 3–4+) is usually seen in nephrotic syndrome, acute glomerulonephritis and rarely in medication-associated interstitial nephritis. Hematuria is suggestive of glomerular, vascular or other structural causes (*e.g.*, stones, trauma). Leukocyturia is seen in pyelonephritis, tubular injury and nephrolithiasis. An abnormality in urinary sediment strongly suggests intrinsic renal failure. Red blood cell casts are seen in glomerulonephritis, while acellular urine with/without hyaline or granular casts is seen in acute tubular necrosis or acute interstitial nephritis. Eosinophiluria is seen in up to 25 % cases of acute interstitial nephritis. Hemoglobin and myoglobulin in urine may suggest pigment induced AKI. Uric acid and calcium oxalate crystals are seen in tumor lysis syndrome and glycol poisoning, respectively. Drug induced crystals are diagnostic of sulfonamides and indinavir toxicity.

Several parameters, including osmolality, sodium concentration and fractional excretion of sodium are proposed to differentiate pre-renal injury from hypoxic/ischemic AKI. If fractional excretion of sodium and urea is low (<1 % for sodium and <35 % for urea), it suggests fluid-responsive prerenal AKI, whereas with acute tubular necrosis fractional excretion will be higher (>2 % for sodium and >35 % for urea). These indices will not be helpful in children who have received diuretics and in those with chronic kidney disease.

Blood urea and creatinine, electrolytes, pH and bicarbonates help in assessing severity and complications of AKI. While blood urea/creatinine ratio >20:1 suggests prerenal AKI, this ratio may be high in hypercatabolic states, steroid therapy and gastrointestinal bleeds. Hemogram, liver function tests, complement levels (C3, C4), ANA, calcium, phosphate, creatinine kinase, lactate dehydrogenase are required based on clinical diagnosis. Measurement of anion and osmolar gaps is important for evaluation of AKI due to ingestion of ethylene glycol [7].

Imaging

Ultrasonography is the most commonly used imaging modality while evaluating children with AKI. It is non invasive and is easily available. It plays an important role in the diagnosis of obstructive causes of AKI (*e.g.*, stones, posterior urethral valves). Enlarged kidneys are seen in acute interstitial nephritis, obstruction and renal vein thrombosis. The echogenicity of kidneys may be increased. A small-sized, contracted kidney suggests an acute on chronic kidney disease. Renal doppler helps in diagnosis of renal artery thrombosis and stenosis, but is observer dependent. Contrast computed tomography scanning is valuable for trauma assessment involving the kidney and for evaluation of stones/obstruction if the ultrasonography is inconclusive [8]. Magnetic resonance imaging is safer than conventional angiography to diagnose vascular causes, including renal artery stenosis and renal artery thrombus [9]. Intravenous pyleography is best avoided in children with AKI or at risk of AKI. Chest X ray is indicated in children with fluid overload or chest infection.

Renal Biopsy

It may be indicated in some cases of AKI for diagnosis and prognostication. Biopsy is indicated for specific diagnosis, which allows initiating specific treatment (*e.g.*, rapidly progressive glomerulonephritis, drug induced acute interstitial nephritis, systemic lupus, Henoch Scholein pupura). It may be indicated in cases where the cause of AKI is not easily identified, where it helps to confirm the diagnosis and provide prognostic information.

Biomarkers for Early Diagnosis of AKI

Diagnosis of AKI relies on estimation of renal function by serum creatinine level, which has many limitations. Serum creatinine concentrations do not change until significant injury and loss of renal function has occurred. Besides, serum creatinine levels can vary with muscle mass and with different methods of measurement. Biomarkers that are found useful in early diagnosis and prognosis are plasma panel (neutrophil gelatinase-associated lipocalin and cystatin C) and urine panel (neutrophil gelatinase-associated

Table 2 Characteristics of laboratory evaluation in AKI

Hemolytic uremic syndrome: Anemia/ Peripheral smear shows microangiopathic hemolysis/ Thrombocytopenia/Elevated lactate dehydrogenase
Sepsis: Anemia/Thrombocytopenia/ Leucocytosis/ Features of coagulopathy/ Blood culture may show growth of organisms
Rhabdomyolysis: Hyperkalemia/ Raised creatinine kinase/ Hyperphosphatemia/ Myoglobin in urine
Tumor Lysis Syndrome: Hyperuricemia/Hyperphosphatemia/Hypocalcemia/Uric acid crystals in urine
Ethylene Glycol Toxicity: High anion gap metabolic acidosis, Calcium oxalate crystals in urine
Acute Tubular Necrosis: Bland acellular urine with or without granular or muddy brown casts
Acute Interstitial Nephritis: Eosinophiluria and white blood cells
Acute Glomerulonephritis: Hematuria, Red blood cell casts, White blood cell casts/Proteinuria and Low C3, C4

lipocalin, interleukin-18 and kidney injury molecule-1) [10–13].

Management

Maintenance of Fluid Balance

Patients with AKI and at risk for AKI require careful attention to their hemodynamic status. The fluid and electrolyte prescription depends on the underlying volume status of children with AKI who may present with hypovolemia, euvolemia, or fluid overload and pulmonary edema. Children with low intravascular volume require fluid resuscitation and titration of ionotropes in concert with hemodynamic monitoring to avoid fluid overload. Children who are severely dehydrated are likely to be in prerenal azotemia. Aggressive fluid resuscitation with 10–20 mL/kg normal saline boluses (maximum 60–80 ml/kg) to re-establish intravascular volume is warranted. Further boluses may be given if hypovolemia persists on reassessment. If there is no urine output, a dose of frusemide (2 mg/kg IV) is given. Children with prerenal azotemia who do not pass urine after adequate fluid resuscitation or children with normal intravascular volume and oliguria are likely to have intrinsic AKI. Fluids prescribed should be restricted to insensible losses (400 ml/m^2/24 h) plus replacement of urine output and extrarenal losses (*e.g.*, diarrhea, vomiting) as continued fluid resuscitation despite increased intravascular volume in these children can cause harm. Meticulous attention is given to fluid balance after initial resuscitation in critically ill childen. Urinary and insensible losses are replaced with 0.45 % saline and 5 % dextrose respectively. Potassium containing fluids should not be given unless they are hypokalemic.

Those with overt fluid overload will require dialysis in addition to fluid restriction. Patients with polyuric AKI such as aminoglycoside toxicity are given appropriate fluid and electrolyte replacement based on urine output and serum electrolytes.

Ongoing fluid therapy is guided by weight, blood pressure, accurate input and output records, physical examination, nutritional needs and serum electrolytes. In critically ill children, central venous pressure monitoring may be necessary to guide fluid therapy. Cumulative fluid overload is calculated as follows:

$$[(\textit{fluid input in liters}) - (\textit{fluid output in liters})]/\textit{ICU admission weight in kilograms}$$

If the child has >10 % cumulative fluid overload, evaluation for renal support therapy should occur and if cumulative fluid overload is >20 %, initiation of renal support therapy should be strongly considered [14].

Treatment of Complications

Dyselectrolytemia

In children with AKI, serum potassium should be measured at least daily since hyperkalemia is a life threatening complication of AKI. Therapy for hyperkalemia is indicated if cardiac conduction abnormalities are noted or if potassium levels are >6 mEq/L and is summarized in Table 3. Children with AKI may have hyponatremia or less commonly hypernatremia. If hyponatremia is due to salt wasting, then appropriate fluid replacement is indicated. If hyponatremia is dilutional, fluid restriction or water removal by dialysis will correct the serum sodium level. Intravenous 3 % saline (5 ml/kg) given over 30–60 min should be considered in children with severe symptomatic hyponatremia. Hypocalcemia may be seen in AKI secondary to hyperphosphatemia, vitamin D deficiency, parathyroid resistance and hypoalbuminemia. IV 10 % calcium gluconate (1 mL/kg up to 10 mL) should be given over 30 min with continuous electrocardiographic monitoring. Hyperphosphatemia is a common electrolyte abnormality. Severe and persistent hyperphosphatemia should be treated with dietary phosphorus restriction and with oral phosphate binders (oral calcium carbonate or other calcium compounds) to prevent gastrointestinal absorption of phosphorus.

Metabolic Acidosis

Severe acidosis can be treated with IV/oral sodium bicarbonate with careful monitoring for fluid overload and worsening of hypertension. Refractory acidosis may require dialysis therapy. It is important to obtain total and ionized calcium levels prior to initiation of treatment to prevent

Table 3 Management of hyperkalemia

Drug	Dose
10 % Calcium gluconate	0.5–1 ml/kg IV over 10 min (1:1 dilution with cardiac monitoring)
Sodium bicarbonate (8.4 %)	1–2 mEq/kg IV over 10–30 min
Salbutamol	400 mcg by nebulizer
Glucose and insulin[a]	Glucose 0.5 gm/kg and insulin 0.1 unit/kg IV over 30 min
Potassium binders (sodium or calcium polystyrene sulfonate)	1 g/kg/dose every 4–6 h, with 20 % sorbitol orally or mixed with 250 ml of water as retention enema
Dialysis	If hyperkalemia is refractory to conservative therapy

[a] Can also be given as infusion

Table 4 Specific treatment for underlying cause of AKI

- Withdrawal or replacement of offending medication (*e.g.*, aminoglycosides, non-steroidal anti-inflammatory drugs)
- Anti microbial therapy (*e.g.*, malaria, leptospirosis, sepsis, urinary tract infection)
- Surgical intervention for obstruction (*e.g.*, removal of stones)
- Diuretics and alkalinization of urine in crush injury/myoglobinuria/hemoglobinuria
- Plasmapheresis in non diarrheal hemolytic uremic syndrome, rapidly progressive glomerulonephritis, vasculitis
- Pulse steroids in rapidly progressive glomerulonephritis, vasculitis, drug induced acute interstitial nephritis

hypocalcemic seizure/tetany (due to increased binding of calcium to proteins, which decreases ionized calcium).

Hypertension

The symptoms of hypertension depends on the degree of blood pressure elevation and often may be incidentally detected. The choice of anti hypertensive therapy is dependent on the symptoms and severity of hypertension. Mild hypertension is managed with fluid and salt restriction and diuretics. Calcium channel blocker is the oral antihypertensive drug of choice. ACE inhibitors are generally not used during the acute phase due to the potential for further lowering of GFR and hyperkalemia. In severe hypertension and/or encephalopathy, therapy with sodium nitroprusside or labetalol is indicated. Sublingual nifedepine is safe and effective for management of hypertensive emergency.

Infections

Children with AKI are at risk of infection due to their azotemia and underlying nutritional status. It is important to adhere to aseptic techniques while performing invasive procedures. Long term urinary catheterization is best avoided. If infection is suspected, appropriate samples for culture are obtained prior to initiation of antibiotics.

Nutritional Support

AKl can be associated with severe anorexia, and child may be at risk for malnutrition when AKI is prolonged. Child should receive appropriate maintenance calories and proteins. If sufficient calories cannot be achieved, while maintaining appropriate fluid balance, earlier initiation of dialysis is considered.

Treatment of Underlying Cause

Prompt recognition and management of underlying cause for AKI is important (Table 4).

Pharmacologic Therapy

Diuretics, renal dose dopamine (5 μg/kg/min) and mannitol are commonly used to prevent or limit AKI. However, these interventions have not shown to be clinically useful [15–17]. Diuretics increase urine output thereby easing management of fluid balance. Fenoldopam, a dopamine-1 receptor agonist that decreases vascular resistance and increases renal blood flow, has been shown to improve urine output in pediatric AKI, but has no effect on serum creatinine [18]. There is no specific therapy that promotes recovery in acute tubular necrosis [19, 20].

Table 5 Prevention of AKI

Condition	Prevention
Contrast nephropathy	Intravenous hydration (0.9 % NS @ 1.0 ml/kg/h 3–12 h before and 6–12 h after contrast media exposure
	Use of low/iso osmolar contrast agents in smallest possible volume
	Use of oral N acetylcysteine together with hydration
Tumor lysis syndrome	Controlling uric acid level by allopurinol and rasburicase
	Avoid nephrotoxic medications and volume depletion
Crush Injury	Hydration and alkalanization of urine to prevent myoglobin associated acute kidney injury
Nephrotoxic drugs	Avoid nephrotoxic drugs as much as possible and adjust the doses based on the estimated glomerular filtration rate
	Administer aminoglycosides as single daily dose
	Suggest using liposomal amphotercin B rather than conventional amphotericin B
Maintenance of fluid balance	Prevent hypotension which may lead to hypoperfusion of kidneys

Drug Dosing in AKI

Prescribing medications requires attention as many drugs are metabolized and/or excreted by kidneys. The dose and frequency of administration of drugs should be adjusted for the degree of renal failure, based on level of GFR. Drugs that are nephrotoxic (*e.g.*, aminoglycosides, NSAIDs, radiocontrast agent, vancomycin) should be avoided.

Renal Replacement Therapy (Dialysis)

The goals of dialysis in AKI are to correct metabolic abnormalities, restore and maintain fluid and electrolyte balance and maintain the physiological mileu to preserve organ function. The indications to initiate renal replacement therapy are not absolute. It is determined by a number of factors, including the underlying cause, rapidity of the onset of renal failure and the severity of fluid and electrolyte abnormalities (azotemia, hyperkalemia and severe acidosis). There is no single blood urea or creatinine threshold to initiate dialysis and the optimal timing of dialysis for AKI is not defined [21].

Renal replacement therapy may be provided by peritoneal dialysis, intermittent hemodialysis, or continuous renal replacement therapy (CRRT). Each of these modalities have their own advantages and limitations and comparisons are difficult due to effect of bias by indication [22, 23]. The need for rapid solute or fluid removal, hemodynamic status of the child, age of the child and availbilty of resources and expertise detetrmines the modality of choice for dialysis.

Peritoneal dialysis is a widely available form of renal replacement therapy. It is an inexpensive modality in developing countries and does not require highly trained staff or a complex apparatus. It can be easily performed even in sick children with unstable hemodynamic status. Peritoneal dialysis may be the easier option in infants and small children given their low blood volume and difficulties in obtaining vascular access. The major limiting factors are low efficiency of the procedure, especially in hypercatabolic states and its limitation in children with intra abdominal surgery.

Hemodialysis and CRRT requires specially trained staff and equipment. Hemodialysis is preferred in disease processes, which cause acute disruptions in homeostasis and require rapid removal of solutes and fluids such as severe hyperkalemia, drug ingestion and tumor lysis syndrome. Children with shock do not tolerate hemodialysis. In addition vascular access may be difficult in young children. CRRT is an expensive modality and is available only in few centers in our country. Its major advantage is that continuous adjustment of ultrafiltration is possible and is tolerated well by children in shock.

Prevention of AKI

Identifying children at risk for AKI helps in modifying factors and initiate specific interventions to prevent AKI (Table 5).

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