Developed by: ANNA Specialty Practice Networks

Acute Kidney Injury Fact Sheet

Clinical Issues

Overview

With over 30 definitions of acute kidney injury (AKI) in published studies, there is no consensus among experts regarding how AKI is defined (Palevsky et al., 2013). Palevsky and colleagues (2013) describe AKI as a sudden loss of kidney function occurring over hours to days, resulting in fluid and electrolyte imbalance, retention of nitrogenous waste products in the blood, and acid base irregularity. More specifically, AKI is defined as an increase in serum creatinine of 0.3 m/dL or higher within 48 hours, or an increase in serum creatinine 1.5 times baseline within the prior 7 days, or a decrease in urine volume less than 0.5 mL/kg/hr for 6 hours (Kidney Disease: Improving Global Outcomes [KDIGO], 2012).

In the United States, AKI has grown by 14% annually since 2001, with approximately 5% to 25% of all hospitalized patients diagnosed with AKI (Brown, Rezaee, Marshall, & Matheny, 2016). AKI is associated with high mortality rates. The number of AKI hospital-related deaths increased almost twofold, from 147,943 deaths in 2001 to 285,768 deaths in 2011 (Brown et al., 2016). Patients who develop AKI are at risk for adverse outcomes, such as cardiovascular events, pulmonary complications, and end stage renal disease (ESRD); however, it is important to note that approximately 66% of all AKI episodes develop in the community, outside of the hospital setting (Harty, 2014). Therefore, prevention, early recognition, and management are essential to decrease poor health outcomes associated with AKI.

Causes and Risk Factors

The etiologies of AKI are multifactorial and categorized as pre-renal, intra-renal, or post-renal (Lameire, Van Biesen, & Vanholder, 2005).

Pre-Renal

Reversible kidney hypoperfusion associated with:

- Hypotension.
- Volume depletion due to hemorrhage, diarrhea, dehydration, severe burns, or diuretics.
- Organ failure, such as pancreatitis and liver disease, which causes shift of fluid in the abdomen.
- Heart failure or heart attack.
- Severe allergic reactions.
- Overuse of medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs, such as ibuprofen, naproxen) and angiotension-converting enzyme (ACE) inhibitors.

Intra-Renal or Intrinsic

Direct injury to the kidney or renal parenchyma due to factors, such as:

- Cancer.
- Systemic Infection or sepsis.
- Interstitial nephritis or allergic reaction to medications.
- Scleroderma or other connective tissue disorder.
- Damage to the renal tubules, such as glomerulonephritis, vasculitis, or thrombotic microangiography.

Post-Renal

Obstruction of the urinary tract leading to increase pressure in the Bowman's capsule and reduced glomerular filtration rate (GFR) caused by:

- Enlarged prostate.
- Cancer of bladder, cervix, or prostate.
- Blood clot (thrombus).
- Kidney stones (renal calculi).
- Urethral strictures.

Risk Factors Associated with AKI

- Advanced age.
- Surgical procedures, especially cardiovascular surgery.
- Sepsis.
- Major trauma resulting in blood loss and muscle damage.
- Multiple organ dysfunction syndrome (MODS) seen in critically ill patients.
- Exposure to radiocontrast agents.
- Nephrotoxic drugs (NSAIDs, methotrexate, ACE inhibitors).
- Dehydration.

Signs and Symptoms

The clinical presentation of patients with AKI vary depending on the severity and cause of AKI (Rahman, Shad, & Smith, 2012). Many patients with mild or moderate AKI are asymptomatic (Rahman et al., 2012). According to the National Kidney Foundation (2019), patients with more severe AKI may have the following symptoms:

- Jugular venous distention.

- Edema (peripheral, periorbital).
- Dyspnea.
- Nausea and vomiting.
- Fatigue.
- Confusion.
- Chest pain or pressure.
- Back pain.
- Decreased urine output (oliguria).
- Seizure or coma in severe cases.

