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Acute kidney injury

Limiting the damage

By Becky Thornburg, MSN, RN, and Peg Gray-Vickrey, DNS, RN

MR. R, 74, IS ADMITTED to the ED following a fall at his home. He'd been found by a family member lying on the floor of his garage, where he'd been for about 8 hours. X-rays reveal a right hip fracture. Lab results are largely within normal limits except for an elevated blood urea nitrogen (BUN) of 31 mg/dL (normal in older adults >60 years, 8-23 mg/dL), creatinine of 1.1 mg/dL (normal in adult men, 0.9-1.3 mg/dL), and a BUN:creatinine ratio of 28 (normal, 6 to 22).[1]

In the ED, Mr. R's health history includes systemic hypertension and osteoarthritis of both knees. He has no known cardiac or renal disease and has never had any surgical procedures. His prescribed medications include only lisinopril, an angiotensin-converting enzyme (ACE) inhibitor, to treat his hypertension. He states that he's also been taking ibuprofen two or three times a day for knee pain.

Mr. R's history and initial lab values suggest that he's at risk for acute kidney injury (AKI). Because kidney function usually returns to baseline if AKI is identified early and appropriately treated, all nurses need to be alert for risk factors for AKI, able to recognize the early signs and symptoms, and prepared to implement appropriate nursing interventions and administer treatment prescribed by the healthcare provider. This article discusses the nurse's role in assessing and

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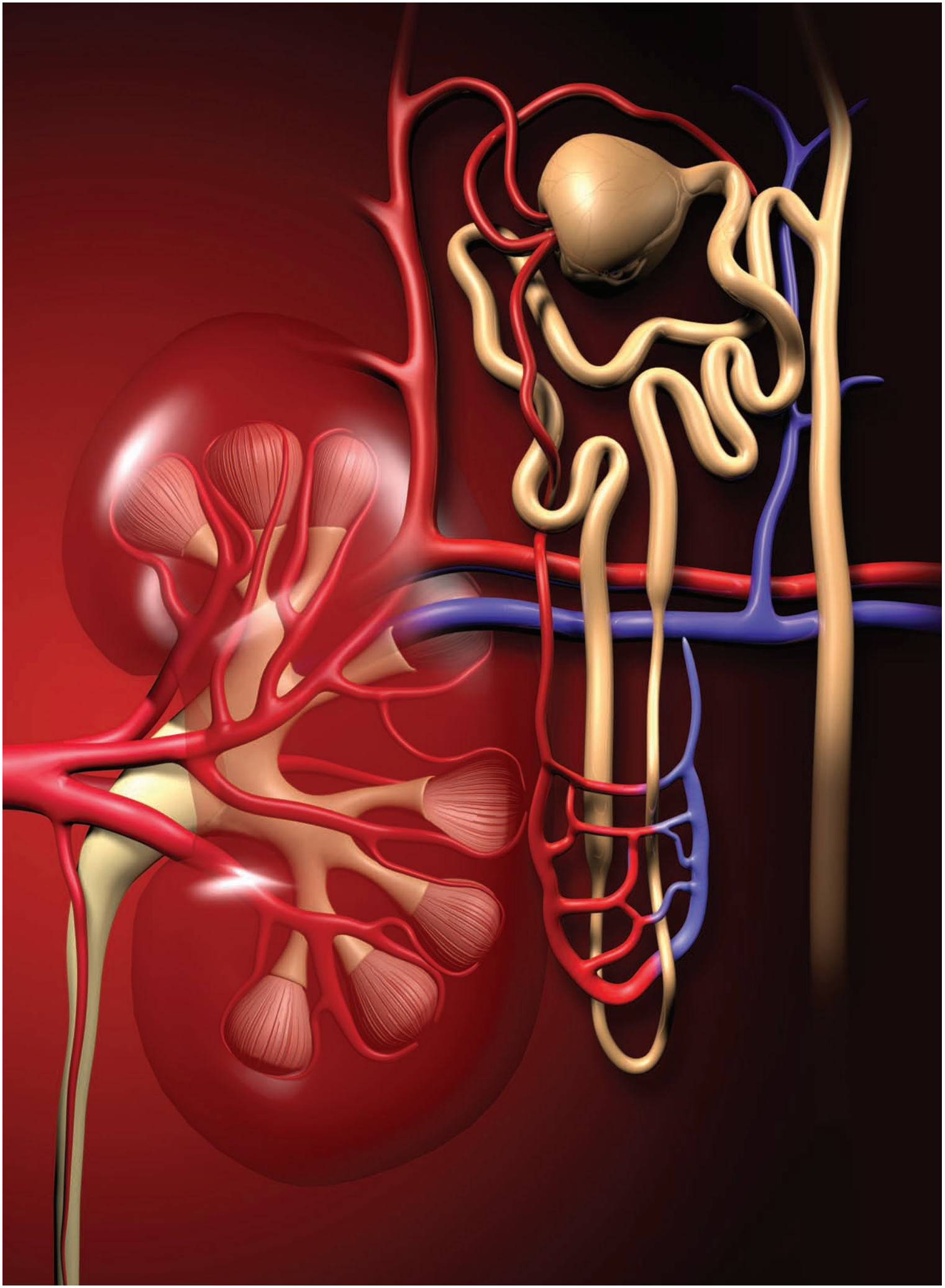


treating this complex disorder in older adults.

Raising suspicions

Mr. R is admitted to the medical/surgical unit after undergoing right total hip arthroplasty (THA) with regional anesthesia. Upon initial assessment, his nurse notes that he's alert and oriented with stable vital signs. His pain level was reported as 1 on a numeric pain scale of 1-10. The rest of his physical assessment is within normal limits with the exception of an S3, bibasilar pulmonary crackles, and ecchymoses involving the right side of his body. He's receiving an infusion of 0.9% sodium chloride solution at 100 mL/hour. His urine output had been approximately equal to intake for the past 8 hours per report from the previous shift.

Examining the 24-hour intake and output record closely, the nurse notes that although the urine output total was reported correctly, the hourly output documented by unlicensed assistive personnel has been dropping for the past 4 hours. The nurse notes that labs have been ordered for post-op day 2.

Troubled by these findings, the nurse immediately contacts the surgeon, who orders additional diagnostic studies. Results reveal that Mr. R has AKI.

What's AKI?

The kidneys are highly complex organs with numerous functions and regulatory processes. (See *An inside look at the kidney* and *How the kidneys work*.) By definition, AKI is an abrupt decrease in kidney function meeting any of these criteria:[2,3]

- an increase in serum creatinine (SCr) by 0.3 mg/dL or more within 48 hours
- an increase in SCr to 1.5 times or more the baseline value within the prior 7 days
- a decrease in urine output to less than 0.5 mL/kg/hour for 6 hours.

The term AKI replaces the earlier term acute renal failure (ARF) to better reflect the spectrum of injury ranging from minor changes in renal function markers to the need for renal replacement therapy (RRT) that may arise from multiple factors.[4,5] The term ARF is still used when kidney injury results in the need for RRT, including traditional intermittent hemodialysis and newer continuous renal replacement therapies (CCRT) such as slow continuous ultrafiltration, continuous venovenous hemofiltration, continuous venovenous hemodialysis, continuous venovenous hemodiafiltration, and continuous arteriovenous hemofiltration.[6]

Risk factors

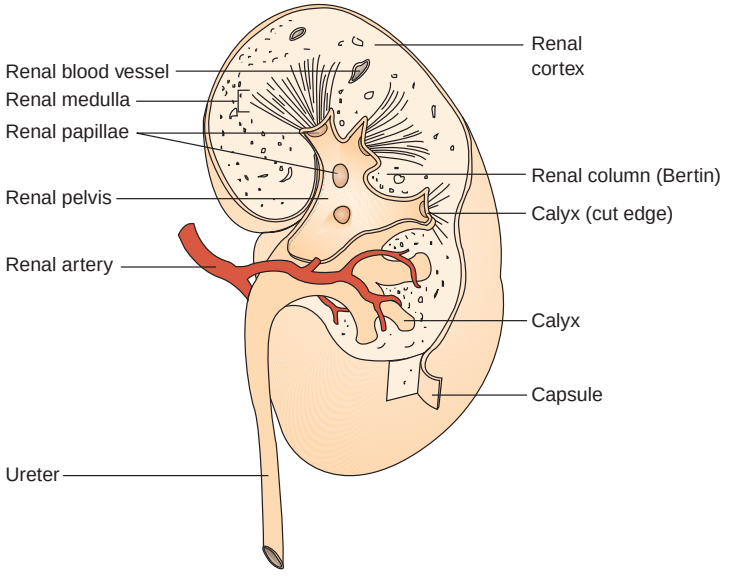
AKI is seen in 15% of all hospitalized patients and up to 66% of patients in the ICU.[7-11] Over the past decade, the incidence of AKI has increased due to higher patient acuity, the growing population of older adults, and improved identification of AKI.[12] The greatest risk factors for AKI include:[10,12-14]

- age 75 or older
- diabetes
- hypertension
- preexisting chronic kidney disease (CKD)
- heart or liver failure
- sepsis
- use of intravascular radiocontrast agents
- cardiac surgery after use of a radiocontrast agent
- polypharmacy.

Advancing age is an important factor predisposing a patient to AKI. As a person ages, the kidney undergoes structural and functional changes; for example, overall cortical mass is reduced.[12] In addition, renal blood flow decreases by 1% per year after age 30.[15] For these and other reasons, renal function may decline by 50% or more by the time a patient reaches age 70.[16]

Functional changes in the renal system include decreased ability to excrete a sodium load, decreased ability to conserve water when the patient is dehydrated, and decreased glomerular filtration rate (GFR).[16,17] GFR decreases 10% per decade after age 30.[12,18] Combined with other pathophysiologic challenges, these renal changes associated with aging can make the development of AKI more likely.[15]

An inside look at the kidney



How the kidneys work[33,34]

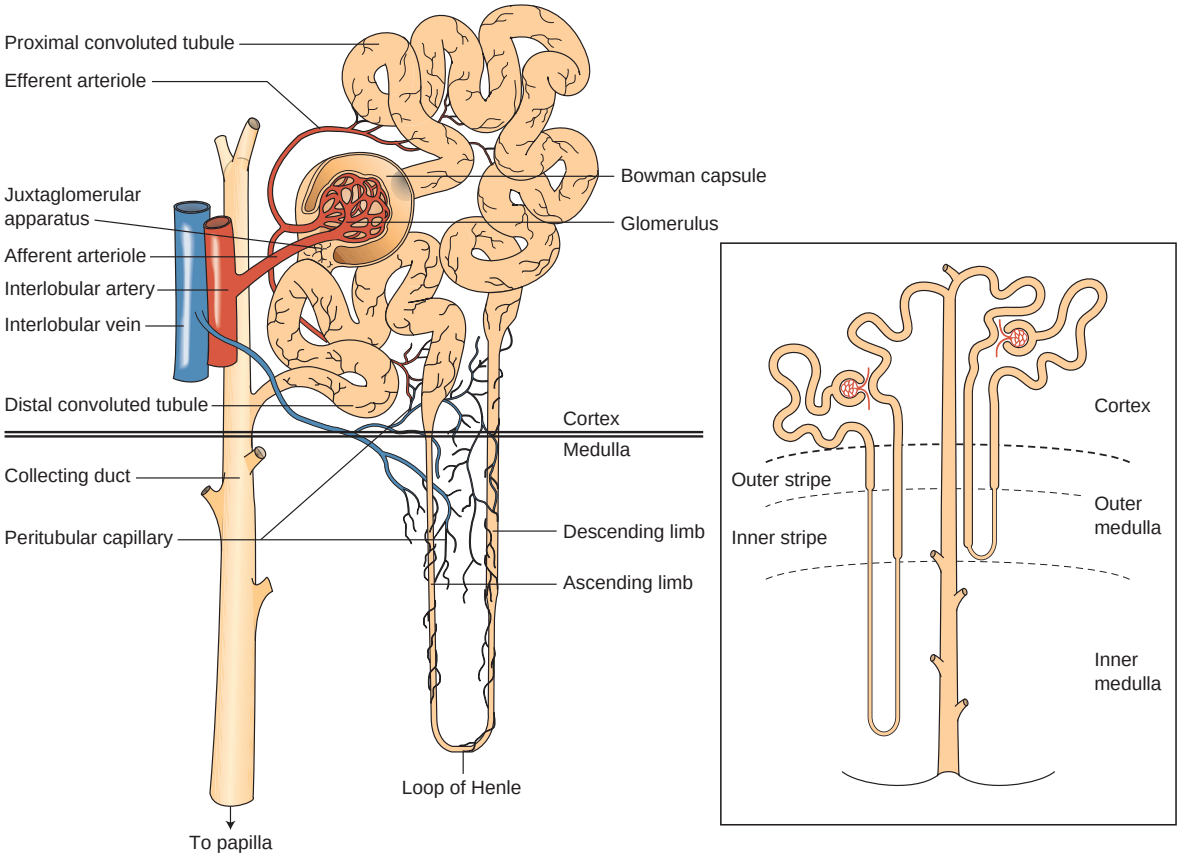
The kidneys help regulate many processes in the body, but they primarily function to excrete metabolic wastes, maintain fluid and electrolyte balance, and regulate acid-base balance. They also have endocrine functions, secreting the hormones renin and erythropoietin and 25-hydroxyvitamin D3-1-hydroxylase, the enzyme that converts vitamin D to the active form.

The functional units of the kidneys are the nephrons (see illustration below). Each kidney contains approximately 1.2 million nephrons and is supplied by a renal artery, which divides into smaller vessels to the level of the nephron where an afferent arteriole delivers blood to the glomerulus. Beyond the glomerulus, the capillaries combine to become an efferent arteriole, which then divides again to form another capillary network that surrounds the tubular system. Eventually the blood returns to the venous circulation.

From 20% to 25% of all cardiac output (1,200 mL/minute) is delivered to the kidneys each minute. The glomerulus has a porous membrane that allows about 125 mL/minute of filtrate to pass through to the Bowman capsule. The glomerular filtration rate (GFR) is the volume of plasma filtered at the glomerulus per unit of time. Normal GFR is 90 to 120 mL/minute/1.73 m^2. In the tubular system, all but about 1 mL/minute is reabsorbed through the peritubular capillary network and becomes urine.

Although the glomerulus allows for so much fluid to be filtered, it doesn't allow larger proteins, such as red blood cells and platelets, to be filtered under normal conditions. However, renal disorders may disrupt this process, allowing protein or blood cells to pass into the urine.

The tubular system functions to reabsorb all glucose, most amino acids and small proteins, many of the electrolytes, bicarbonate, and most of the fluid. This is accomplished by both active and passive transport and is regulated by parathyroid hormone, aldosterone, and antidiuretic hormone as well as other hormones. The tubules also secrete hydrogen ions, potassium ions, creatinine, and ammonia.



In many cases, AKI is related to underlying medical disorders. Older adults are more likely to have one or more chronic diseases that increase the risk of AKI, such as coronary artery disease, heart failure, or diabetes mellitus. Infection such as sepsis also increases the risk of AKI in vulnerable older adults.[19-21] Other causes are iatrogenic—that is, resulting from the patient's medical or surgical treatment. In older patients, AKI usually has several causes and may develop from even mild insults to the kidney.[15]

Many medications are associated with AKI. Some of the most common culprits are nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen; antimicrobials such as aminoglycosides, amphotericin B, vancomycin, and acyclovir; cardiovascular drugs such as ACE inhibitors and angiotensin-receptor blockers (ARBs); diuretics; antidepressants; chemotherapy agents such as cisplatin and methotrexate, and intravascular contrast media.[10-12,22]

Many of the others damage the kidneys directly.[7,10,22] The risk of nephrotoxicity increases when these medications are given over a long period or at high doses, and when more than one nephrotoxic medication is administered.[22] Age-related changes in pharmacokinetics and pharmacodynamics, along with polypharmacy (taking more than five medications) place older patients at a high risk for adverse drug reactions and poor outomes.[14,23]

AKI staging

Guidelines for staging AKI according to severity are included in the Kidney Disease: Improving Global Outcomes (KDIGO) Clinical Practice Guideline for AKI (see *Resources for nurses and patients*).[2,12,14]

- Stage 1: SCr 1.5-1.9 times baseline or greater than/equal to 0.3 mg/dL increase with urine output of less than 0.5 mL/kg/h for 6-12 hours.
- Stage 2: SCr 2.0-2.9 times baseline and urine output of less than 0.5 mL/kg/h for greater than/equal to 12 hours.
- Stage 3: SCr 3.0 times baseline or increase in SCr to greater than/equal to 4.0 mg/dL; or initiation of RRT; or in patients <18 years, decrease in estimated glomerular filtration rate (eGFR) to <35 mL/min per 1.73 m^2 and urine output less than 0.3 mL/kg/h for 24 hours or more; or anuria for 12 hours or more.[2,12]

Clinical etiology

The causes of AKI are traditionally categorized by the part of the renal anatomy most affected.[11,13,19]

- Prerenal AKI refers to conditions that decrease renal perfusion, such as hypovolemia, decreased cardiac output, and acute hemorrhage.[11,13,24,25] NSAIDs can cause afferent arteriole vasoconstriction, resulting in acute prerenal AKI (See *Efferent and afferent arterioles*).
- Intrarenal (intrinsic) AKI results from direct injury to the kidneys (blood vessels, glomeruli, or tubules-interstitium). Possible causes include ischemia resulting in prolonged decrease in renal perfusion, nephrotoxins,[14] infections, atheroembolic renal disease, and primary renal disease.
- Postrenal (obstructive) AKI results from obstruction of urine outflow by tumors, calculi, neurogenic bladder, or prostate gland enlargement.[10,11,13,25] The obstruction causes urine to back up into kidney structures, causing increased pressure, decreased GFR, and kidney injury. Although postrenal AKI is less common in the general population, aging increases the risk.

Clinical presentation

When kidney function abruptly declines in AKI, SCr and BUN increase.[9] Oliguria (urine output less than 0.5 mL/kg/hour) usually occurs, although in some cases urine output is initially normal or even increased.[19] Additionally, fluid, electrolyte, and acid-base balance can all become impaired.[9,24]

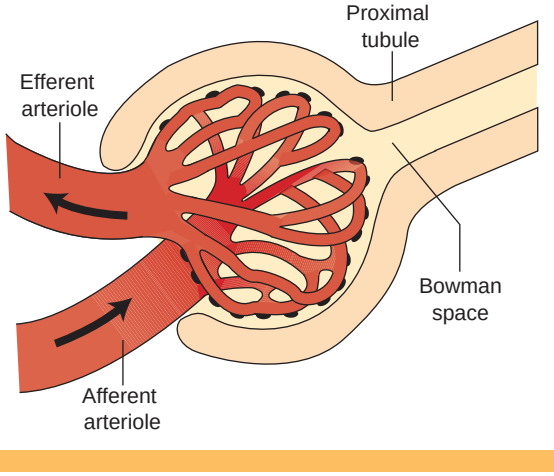
Patients with mild to moderate AKI may be asymptomatic despite abnormal lab values. As the kidney injury progresses, signs and symptoms include listlessness, confusion, fatigue, anorexia, nausea, vomiting, peripheral edema, and weight gain.[10,13]

AKI usually follows a predictable clinical course, although the severity of the injury, complexity of the kidney dysfunction, development of complications, and length of time to recovery can vary considerably. The clinical course generally follows these four phases:[13,25]

- Initial (onset) phase. This is the time between the kidney injury and

Efferent and afferent arterioles

This cross-section of the glomerulus shows the efferent and afferent arterioles, which directly control glomerular blood flow.



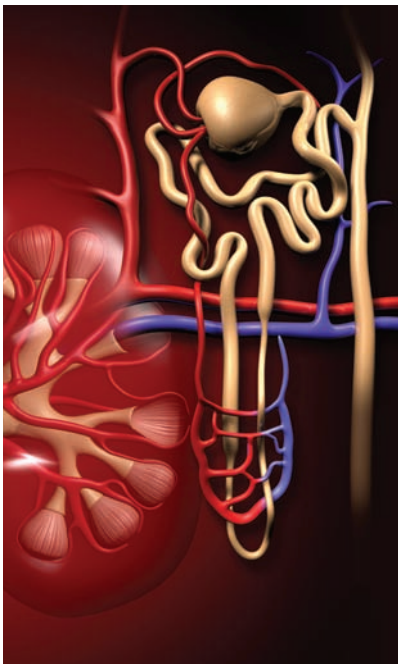
the reduction in kidney function. Nurses can help prevent or minimize subsequent injury by identifying hypotensive episodes, nephrotoxic agents, and other risks, and then intervening appropriately.

• Oliguric phase. During this phase, urine output decreases to below 400 mL/day.[13] In AKI, this phase usually occurs 1 to 7 days after the kidney injury and lasts 10 to 14 days. However, in some cases the oliguric phase lasts for weeks or months.[25]

Some patients with AKI don't develop oliguria (nonoliguric AKI) and maintain adequate urine output. No consensus exists as to whether patients with nonoliguric AKI have a poorer outcome than those with oliguric AKI. Although some sources state that patients with nonoliguric AKI have better outcomes, others dispute this because, in some instances, it may result in a delay of aggressive treatment, increasing mortality.[7,25]

If a patient develops oliguria, likely findings include fluid volume overload, hyponatremia (impaired renal reabsorption of sodium and dilutional hyponatremia), hyperkalemia, metabolic acidosis, and elevations of BUN and SCR, reflecting nitrogenous waste accumulation.[13,25] If this phase continues for a significant period, the patient may also develop hypocalcemia, hyperphosphatemia, anemia, platelet abnormalities and bleeding, immune system dysfunction, and various neurologic changes ranging from fatigue to seizures and coma.[9,13,25]

Oliguria can also be caused by hypovolemia, which must be recognized and corrected to prevent AKI. To determine whether oliguria stems from hypovolemia or AKI, assess urine specific gravity and urine sodium levels. Specific gravity reflects the ability of the kidneys to concentrate or dilute urine and is normally between 1.010 and 1.025 in adults.[1] (Reference values may differ slightly from one lab to another.) Urine of a patient with oliguria related to hypovolemia will have a high specific gravity and low urine sodium level (normal is 40 to 220 mEq/24 hours in adults). In a patient with oliguria from AKI, specific gravity is fixed at 1.010 and the urine sodium level is high.[1,25]



Each kidney contains approximately 1.2 million nephrons and is supplied by a renal artery.

• Diuretic phase. In this phase, urine production increases because the nephrons have regained the ability to excrete urea, which draws the fluid across the glomerular membrane (osmotic diuresis). However, the kidneys can't concentrate that filtrate. Urine output during this phase is usually 1 to 3 L/day, but can be as high as 5 L/day.[25] Because of fluid loss, the patient experiences hypovolemia, hypotension, continuing hyponatremia (due to sodium loss in the filtrate), and hypokalemia.

As this phase continues, acid-base and electrolyte imbalance begin to normalize and BUN and SCr values improve. This phase can last for 1 to 2 weeks.[13,25]

• Recovery phase. The kidneys regain the ability to manage metabolic waste, and BUN and SCr return to baseline. This process usually takes several weeks, but in some cases, it continues for up to a year.[13,25] In some patients, the kidneys never fully recover and mild elevations in BUN and creatinine may continue. Other patients will progress to CKD and need lifelong management.

The nurse should obtain a detailed history for any patient at risk for AKI to investigate whether the patient might have CKD rather than AKI. A patient with CKD is more likely to have chronic normocytic anemia, hypocalcemia, hyperphosphatemia, and a grayish cast to skin color, in addition to elevated BUN and SCr.[9]

Assessment guidelines

When assessing a patient at risk for AKI, follow these guidelines.

• Ask about any recent infections, because some kidney injuries are associated with infections. Acute poststreptococcal glomerulonephritis and *Escherichia coli* gastrointestinal infection are examples.[9,20,21]

• Assess for the possibility of rhabdomyolysis, which results from large amounts of myoglobin, which is nephrotoxic, being released from injured skeletal muscle. Possible causes include trauma, muscle overexertion, drug overdose, or other types of skeletal muscle injury.[9] Muscle compression, crush injuries, and prolonged immobility can also trigger myoglobin release. For example, Mr. R is at risk for rhabdomyolysis because he was forced to lie in one position for hours after falling and fracturing his hip.

• Determine whether the patient has any history of cardiovascular disease, which may increase the likelihood of impaired renal perfusion. Any drop in cardiac output negatively affects renal perfusion, so assess for any dysrhythmias or evidence of heart failure or hemodynamic instability.

• Also assess for adverse events during the course of the patient's hospitalization that might affect kidney function. For example, determine whether the patient experienced any episodes of hypotension that could have damaged the kidneys. Investigate any possibility of sepsis, as this can cause profound hypotension.
• Determine whether the patient received mechanical ventilation. Some studies have suggested that mechanical ventilation, including the use of positive end-expiratory pressure, can alter cardiac output and contribute to development of AKI.[24]
• Perform medication reconciliation on patient admission and assess for potentially nephrotoxic drugs. Besides prescription drugs, specifically ask about over-the-counter medications, as well as herbal remedies and nutritional supplements.
• Review medications prescribed for your patient while hospitalized. Include any intravascular contrast media exposure, which could have been administered for various diagnostic or interventional radiologic procedures, including computed tomography (CT) and cardiac catheterization. Double-check the dosing of prescribed medications as well. In high-risk patients, anticipate whether peak and trough levels are to be monitored. Also consider that the dosages of some medications are weight-based. Note whether dosing was based on ideal or actual body weight. Some medications have been implicated in AKI when dosing is incorrect.[26]
• Assess and closely monitor the patient's fluid status. Start with identifying any recent changes in urination pattern, which may signal hypovolemia or fluid overload. Many patients with hypovolemia experience orthostatic hypotension.[27] Instruct patients with orthostatic hypotension to change their positions slowly and to sit or lie down immediately if they feel lightheaded when standing. Compression stockings can also help with this.
• Signs and symptoms of fluid overload include hypertension, jugular vein distension, acute weight gain, and peripheral edema.[9,13] An S3 may also be auscultated. Assess for pulmonary crackles and monitor for dyspnea, orthopnea, and paroxysmal nocturnal dyspnea as well. If fluid overload continues, the patient may develop ascites and pericardial and/or pleural effusions.[25] Prepare the patient for invasive monitoring if the patient is hemodynamically unstable.[7]

Resources for nurses and patients

- **National Kidney Foundation.** This is a leading organization in the United States dedicated to the awareness, prevention, and treatment of kidney disease. www.kidney.org.
- **National Kidney Foundation. Kidney Disease: Improving Global Outcomes (KDIGO).** Issued in March 2012, these were the first published guidelines for AKI. They cover the definition, risk assessment, evaluation, treatment, and prevention of AKI. www.kdigo.org/clinical_practice_guidelines/pdf/KDIGO%20AKI%20Guideline.pdf.
- **National Institute of Diabetes and Digestive and Kidney Diseases.** This National Institute conducts, supports, and coordinates research on kidney disease as well as diabetes and digestive diseases. It provides patient resources and clinical trial referrals and information. www.niddk.nih.gov.
- **National Institute for Health and Care Excellence (NICE).** Acute Kidney Injury: Prevention, Detection and Management. NICE guidelines [CG169] (United Kingdom) was published in August 2013. It's designed for nonrenal specialists to aid in the identification of patients with AKI. Placing an emphasis on patient-centered care and patient wishes, these guidelines address risk factors, prevention, causes, disease management, and support for patients and caregivers. www.nice.org.uk/guidance/cg169/chapter/1-recommendations#assessing-risk-of-acute-kidney-injury.

Monitor lab values closely

As fluid status changes, so does the patient's electrolyte status. Monitor BUN, SCr, and serum electrolytes, particularly sodium, potassium, magnesium, calcium, and phosphate.

Many older adults have decreased muscle mass and protein intake, which impacts the rate of creatine production. SCr levels are often within normal limits despite a significant decrease in GFR, so don't rely solely on SCr to estimate the GFR in older adult patients.[7,12,15] Monitoring hourly urine output along with SCr measurement can be a sensitive and early biomarker for AKI.[13] Serum sodium levels will be low as the damaged tubules prevent sodium reabsorption from the glomerular filtrate.[25]

Hyperkalemia occurs because the normal excretion of potassium is impaired. Additionally, potassium levels will increase following tissue trauma, bleeding, or blood transfusions because potassium is released from damaged cells. If the patient is experiencing metabolic acidosis, the serum potassium levels increase as hydrogen ions enter the cells and force potassium out of the cells.[25]

Hyperkalemia is particularly worrisome because it can cause cardiac dysrhythmias, so patients should be placed on a cardiac monitor and monitored for abnormalities including tall, peaked T waves, prolongation of the PR interval, widening QRS complexes, and premature ventricular contractions.[9,25] Untreated hyperkalemia may lead to ventricular tachycardia, ventricular fibrillation, and cardiac arrest.

Metabolic acidosis is another common sign of AKI. It develops because hydrogen ion secretion is impaired. Arterial blood gas (ABG) analysis shows a low pH and low bicarbonate. Bicarbonate is low because it's used to buffer the elevated hydrogen ions.[25]

The patient may also exhibit Kussmaul respirations (rapid, deep breaths), which is the body's attempt to restore the acid-base balance

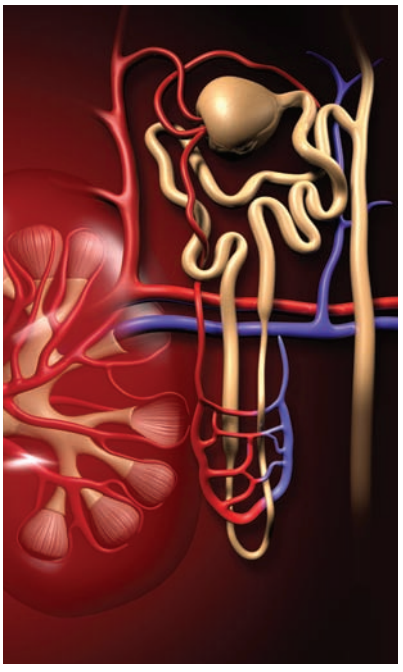
by removing more carbon dioxide from the system.[9,25] Other signs and symptoms of metabolic acidosis include flushed skin, headache, tachycardia, nausea, and vomiting. As acidosis becomes more severe, the patient may develop hypotension, bradycardia, and altered level of consciousness.[9]

Carefully assess the patient's current and prehospital nutritional status. ***Protein-calorie malnutrition*** has been implicated in increased mortality for patients with AKI.[9] Studies show that 20% of hospitalized patients are undernourished, and older adults are at particular risk.[7,28] The accumulation of metabolic waste products in AKI is associated with anorexia, nausea, vomiting, and fatigue, which also impair your patient's dietary intake. Pay close attention to serum albumin, total protein, blood glucose, hemoglobin, cholesterol, prealbumin, transferrin, creatinine, and BUN levels. While serum albumin is the best measure of long-term malnutrition due to its longer half-life, all of these lab tests can provide important information about a patient's nutritional status.

Diagnostic studies

These diagnostic studies may help to determine the underlying cause of AKI.

- urinalysis. Urine will be checked for specific gravity, osmolality, and sodium levels. Protein or cells in the urine may indicate intrarenal damage, such as glomerulonephritis, kidney infection, or Goodpasture syndrome. Hematuria, pyuria, or urinary crystals may indicate a postrenal cause of AKI such as benign prostatic hyperplasia, neurogenic bladder, or renal calculi.[25]
- urine culture and sensitivity, if the healthcare provider suspects an infectious cause.
- renal ultrasound, which may show renal tissue damage or urinary tract obstruction.
- renal scan to assess renal blood flow.



Correct low blood volume and low cardiac output as soon as possible to prevent further kidney damage.

- CT scan or magnetic resonance imaging to identify masses or obstructions.

Occasionally a renal biopsy is performed to investigate possible intrarenal disorders such as glomerulonephritis and interstitial nephritis.

Nursing and collaborative care

Because no curative treatment exists for AKI, the goal of care is to prevent further injury and facilitate recovery.[12,29] Patient care includes prompt identification and appropriate management of the underlying cause of AKI. Other priorities are correcting fluid and electrolyte imbalances, maintaining acid-base balance, providing optimal nutrition, and preventing complications.[7,9,11] Reassess the patient frequently to detect subtle changes that may signal the progression of AKI, the development of complications, or the need for RRT. To provide holistic care, also attend to the patient's and family's spiritual, emotional, and educational needs.

Monitoring the patient for fluid imbalances is critical. Accurately document intake and output and daily weights. If blood volume or cardiac output is low, this must be corrected as soon as possible to prevent further kidney damage. In the absence of hemorrhagic shock, KDIGO guidelines suggest administering isotonic crystalloids rather than colloids (albumin or starches) as the initial management for intravascular volume expansion in patients with AKI or at risk for AKI.[2] Fluid replacement usually begins with 0.9% sodium chloride.[9] KDIGO guidelines suggest not using diuretics to prevent or treat AKI except in the management of volume overload.[2]

If cardiac output continues to be compromised, the patient may require positive inotropes. However, administering low-dose dopamine, a strategy once thought to benefit patients with AKI by improving renal perfusion, is no longer recommended.[2] Research has shown that using it makes no difference in clinical outcomes. In addition, dopamine can cause cardiac complications and even decrease renal perfusion in patients over age 55. For these reasons, inotropic medications are indicated only for patients with inadequate cardiac output.[7]

If the patient is hypervolemic, restricted fluid intake may be prescribed. Generally, patients should receive only 500 to 600 mL above any fluid loss over the previous 24 hours.[7,25] In other words, if a patient had 400 mL of urine output yesterday and no other fluid loss (from vomiting, diarrhea, bleeding, or other causes), he or she may be restricted to a total of 900 to 1,000 mL of total fluid intake for the day.

Monitor a hypervolemic patient closely for signs and symptoms of worsening heart failure and pulmonary edema. Respiratory deterioration can occur rapidly, so frequently

assess for increased work of breathing and decreasing oxygenation. The patient may need supplemental oxygen or possibly mechanical ventilation.[7]

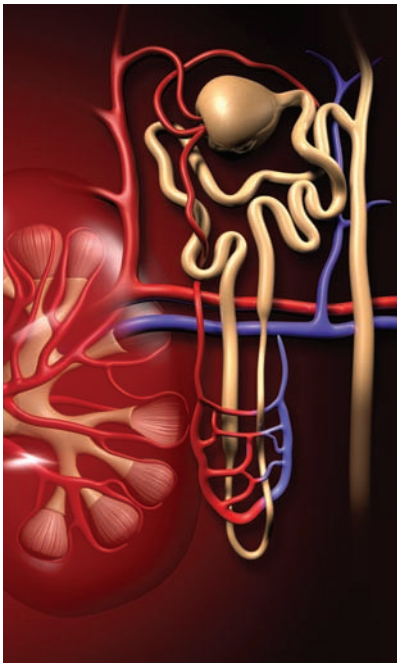
As previously discussed, patients with AKI are at high risk for developing hyperkalemia. If serum potassium is greater than 6.5 mEq/L, I.V. insulin may be prescribed to help drive potassium into the cells. In addition, I.V. dextrose may be prescribed to prevent hypoglycemia, depending on the patient's serum glucose level. Sodium bicarbonate will also help drive potassium into the cells and help correct metabolic acidosis as well.[7,25]

The effects of both of these therapies (insulin/glucose and sodium bicarbonate) are only temporary because potassium will eventually shift out of the cells again. Carefully monitor lab values as prescribed to evaluate the patient's response to treatment.[7,25]

Calcium gluconate or calcium chloride may be prescribed to antagonize the toxic effects of hyperkalemia at the myocardial cell membrane and prevent dysrhythmias.[7,25] Additional emergency treatments for hyperkalemia include nebulized albuterol and cation-exchange resin (sodium polystyrene sulfonate).

If these therapies don't resolve the hyperkalemia, the patient may need RRT.[7] Besides ongoing hyperkalemia, indications for RRT include volume overload, compromised oxygenation, metabolic acidosis, cardiac dysrhythmias, pericarditis, pericardial effusion, and impaired neurologic status.[25]

A thorough nursing assessment helps identify any possible nephrotoxic agents; work with the healthcare team to eliminate them from the patient's care plan. If your patient has received intravascular contrast media, note any contrast-induced AKI prophylaxis used. Current recommendations include I.V. fluid hydration with 0.9% sodium chloride solution, which has been shown to be most beneficial when administered before and after the procedure.[30] Additional prevention strategies include the use of low-osmolar or iso-osmolar contrast media and using the lowest dose of contrast media possible.[2]



KDIGO guidelines suggest not using diuretics to prevent or treat AKI except in the management of volume overload.

Be alert to any prescribed medications, or metabolites of medications, that are excreted by the kidneys. Therapeutic drug level monitoring (peak and trough) may be needed to help determine appropriate dosing. Be aware of specific timing of peak and trough specimen collection, communicate the results to the healthcare team, administer medications on a strict schedule, and adjust doses frequently as prescribed.

