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Evaluation and management of acute kidney injury emergencies

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Acute kidney injury (AKI) presents unique challenges for physicians caring for patients in the Emergency department. The presentation and severity of AKI is variable. AKI, with severe life-threatening laboratory abnormalities, may present with multi-organ failure or with vague/minimal clinical findings. Therefore, physicians must have a high index of suspicion for AKI.

DEFINITION OF AKI:

AKI can be defined as a rapid decrease in glomerular filtration rate (GFR) over hours to weeks in the setting of previously normal renal function or preexisting renal disease. Acute renal failure (ARF) represents a subset of patients with AKI who require renal replacement therapy or dialysis and are more often critically ill.[1]

Several criteria exist to define and help diagnose AKI. One of the more commonly used is the RIFLE criteria, developed by the Acute Dialysis Quality Initiative which defines AKI severity based on serum Cr, GFR, and urine output (see table 1).[1] Serum creatinine levels lag renal injury and recovery, therefore creatinine has poor sensitivity and specificity for AKI by itself.[2] The pRIFLE criteria include modifications for pediatric patients based on estimated creatinine clearance (eCCl) as calculated by the Schwartz formula.[3-4] The KDIGO stages of AKI are also applicable to the pediatric population (table 2).[5]

Table 1: RIFLE and pRIFLE Criteria for Acute Kidney Injury

	RIFLE Criteria	pRIFLE (Pediatric) Criteria
R – Risk of renal injury	1.5x increase of baseline serum Cr (sCr) (50% increase) OR decrease in GFR >25% from baseline OR urine output of <0.5 ml/kg/hr for ≥6 hr	Estimated Cr Clearance (eCCl) decrease of 25% OR urine output of <0.5 ml/kg/hr for ≥8 hr
I – Renal injury	2x increase of baseline sCr OR urinary output <0.5 ml/kg/hr for ≥12 hr	eCCl decrease of 50% OR urine output of <0.5 ml/kg/hr for ≥16 hr
F – Renal failure	3x increase of baseline sCr OR sCr > 4mg/dl with an acute rise of 0.5 mg/dl	eCCl decrease of 50% OR urine output of <0.3 ml/kg/hr for ≥24 hr or anuria for ≥12 hr

	OR decrease in GFR of >75% from baseline OR urine output <0.3 ml/kg/hr for ≥24 hr or anuria for ≥12 hr	
L – Loss of Renal function	Renal failure with need for RRT (>4 wk)	
E – End stage renal disease	Non-recovery of renal function (RRT > 3 mo)	

Table 2: AKI Staging per the KDIGO Clinical Practice Guideline

	Serum Criteria	Urine Criteria
Stage 1	Increase in serum creatinine (sCr) by >0.3 mg/dL 1.5-1.9 times baseline.	Output <0.5 ml/kg/hr for 6-12 hours
Stage 2	Increase in sCr by 2-2.9 times baseline	Output <0.5 ml/kg/hr for >12 hours
Stage 3	In pediatric patients: a decrease in eGFR to 35 ml/min/1.73 m^2 sCr >4 mg/dL Started on renal replacement therapy Increase in sCr by 3 times baseline	Output <0.3 ml/kg/hr for >24 hours Anuria for >12 hours

ETIOLOGIES OF AKI:

The etiologies of AKI can be separated into three general categories: pre-renal, intrinsic renal, and post-renal causes. Pre-renal kidney injury is often due to a relative hypovolemic state or decreased kidney perfusion. This can occur in patients with dehydration, gastrointestinal losses, severe blood loss, distributive or hypovolemic shock, or some medications (i.e. NSAIDS, ACE-inhibitors). If caught early, this is often reversible with volume resuscitation or discontinuation of the offending agent. Intrinsic renal injury is due to glomerular or tubular injury and is not always reversible. This can occur in the setting of a prolonged pre-renal state, nephrotoxic medications, systemic disease, or primary kidney disease. Post-renal injury occurs with urinary obstruction either from bilateral ureteral obstruction from kidney stones or mass, severe unilateral kidney obstruction, bladder outlet obstruction, or neurogenic bladder.[6]

The etiology of kidney injury can be determined with a thorough history and physical examination along with supportive laboratory studies. Calculating the fractional excretion of sodium (FeNa) may be useful in patients with previously normal kidney function. A value less than 1% may indicate pre-renal etiology while values >2% supports intrinsic renal injury. If the patient has chronic kidney disease or prior diuretic use, calculating the fractional excretion of urea (FeUrea) is more helpful. A value <35% is indicative of pre-renal etiology.

MANAGEMENT OF AKI EMERGENCIES

As with any emergency medical condition, initial assessment begins with airway, breathing, and circulation (A, B, C's). Vital signs should be obtained, including blood pressure. Ill-appearing patients should be placed on continuous monitoring and IV access obtained. If AKI is suspected, evaluation should be focused on life threatening complications of AKI or renal failure (table 3). Preliminary laboratory studies that might be helpful include complete blood counts, electrolytes, BUN, Cr, urinalysis, and further urine studies (typically Na, urea, protein, and creatinine).

Table 3: Life threatening complications of acute kidney injury

Life threatening complications of AKI
Hypervolemia (fluid overload) Hypovolemia Hypertension Electrolyte derangements o Hyperkalemia o Acidosis o Uremia

FLUID MANAGEMENT

Hypervolemia is a severe complication of oliguric/anuric AKI. It can manifest with peripheral or facial edema, pulmonary edema, respiratory distress/failure, and heart failure. Fluid overload may be managed with diuretic medications, fluid restriction, or renal replacement therapy (RRT) in severe cases. Diuretics may improve fluid overload and reduce use of RRT, but has not shown to improve AKI outcomes.[4] Immediate

initiation of dialysis should be considered in consultation with a nephrologist if fluid overload is refractory to medications or the patient has anuria.

Patients with AKI due to pre-renal causes can be hypovolemic and fluid resuscitation may improve the kidney injury. Intravenous (IV) crystalloid or colloid fluids may be used, but adult studies have shown that there is no benefit of albumin or starch containing fluids over saline and some starch containing IV fluids may worsen AKI.[5,7] For these reasons, it is recommended to use normal saline (NS) fluids[5] keeping in mind that aggressive fluid administration may cause acute fluid overload in a patient with AKI or heart disease. One must monitor urine output and watch for symptoms of pulmonary edema or heart failure, especially if prescribing multiple boluses. One caveat regarding the preference for NS fluids is that some patients may benefit from 25% albumin infusion followed with IV diuretics, particularly if they have had significant saline volume resuscitation and have nephrotic syndrome with hypoalbuminemia. Vasopressors should be used if the patient is persistently hypovolemic despite adequate resuscitation.

URINARY OBSTRUCTION

Urinary obstruction causes post-renal AKI and is a condition that should be recognized and treated quickly. Obstructive AKI should be suspected in patients with voiding difficulties, bladder outlet obstruction, oliguria or anuria. Placement of a urinary catheter can relieve obstruction, improve AKI, and help quantify urine output. Physical exam may reveal a suprapubic or abdominal mass and bedside ultrasound may confirm a distended bladder. It is important to remember that obstructive uropathy leading to AKI can also occur above the level of the urethra and catheterization may not improve the obstruction. Further imaging such as ultrasound and consultation with nephrology or urology should be considered.

HYPERTENSION

Acute or severe hypertension can be life-threatening as it can cause end organ damage, particularly in the heart, brain, and kidney. The etiology of hypertension in acute kidney injury patients may be due to fluid overload or elevated activity of the renin-angiotensin pathway causing systemic vasoconstriction. Hypertensive urgency is generally defined as SBP >= 180 mmHg and/or DBP >= 110 or greater than 5 mmHg

above the 99th%tile for age in pediatric patients. Hypertensive emergency is defined as hypertensive urgency with associated clinical symptoms of end-organ dysfunction such as headache, confusion, vision changes, weakness, chest pain, shortness of breath, etc.

In hypertensive urgency and emergency, blood pressure should be lowered carefully over minutes to hours. The treatment goal is to reduce severity of hypertension and alleviate signs of end organ dysfunction, not to completely normalize blood pressure. Medications commonly used in the acute care or emergency setting include labetolol, nicardipine, hydralazine, esmolol, or nifedipine.[8] ACE inhibitors may exacerbate a pre-renal state so should be used with caution in the emergency setting.

ELECTROLYTE ABNORMALITIES

HYPERKALEMIA

Hyperkalemia or elevated serum potassium level is a life-threatening electrolyte abnormality that can progress to fatal cardiac arrhythmia. Symptomatic hyperkalemia may not be present unless levels are ≥ 7 mEq/L. Indications for immediate treatment of hyperkalemia include EKG findings of narrow, peaked T-waves, shortened QT interval followed by widened QRS, lengthening of PR interval, and low amplitude P waves.[6]

Strategies for treating hyperkalemia begin by stabilizing the cardiac cellular membranes with IV calcium therapy then reducing circulating potassium levels by shifting potassium into cells and thereafter decreasing total body potassium. IV calcium gluconate or calcium chloride may be used for calcium therapy, but both cause tissue necrosis and central access is preferred.

