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Acute Kidney Injury

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Abstract

This article discusses the role of nurses in early identification of signs and symptoms of acute kidney injury (AKI) and then planning and implementing an appropriate nursing intervention. This article will give brief details about the physiology of the kidneys, risk factors, etiology, staging, clinical presentation, assessment guidelines and holistic nursing care of acute kidney injury.

Defining acute kidney injury

Acute Kidney Injury is an abrupt decrease in kidney function significant by any one of these parameters;

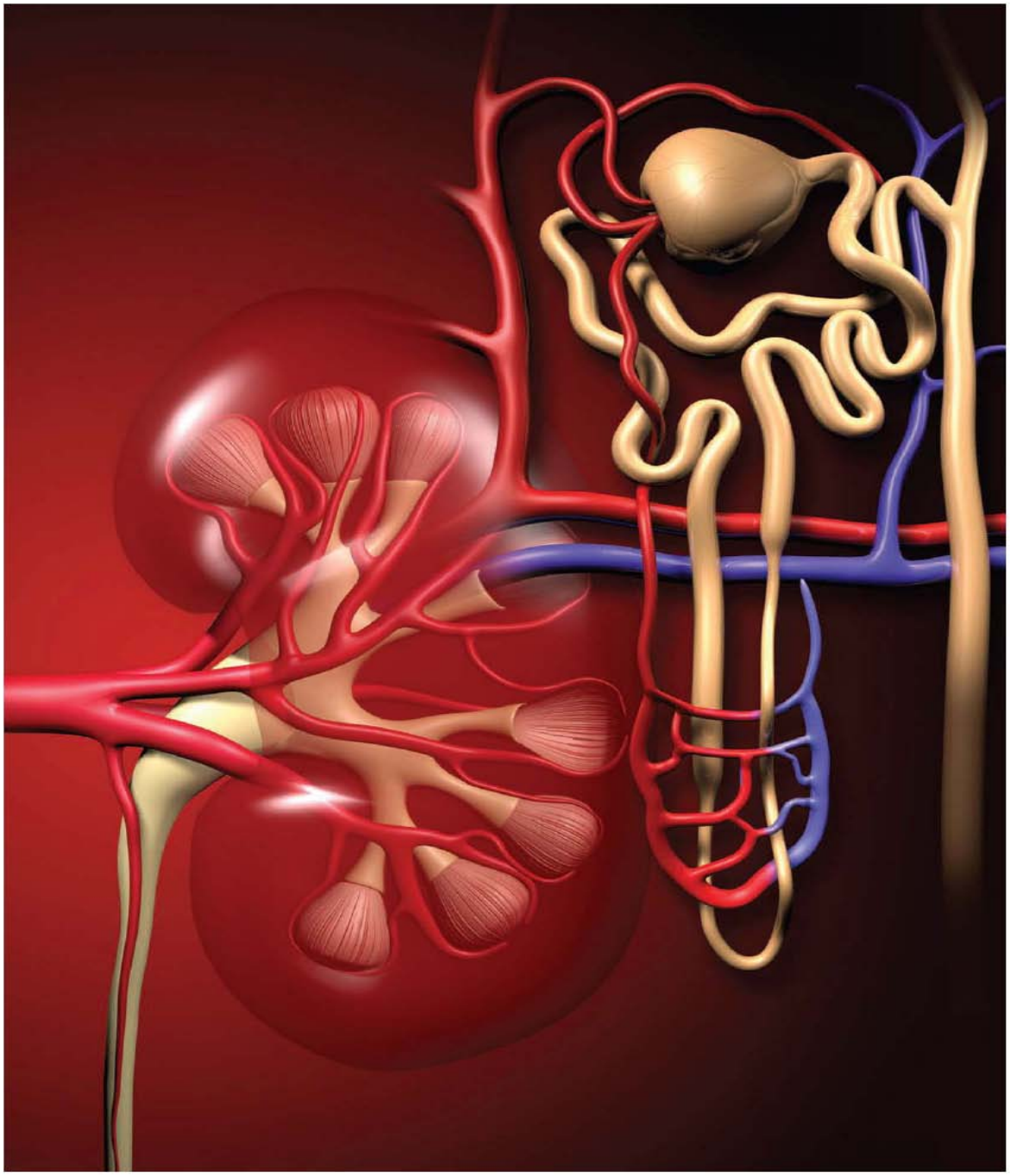
Serum creatinine increase by 0.3 mg/dL or more within 48 hours

Serum creatinine increases up to 1.5 times or more the baseline value within the prior 7 days

Urine output decreases to less than 0.5 mL/kg/h for 6 hours.

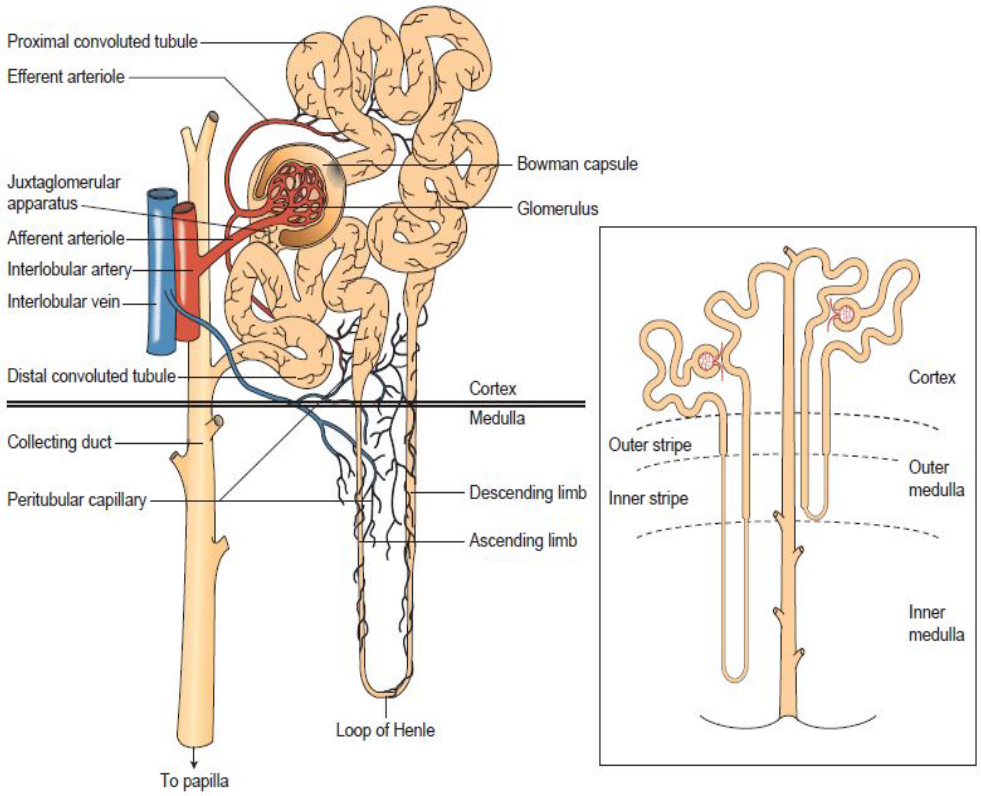
Acute renal failure (ARF) was replaced by the term acute kidney injury to better reflect the spectrum of injury ranging from minor changes in renal function markers to the need for renal replacement therapy. Renal replacement therapy includes intermittent hemodialysis, slow continuous ultrafiltration, continuous venovenous hemofiltration, continuous venovenous hemodialysis, continuous venovenous hemodiafiltration, and continuous arteriovenous hemofiltration.

This condition is marked usually by a rise in serum creatinine concentration or by a rise in blood urea nitrogen [BUN] concentration (azotemia). But, in some cases soon after a kidney injury, BUN or creatinine levels may be normal, and the only possible sign of a kidney injury may be decreased urine production.



Physiology of kidneys

Kidneys primary function is to excrete metabolic waste, maintain fluid and electrolyte balance and regulate acid-base balance. Along with this, it also secretes hormones such as renin, erythropoietin. An enzyme which converts vitamin D to the active form, 25-hydroxyvitamin D3-1-hydroxylase is also secreted by the kidneys. These functions are carried out by the functional units called nephrons. Each kidney comprises of 1.2 million nephrons which are supplied by a renal artery and its branches. Each nephron consists of afferent arteriole which delivers blood to the glomerulus. After which these capillaries combine into efferent arteriole, this is further divided to a network surrounding the tubular system returning blood to the venous system. Each minute 20 – 25 percentage of cardiac output is delivered to kidneys.



Glomerulus, a porous membrane which allows about 125ml per minute to pass through Bowman capsule. This 90 to 120 ml of plasma filtered per minute through the glomerulus is called as the glomerular filtration rate (GFR). Urine is formed in the capillary network by reabsorption at a rate of 1 ml per minute in the tubular system through the peritubular capillary network. Normally larger proteins like red blood cells and platelets do not pass through these filter but, renal disorders may disrupt this allowing the blood cell filtered which results in presence of protein and blood cells in urine. Secretory function of the kidneys is responsible for the production of hydrogen, potassium ions which help in regulation of acid-base balance of the blood. The mechanism of active and passive transport allows kidneys to reabsorb glucose, amino acids, electrolytes, bicarbonates and small proteins which are regulated by the parathyroid hormone.

Risk factors

1. Age 75 or older: Advancing age is an important factor predisposing a patient to AKI. As a person ages, the kidney undergoes structural and functional changes
2. Diabetes;
3. