Acute Kidney Injury Fact Sheet

Overview

Acute kidney injury (AKI) is a major global health problem affecting more than 13.3 million people worldwide each year (Abebe et al., 2021). Dasta and Kane-Gill (2019) stated that in the United States, AKI is associated with an increase in hospital costs that range from $5.4 to $24.0 billion annually. The death rate from AKI can range anywhere from 10%-80% depending on factors such as patient population, the severity of the kidney injury, and the level and availability of medical care. Chronic morbidities associated with AKI include continued loss of kidney function, as can be seen in chronic kidney disease, hypertension, cardiovascular disease, heart failure, and cerebrovascular events (Odum & Lynch, 2022). Since AKI is associated with significant increases in morbidity and mortality, the goal is to prevent the condition by employing a multidisciplinary team approach. All health care workers must be aware of the condition and its causes.

This Fact Sheet will provide an overview of AKI, including defining AKI, common causes of AKI with associated symptomology, diagnosis and evaluation, new nomenclature, and describing the different types of dialysis and associated nursing management. It includes a new section on patient education, which is crucial for patients post-AKI recovery (Ostermann et al., 2020).

Definition of AKI

AKI is a condition characterized by a sudden loss of kidney function developing over the course of 7 days or less or a sudden decrease in kidney function (Kidney Disease: Improving Global Outcomes [KDIGO] 2012; 2023). According to KDIGO AKI clinical guideline (KDIGO, 2012; 2023), AKI is defined by:

- An increase in serum creatinine by .3 mg/dL or more within 48 hours, or
- An increase in serum creatinine to 1.5 or more times of the patient's baseline, which is known or presumed to have occurred within the prior 7 days, or
- Urine volume less than 0.5 mL/kg/hour for 6 hours

AKI diagnosis and treatment are complex, resulting in a variety of perspectives and practice variations for AKI. The stages of AKI are further defined by KDIGO (2012) in Table 1:

Table 1.
Stages of Acute Kidney Injury

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 kidney replacement therapy OR In patients <18 years, decrease in eGFR to < 35 mL/min/1.73m^2	< 0.3 mL/kg/h for X 24 hours OR Anuria for X 12 hours

Source: KDIGO, 2012.
Note: eGFR = estimated glomerular filtration rate.

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Revised AKI Nomenclature

AKI was previously referred to as acute renal failure (ARF) or acute kidney failure (AKF). The word *kidney* replaced *renal* because it is more recognizable by patients. The word *injury* replaced *failure* because it highlights the complexity of the varying degrees of kidney impairment rather than just pointing out that the kidneys are failing (KDIGO, 2020). This is significant because it has been well-documented that even a modest reduction in kidney function is associated with adverse outcomes, especially among adults younger than 40 years (Hussein et al., 2023).

Kidney Replacement Therapy (KRT) Options

KRT is commonly required in patients with severe AKI. Acute (hospital-based) KRT options include intermittent hemodialysis (IHD), peritoneal dialysis (PD), continuous kidney replacement therapy (CKRT), and hybrid therapies, such as prolonged intermittent kidney replacement therapy (PIKRT) or sustained low-efficiency dialysis (SLED), which provide prolonged but still intermittent dialysis (Luehr et al., 2020).

Intermittent Hemodialysis (IHD)

- IHD is performed with pump-driven dialysis machines using extracorporeal circuits and dialyzers to act as renal support for the patient with severe AKI.
- IHD requires a vascular access. A central venous catheter (CVC) is most often used in the emergent initiation of dialysis.
- IHD is highly effective in restoring electrolytes, correcting acid-base abnormalities, solute clearance, and fluid removal by ultrafiltration over a short period of time, typically 3-5 hours, 3 times a week or more frequently if medically indicated, such as for fluid overload.

Peritoneal Dialysis (PD)

- PD uses the peritoneum of the abdominal cavity as a natural filter to remove solutes and fluid.
- The sterile dialysis solution is infused and drained through an abdominal catheter after it has dwelled for a period (usually 1.5 hours or more) within the peritoneal cavity, which is called an exchange. Each exchange (connect/drain/flush/fill/disconnect) takes approximately 30 to 40 minutes.
- Options include manual exchanges called continuous ambulatory peritoneal dialysis (CAPD) or the use of a machine (called a cycler) for automated peritoneal dialysis (APD) sessions. PD is performed daily using CAPD, APD, or a combination of both.

Prolonged Intermittent Kidney Replacement (PIKRT)/Sustained Low Efficiency Dialysis (SLED)

- PIKRT/SLED is a gentler IHD treatment for an extended time (6 to 18 hours)
- PIRKT is for patients who cannot tolerate the intensity of a 3 to 5-hour IHD treatment or is used when CKRT is unavailable at the hospital.

Continuous Kidney Replacement Therapy (CKRT)

- CKRT runs 24 hours nonstop in intensive care units for severely ill patients requiring hemodialysis treatment. It is the continuous, slow removal of solutes and fluid for patients who are hemodynamically unstable.
- CKRT is indicated in patients who meet criteria for hemodialysis therapy but cannot tolerate conventional IHD due to hemodynamic instability.
- CKRT replacement fluids that contribute to the correction of the acid-base and electrolyte balance, which is often disturbed in critically ill patients needing CKRT.

Causes of AKI

The kidneys filter the blood, process the filtrate, and then excrete it through the ureters, bladder, and urethra. The three categories of AKI all relate to a disruption in this process. They include pre-kidney, which is caused by a reduction in blood flow to the kidneys; intrinsic (intra-kidney), caused by a process going on inside the kidneys; and post-kidney, caused by an obstruction in the flow of urine away from the kidneys.

Pre-Kidney Injury

According to Cotton and Inglese (2020), and Mercado and colleagues (2019), the four major causes of pre-kidney AKI are:

- Decreased cardiac output.
- Decreased intravascular volume.
- Vasodilation.
- Obstruction of kidney blood vessels.

Generally, pre-kidney AKI can be quickly reversed if the underlying cause is corrected and blood flow is restored. However, irreversible injury can occur if the hypoperfusion is sustained for a prolonged period.

Intrinsic or Intra-Kidney Injury

Intrinsic or Intra-kidney AKI is caused by damage to renal parenchyma (functional tissue of the kidney consisting of nephrons) and is categorized by the location of the injury. Intrinsic AKI can account for 50% of AKI incidences.

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According to Mercado and colleagues (2019), the three most common causes of intrinsic AKI are:

- **Acute tubular necrosis (ATN)** is an injury to the tubules
  - Common causes are hypoperfusion, sepsis, ischemia/reperfusion injury, and nephrotoxins. It is associated with higher morbidity and mortality rates.
- **Acute glomerulonephritis (AGN)** is an injury to the proliferation of glomerular tissue in the basement membrane of the kidney.
  - Common causes are non-infections, such as abnormal immune responses (for example, lupus erythematosus, granulomatosis with polyangiitis, or Goodpasture syndrome). Infectious causes include bacterial infections (streptococcal) or viral infections (HIV, hepatitis). AGN represents 10% to 15% of glomerular diseases
- **Acute interstitial nephritis (AIN)** occurs when the spaces between the kidney tubules become edematous and inflamed.
  - The most common cause is to drug reaction, which accounts for 70% to 75% of AIN cases and is more severe in older adults. The most frequent drug categories are antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs), but also include analgesics, diuretics, and proton pump inhibitors.
  - Non-allergic causes include autoimmune disorders, infections (streptococcal), and high calcium or uric acid levels.

Post-Kidney Injury

Post-kidney injury AKI is caused by an obstruction that affects the outflow of urine from the kidneys. The most common cause stems from prostate issues (prostatic hypertrophy, benign or cancerous) in the elderly. Other causes include:

- Bladder or cervical cancer.
- Kidney stones.
- Children: the most common is congenital abnormalities.
- Neurogenic bladder.
- Medications: crystal formation from methotrexate or acyclovir.
- Overdistended bladder.
- Pregnancy: stemming from hydronephrosis due to uterine compression.
- Injury to ureters or bladder during surgery.
- Kidney vein thrombosis.
- Retroperitoneal fibrosis.
- Trauma.
- Uric acid crystals.

Risk Factors Associated with AKI

- Very old or young.
- Post-surgical procedures, particularly cardiovascular surgery.
- Sepsis (especially COVID-19).
- Multiple system organ failure (MSOF), such as from septic shock, rhabdomyolysis from trauma with crush injuries, or drug-induced muscle injury.
- Exposure to radiographic contrast.
- Those receiving nephrotoxic medications (NSAIDs, methotrexate, angiotensin-converting enzyme (ACE) inhibitors, or aminoglycosides).
- Decreased intravascular volume.
- Mechanical ventilation.
- Traditional herbal medication with aristolochic acid content.
- Previous AKI episode.
- Chronic kidney disease (CKD).

Signs and Symptoms of AKI

Patients may or may not be symptomatic with AKI. According to the National Kidney Foundation (NKF, 2019), symptomatic patients may have the following symptoms:

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

Diagnosis and Evaluation

History and physical examination are essential components in diagnosing AKI, including assessing volume status (Patil & Salunke, 2020). Noting the time of onset of AKI can be helpful when dealing with hospitalized patients. If a sudden increase in BUN and creatinine is observed, the inciting factor usually occurs 24 to 48 hours before onset (Goyal et.al, 2023). It is also important to review medications the patient receives and determine if the doses need to be modified based on decreased creatinine clearance. ACE inhibitors and angiotensin II receptor blockers (ARBs) are often the co-contributors to AKI. While ACE inhibitors and ARBs are not likely to cause AKI when combined with other factors, the risk of developing AKI goes up. All patients presenting with AKI warrant a comprehensive metabolic panel. 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 urea reabsorption, such as significant volume loss, suggesting pre-kidney 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 result from glomerular inflammation or glomerulonephritis. The presence of white blood cells (WBCs) or WBC casts suggests pyelonephritis or interstitial nephritis. Older adult patients may warrant serum and urine protein electrophoresis (SPEP and UPEP) to rule out monoclonal gammopathy and multiple myeloma.
- **Urine electrolytes:** Urine studies should be checked for electrolytes, protein, osmolality, and albumin-to-creatinine ratios.
- **Peripheral blood smear:** The presence of schistocytes suggests hemolytic uremic syndrome and thrombotic thrombocytopenic purpura (TTP).
- **Renal ultrasound:** Used to evaluate potential post-kidney obstruction of the renal collection system and existing kidney disease. Hydronephrosis may be present, and small kidneys indicate CKD. Doppler ultrasound assists in the diagnosis of renovascular or thromboembolic disease. Elevated resistive indices may suggest hepatorenal syndrome.
- **CT Scan:** Perform CT scan without contrast to evaluate for nephrolithiasis or urolithiasis,
- **Renal angiography:** Helpful in diagnosing renal vascular disorders, such as atherosclerosis with aortorenal occlusion, certain cases of necrotizing vasculitis, renal artery stenosis, and renal athero-embolic disease. Due to the need for contrast injection and the potential for further kidney injury after weighing risk vs. benefit, it may or may not be indicated for patients with AKI.
- **Renal biopsy:** Useful in determining intra-kidneyl causes of AKI. It also indicates if kidney function has not returned to baseline for a prolonged period and the diagnosis is needed for long-term management. It is the gold standard for the diagnosis of intrinsic kidney disease.

In addition to routine diagnostic studies, biomarkers are emerging as significant tools for identifying AKI. New biomarkers, such as serum cystatin C, for the prediction and early detection of AKI are being extensively researched. While new biomarkers are being validated, functional biomarkers (serum creatinine, serum urea, and urine output) continue to be used. In the future, existing or even newer biomarkers will likely be combined with the use of functional markers to define and diagnose AKI.

KDIGO Recommendations

KDIGO (2012; 2023) makes the following recommendations for patients at risk for AKI:

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

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. The goal of management is to ensure adequate kidney perfusion by maintaining and achieving hemodynamic stability, avoiding hypovolemia, and preventing further kidney damage (Patil & Salunke, 2020). KDIGO (2012; 2023) 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 hemorrhagic 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 the perioperative setting, use protocol-based management of hemodynamic and oxygenation parameters to prevent the 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 managing 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.

- Before 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 (IV) volume 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.
- Kidney replacement therapy (KRT) should be initiated immediately when life-threatening changes in fluid, electrolyte, and acid-base imbalance exist.
- Do not rely solely on BUN and creatinine thresholds when deciding to start KRT; instead, consider the broader clinical context, the presence of conditions that can be modified with KRT, and trends of laboratory tests.
- When initiating KRT, 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 an internal jugular or subclavian vein hemodialysis catheter.
- For patients who are hemodynamically unstable, use CKRT rather than standard IHD.
- Use bicarbonate, rather than lactate, as a dialysate and replacement fluid buffer for KRT in patients with AKI.
- 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).
- Every nursing shift requires strict measurement and documentation of intake and output (I&O).
- Implement infection control measures. Use aseptic techniques 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 to determine fluid retention.
- 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 the dialysis access site for inflammation and exudate.

Education and Communication

Shared decision-making and communication among caregivers, patients, and family members are crucial for patients post-AKI recovery (Ostermann et al., 2020). Nurses caring for patients with AKI are critical to these overall management strategies. Whether in the hospital or clinic setting, nurses begin vital patient education that should include risks of certain medications; fluid and electrolyte management; infection risks; the potential for cardiovascular, gastrointestinal, and neurologic complications; AKI risks from iodine-containing contrast media; and the need to manage hypertension and diabetes (Davenport et al., 2020; Goyal et al., 2023). This information can help the patient manage their kidney disease, recognize when to seek additional medical care, and ask more questions of their health care provider(s).

Post-Discharge Follow Up

To mitigate the risk of further kidney injury and to facilitate recovery, it is now recommended that patients who have experienced AKI receive post-discharge follow up. Recommendations include:

- Patient education, including the awareness that they experienced AKI (they had a problem with their kidneys) along with the potential future risk of CKD, to encourage the promotion of healthy behaviors.
- BP monitoring and control.
- Monitoring proteinuria, serum creatinine, glomerular filtration rate (GFR).
- Formal post-discharge nephrology appointment within:
  - 90 days for those with residual CKD Stage 4 at discharge.
  - 30 days for those with residual CKD Stage 5 not on KRT at hospital discharge.
  - 30 days for those with ongoing dialysis requirements at the time of hospital discharge.

Discharge Summaries

Discharge summaries should include the following:

- Record of AKI detected, etiology, maximum stage, discharge stage, and if the need for kidney support is temporary or ongoing.
- Specific recommendations for monitoring kidney function
- Advice on drug therapy that may have been implicated in the episode (such as AIN caused by NSAIDs)
- Education is given to patients, relatives, or caregivers.

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Communication Between Health Settings

Nurses in the hospital setting can facilitate continuity of care as the patient moves back into the community by:

- Creating or using available standardized patient education materials.
- Creating a standardized summary/handoff form.
- Encouraging the patient to take the handoff form to their follow-up appointments.
- Encouraging patients to attend any discharge education classes.

Prevention

Nurses in the community setting play a key role in helping reduce the mortality and morbidity of AKI. Community-acquired AKI is associated with a six-fold risk of in-hospital dialysis initiation, and a two-fold risk of ICU admission and in-hospital mortality (Ehmann et al., 2024). In addition, 70% of community-acquired AKI was due to pre-kidney causes (Kaufman et al., 2023). Recently, researchers found that 49.3% of those with COVID-19 also experienced AKI (Pelayo et al., 2020). Of those, 72% had AKI indicators upon admission (community-acquired AKI), with 28 % developing AKI during hospitalization (Pelayo et al., 2020). To improve patients' well-being and quality of life, nurses can address gaps in patient knowledge of AKI, specifically regarding preventing the reoccurrence of AKI.

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ANNA Mission Statement

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

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