Shifting potassium into cells with medications that stimulate the Na-K-ATPase pump in skeletal muscle reduces circulating levels of K^+, but does not reduce the total body potassium. A temporary shift can be accomplished with albuterol inhalation, IV insulin (always given with glucose to avoid complications of hypoglycemia), or IV bicarbonate.

Removing total body potassium can be accomplished by two main modalities: urinary or fecal excretion. Loop diuretic therapy causes renal excretion of K+, but this is not useful in patients with non-functional kidneys, dialysis patients, or anuric paitents. Fecal excretion is often achieved with oral or rectal potassium binding

resins such as sodium polystyrene sulfonate- Kaexylate®. This is a slower route for removal and may require repeat dosing.

If hyperkalemia is refractory to medical management, dialysis should be considered in consultation with a nephrologist.

ACIDOSIS

The retention of hydrogen ions or loss of bicarbonate through renal wasting in AKI can lead to acidosis. Acidosis may also be present in AKI due to an underlying cause such as sepsis, shock, or toxic ingestion. Severe acidosis is a metabolic derangement that can lead to cardiopulmonary compromise if untreated. It is recommended to correct acidosis by treating the underlying cause and with intravenous bicarbonate if necessary. Rarely, emergent dialysis is necessary to correct acidosis alone.[5]

UREMIA

Since the kidney filters urea, elevated serum urea levels (elevated serum BUN level) can occur in the setting of AKI. Azotemia is defined as elevated serum BUN. Uremia is the clinical manifestation of azotemia in the setting of acute renal failure. Notable features of uremia are encephalopathy, nausea/vomiting, metabolic acidosis, and uremic platelet dysfunction. If untreated, it leads to severe encephalopathy, coma, and death. Uremia may improve with correction of kidney injury, but the presence of uremia is an indication for emergent dialysis.

DIALYSIS IN THE EMERGENCY DEPARTMENT

Practices vary by institution depending on access to nephrology specialists and dialysis related resources. Life-threatening indications for dialysis include severe hyperkalemia, severe acidosis, uremia, and pulmonary edema, but the decision to begin RRT remains subjective and consultation with a nephrologist is recommended.[5]

Generally, the emergent modes of dialysis include intermittent hemodialysis or continuous renal replacement therapy (CRRT). CRRT can employ clearance and volume control by diffusion (continuous veno-

venous hemodialysis; CVVHD) or by convection (continuous veno-venous hemofiltration; CVVH) or both diffusion and convection simultaneously (continuous veno-venous hemodiafiltration; CVVHDF). Peritoneal dialysis requires surgical peritoneal catheter placement and provides slow solute and fluid removal over hours, making this modality more appropriate for end stage renal disease management although it can be used for AKI. Intermittent hemodialysis has advantages over CRRT with shorter cycle time and more rapid removal of solute and fluid. However, hemodynamically unstable patients may not tolerate the higher flow rates typically used on intermittent hemodialysis and CRRT may be a better alternative in the critically ill state.

REFERENCES:

1. Van Bisen W, Vanholder R, Lamiere N. Defining Acute Renal Failure: RIFLE and Beyond. CJASN November 2006 vol. 1 no. 6 1314-1319.
2. Lameire N, Hoste E: Reflections on the definition, classification, and diagnostic evaluation of acute renal failure. Curr Opin Crit Care 2004; 10 :468– 475.
3. Akcan-Arikan A, Zappitelli M, Loftis LL, et al. Modified RIFLE criteria in critically ill children with acute kidney injury. Kidney Int. 2007;71:1028–1035.
4. Basu, RK and Wheeler, D. Approaches to the Management of Acute Kidney Injury in Children. Recent Pat on Biomark 2011;1(1):49-59.
5. KDIGO work group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney International Supplements (2012) 2, 1–138.
6. Rodig N, Fleischer G, Ludwig S.. “Acute Kidney Injury” and “Hyperkalemia”. Pediatric Emergency Medicine. 2010. pg 1113-1115.
7. Vincent JL, Sakr Y, Sprung CL, et al. Sepsis in European intensive care units: results of the SOAP study. Crit Care Med. 2006;34:344–353.
8. Constantine E, Fleischer G, Ludwig S. “Hypertension”. Pediatric Emergency Medicine. 2010. pg 315-323.