Continue to monitor for signs and symptoms of infection, the leading cause of death in patients with AKI. Because your patient has immune system dysfunction, implement infection control measures, especially meticulous hand hygiene, and ensure that everyone on the unit does so as well. Educate your patient and family about this, especially hand hygiene.[9,25]

Teach a patient at risk for atelectasis how to use an incentive spirometer and to do coughing and deep-breathing exercises. If mobility is impaired, turn and reposition the patient at least every 2 hours to prevent pooling of pulmonary secretions and to prevent skin breakdown. If the patient can be out of bed, explain the importance of early, aggressive ambulation. Inspect the patient's skin daily and implement additional pressure ulcer prevention strategies as indicated.

Prevent urinary tract infections by using an indwelling urinary catheter only if appropriate indications are met and discontinuing it as soon as possible. If your patient needs a urinary catheter, maintain a closed drainage system and unobstructed urine flow. Perform meatal care with soap and water.[31]

Manage peripheral and central venous access devices and dialysis access catheters with strict sterile technique, following facility policies. Collect urine, stool, blood, or sputum specimens for culture and sensitivity as indicated. Administer prescribed antibiotics strictly on time to prevent any drop in therapeutic drug levels.

The patient with AKI needs adequate calorie intake to prevent catabolism of body protein. KDIGO guidelines suggest a total energy intake of 20-30 kcal/kg/d in patients with AKI.[2] Administer antiemetic medications as prescribed if the patient is experiencing nausea or vomiting. The patient may also need potassium, sodium, and fluid restrictions. Consult with the dietitian to arrange for appropriate nutritional intake and monitor the patient's protein intake.[9] A patient who needs enteral and/or parenteral nutrition may need blood glucose monitoring and RRT to remove excess fluid.[25] Enteral nutrition has been shown to

be safe and effective in patients with AKI and is preferred over parenteral nutrition.[2]

Early identification and prevention of dehydration, especially in older patients, is critically important. In older adults, many typical signs and symptoms of dehydration may be vague or absent. Orthostatic hypotension and tachycardia are the most clinically relevant findings. Unless fluid intake is restricted, older adults should have 30 mL of daily fluid intake per kilogram of body weight. For example, a patient who weighs 75 kg should have a fluid intake of 2,250 mL/day.[32] A BUN-to-creatinine ratio greater than or equal to 25 suggests dehydration in the older adult.[16]

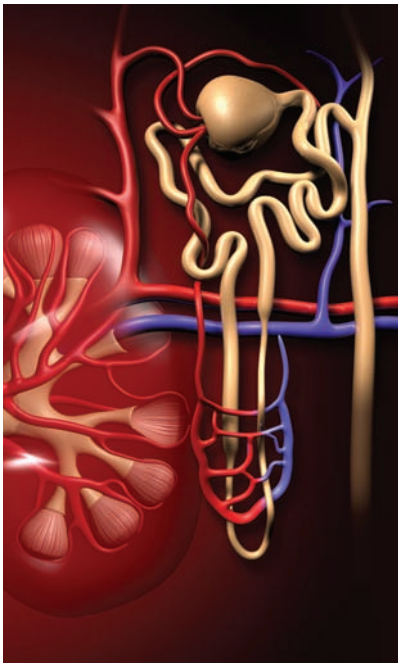
Patients with AKI may experience xerostomia (dry mouth), a metallic taste, and an unusual breath odor from bacterial interaction with urea in the saliva.[9,25] Oral lesions may also develop. Assist with frequent oral hygiene and moisturize the oral mucosa and lips every 2 to 4 hours to prevent breakdown and improve comfort.

Decreased mobility and edema can impair tissue perfusion and increase the risk of pressure ulcers.[9] Assess the condition of your patient's skin every shift and include strategies for preventing pressure ulcers in the care plan, including ambulation depending on the patient's clinical status.

Psychosocial considerations

Patients and families experience a range of emotions associated with an AKI diagnosis. For example, a patient may fear developing CKD and needing lifelong dialysis with all the associated lifestyle changes. Your education about AKI and the course of recovery will be important to helping the patient maintain a positive outlook.

Hospitalized patients often feel overwhelmed due to the amount of complex information, sensory overload from the environment, unfamiliar caregivers, and interruption of normal routines. A sense of losing control and the possibility of a long illness can cause frustration. Disruption of family and work roles is another source of stress. Make rounds with the other members of the healthcare team to stay on top of the interdisciplinary care plan so you can advocate for the patient's needs. This strategy also helps the nurse answer questions when the patient asks for more explanation about what the dietitian or the healthcare provider meant.



Protein or cells in urine may indicate intrarenal damage, such as glomerulonephritis, kidney infection, or Goodpasture syndrome.

Success story

Mr. R was discharged 2 weeks after his right THA. When performing an initial assessment, his nurse had recognized his many risk factors for AKI, including advanced age, prolonged immobility, and use of ibuprofen and an ACE inhibitor. Early identification of his decreased urine output helped prevent many of the complications of AKI, and he regained kidney function without needing dialysis. His healthcare provider and home healthcare nurse will closely monitor his BUN and SCr as his recovery progresses.

Kidney damage associated with AKI is often reversible with quick attention, so never underestimate the importance of the nurse's role in the assessment and care of a patient with AKI. ■

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Acute kidney injury: Limiting the damage

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