There are four phases of AKI. Depending on the phase of AKI, other clinical manifestations of AKI often present in critically ill patients, including metabolic acidosis, hyperkalemia, hypophosphatemia, uremia, and azotemia. Although the severity of symptoms and the complexity of renal dysfunction vary, the clinical course and manifestations of AKI generally progress over the following four phases (Dirkes, 2015; Lewis, Dirksen, Heikemper, Bucher, & Harding, 2014):

- **Onset Phase:** Initial insult to kidney due to factors such as blood loss, dehydration, burns, diabetes insipidos, or infection, resulting in a decrease in renal blood flow and tissue oxygenation 25% of normal and urine output less than 0.5 mL/kg/hr. This phase last hours to days.
- **Oliguric Phase:** The most common initial clinical manifestation of AKI is oliguria, defined as a reduction in urine output less than 400 mL/day. Oliguria is manifested within 1 to 7 days of kidney injury. This phase typically lasts 10 to 14 days but can last months in some cases. The longer the oliguric phase, the poorer the prognosis for the recovery of kidney function. However, approximately 50% of patients will not experience oliguria, making diagnosis difficult. In the oliguric phase, signs of fluid volume overload, such as edema, distended neck veins, hypertension, pulmonary edema, and heart failure, may occur. In addition to signs of volume overload, metabolic acidosis, hyperkalemia, hyperphosphatemia, and uremic symptoms may also be present.
- **Diuretic Phase:** In this phase, daily urine output is approximately 1 to 3 liters but can reach as high as 5 liters or more. The kidneys recover their ability to excrete waste but cannot concentrate the urine. Hypovolemia and hypotension may occur due to massive volume loss. Due to the loss of large volumes of fluid and electrolytes, patients should be monitored for hyponatremia, hypokalemia, and dehydration. This phase may last 1 to 3 weeks. During the end of this phase, fluid and electrolytes, acid-base balance, and waste product values (blood urea nitrogen [BUN] and creatinine) start to normalize.
- **Recovery Phase:** This phase begins when the GFR increases, allowing plateau of the BUN and creatinine, then a gradual decline. Although significant improvement in kidney function is observed in the first 1 to 2 weeks of this phase, it may take up to 12 months for kidney function to stabilize. Some patients do not recover and progress to ESRD. The outcome of AKI is contingent upon the patient's overall health, the severity of kidney damage, and the presence of complications and comorbid conditions. With aggressive treatment, most patients achieve normal kidney function without complications.

Diagnosis and Evaluation

The diagnosis of AKI is multifactorial, with an emphasis on identifying the underlying cause. History and physical examination are important components in the diagnosis of AKI, including assessment of volume status (Rhaman et al., 2012). When conducting the physical examination, assessment of intravascular volume status and skin rash, which indicates systemic illness, is essential (Rahman et al., 2012). Initial laboratory studies to assist in identifying the cause of AKI include complete blood count, urinalysis, and kidney function studies, such as measurement of serum creatinine, BUN, and fractional excretion of sodium (Rahman et al., 2012). According to Workeneh, Agraharkar, and Gupta (2017), the following laboratory and diagnostic studies are often performed to diagnose and determine the cause of AKI:

- **BUN and Creatinine:** Elevation of creatinine and BUN are hallmark indicators of kidney injury. The BUN-to-creatinine ratio can exceed 20:1 in conditions that promote reabsorption of urea, such as large volume loss, which suggests pre-renal AKI.
- **Complete Blood Count:** Used to detect infection, chronic anemia, blood loss, or thrombotic microangiopathy.
- **Urinalysis:** Muddy, brown, granular casts; oxalate crystals; and tubular casts in urine sediment may indicate acute tubular necrosis (ATN). Urine that is reddish-brown in color indicates myoglobinuria or hemoglobinuria. Further, renal tubular injury will result in proteinuria. Urinary red blood cells (RBCs) occur due to bleeding along the collecting system. RBC casts or dysmorphic RBCs in the urine are the result of glomerular inflammation or glomerulonephritis. The presence of white blood cells (WBCs) or WBC casts suggests pyelonephritis or interstitial nephritis.
- **Peripheral Smear:** The presence of schistocytes suggests hemolytic uremic syndrome and thrombotic thrombocytopenic purpura.

Table 1.
Staging of AKI

Stage	Serum Creatinine	Urine Output
1	1.5 to 1.9 times baseline OR ≥ 0.3 mg/dL (≥ 26.5 mmol/l) increase	< 0.5 mL/kg/h for 6 to 12 hours
2	2.0 to 2.9 times baseline	< 0.5 mL/kg/h for ≥ 12 hours
3	3.0 times baseline OR Increase in serum creatinine to ≥ 4.0 mg/dL (≥ 353.6 mmol/L) OR Initiation of renal replacement therapy OR In patients <18 years, decrease in eGFR to < 35 mL/min/1.73 m^2	< 0.3 mL/kg/h for X 24 hours OR Anuria for X 12 hours

Source: KDIGO, 2012.

- **Fraction of Urinary Sodium (FENa**): Measures the percent of sodium excreted in the urine and is used to determine pre-renal vs. ATN as the cause of AKI. This test is useful only in the presence of oliguria. In pre-renal conditions, FENa is usually less than 1%. In ATN, the FENa is typically greater than 1%.
- **Renal Ultrasound:** Used to evaluate potential obstruction of the renal collection system and existing renal disease. Hydronephrosis may be present, and small kidneys indicate chronic kidney disease (CKD). Doppler ultrasound assists in the diagnosis of renovascular or thromboembolic disease. Elevated restrictive indices may suggest hepatorenal syndrome.
- **Nuclear Scanning:** Allows assessment of renal tubular function and blood flow.
- **Aortorenal Angiography:** Helpful in diagnosing renal vascular disorders, such as atherosclerosis with aortorenal occlusion, certain cases of necrotizing vasculitis, renal artery stenosis, and renal atheroembolic disease.
- **Renal Biopsy:** Useful in determining intra-renal causes of AKI. Also indicated if renal function has not returned to baseline for a prolonged period and prognosis is needed for long-term management.

In addition to routine diagnostic studies, biomarkers are emerging as significant tools for the identification of AKI. One example is cyastatin C. In a study conducted by Bongiovanni and colleagues (2015) of 198 emergency department patients, serum cyastatin C alone or in combination with serum creatinine and GFR was a strong predictor of AKI risk. One advantage of cyastatin C was that it identified AKI when serum creatinine levels remained within the normal range (Bongiovanni et al., 2015).

KDIGO (2012) makes the following recommendations for patients at risk for AKI:

- Patients should be tested with measurements of serum creatinine and urine output to detect AKI.
- The frequency and duration of monitoring is individualized according to patient risk and clinical course.
- Evaluate patients with AKI promptly to determine the cause, with special attention to reversible causes.
- Monitor patients with AKI with measurements of serum creatinine (SCr) and urine output to stage the severity.
- Manage patients with AKI according to the stage and cause.

According to KDIGO (2012), AKI is staged by criteria displayed in Table 1.

Management and Treatment

Optimal management of AKI requires close collaboration among the interprofessional team. Most patients with AKI will be hospitalized, unless the condition is mild with a clearly reversible cause (Rahman et al., 2012). The goal of management is to ensure adequate renal perfusion by maintaining and achieving hemodynamic stability, avoiding hypovolemia, and preventing further kidney damage (Rahman et al., 2012). KDIGO (2012) clinical practice guidelines suggest the management of AKI should be based on the stage and cause of AKI, and include the following recommendations:

- In patients at risk for or with AKI who are not in hemor-

rhagic shock, use isotonic crystalloids (0.9% normal saline) rather than colloids (albumin or hetastarch) in the initial management of intravascular volume expansion.
- In patients with vasomotor shock at risk for or with AKI, use vasopressors along with fluids.
- In high-risk patients with septic shock or in the perioperative setting, use protocol-based management of hemodynamic and oxygenation parameters to prevent development or worsening of AKI.
- In patients who are critically ill, initiate insulin therapy targeting plasma glucose 110 to 149 mg/dL (6.1 to 8.3 mmol/L).
- Do not use diuretics to prevent or treat AKI, except in the management of volume overload.
- Achieve a total energy intake of 20 to 30 kcal/kg/d in patients with any stage of AKI.
- Low dose dopamine should not be used to prevent or treat AKI.
- Prior to administration of contrast medium, assess the risk of contrast-induced AKI (CI-AKI) by screening for pre-existing kidney impairment.
- In patients at risk for CI-AKI, intravenous volume (IV) expanders are recommended with either isotonic sodium chloride or sodium bicarbonate solutions.
- Consider oral acetylcysteine along with IV isotonic crystalloids in patients at increased risk for CI-AKI.
- Renal replacement therapy (RRT) should be initiated emergently when life-threatening changes in fluid, electrolyte, and acid-base imbalance exists.
- Do not rely solely on BUN and creatinine thresholds when making the decision to start RRT; rather, consider the broader clinical context, presence of conditions that can be modified with RRT, and trends of laboratory tests.
- When initiating RRT, use an uncuffed, non-tunneled hemodialysis catheter rather than a tunneled dialysis catheter in patients with AKI.
- Obtain a chest X-ray promptly after placement and before the first use of a subclavian or internal jugular vein hemodialysis catheter.
- For patients who are hemodynamically unstable, use continuous renal replacement therapy (CRRT) rather than standard RRT.
- Use bicarbonate, rather than lactate, as a dialysate and replacement fluid buffer for RRT in patients with AKI.
- Deliver a Kt/V of 3.9 per week when using intermittent or extended RRT for AKI.
- Nursing care and management (Thornburg & Gray-Vickrey, 2016). When administering fluid bolus(es), monitor cardiovascular response to the increased intravascular volume by assessing for an increase in blood pressure (BP) and central venous pressure (CVP).
- Strict measurement and documentation of intake and output (I & O) every shift.
- Implement infection control measures. Use aseptic technique and protect the patient from others with infectious diseases.
- Review medications for nephrotoxins.
- Monitor volume status including I & O, BP, heart rate, body weight, jugular venous distention, and peripheral and pulmonary edema.
- Weigh patients with the same scale at the same time each day.
- Perform skin care and take measures to prevent pressure ulcers.
- To prevent stomatitis that develops when ammonia in saliva irritates the mucous membranes, perform mouth care daily.
- Review laboratory results daily for electrolyte and acid base imbalance.
- Monitor continuous electrocardiogram (ECG) to detect cardiac arrhythmias.
- Assess the heart for an S3 gallop, murmurs, or a pericardial friction rub.
- Auscultate the lungs for crackles, rhonchi, or diminished breath sounds.
- Observe dialysis access site for inflammation and exudate.

Prevention

The National Institute of Healthcare Excellence (NICE) (2013) developed the following guidelines for the prevention of AKI in patients at risk:
- Implement tracking and/or trigger systems (early warning scores) to identify adults at risk of AKI due to clinical deterioration or at risk for clinical deterioration.
- In patients with risk factors for AKI scheduled to receive iodinated contrast media, offer IV volume expansion (0.9% normal saline or sodium bicarbonate) prior to the procedure.
- Consider temporarily stopping ACE inhibitors or angiotensin receptor blockers (ARBs) in adults with CKD and an estimated GFR (eGFR) less than 40 mL/min/1.73m^2 prior to receiving iodinated contrast medium and adults experiencing vomiting, diarrhea, or sepsis until their symptoms improve or stabilize.
- Consult pharmacist regarding optimizing medications and drug dosing in patients at risk for AKI.

Other strategies to prevent AKI, according to Rahman and colleagues (2012), include:

- Avoid nephrotoxic medications if possible.
- Measure and follow drug levels, if available, and use appropriate dosing, intervals, and duration of therapy.
- Optimize fluid resuscitation in patients hemodynamically unstable to achieve a mean arterial pressure (MAP) of greater than 65 using isotonic solutions (e.g., 0.9% normal saline).
- For persistent hypotension not responsive to fluid resuscitation, start vasopressors with the goal of a mean arterial pressure (MAP) of 65 or greater.
- Maintain adequate hydration.
- For patients having surgery, adequate volume and prevention of hypotension is important. Optimize cardiac function and consider holding renin-angiotensin system antagonists preoperatively.

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ANNA improves members' lives through education, advocacy, networking, and science.

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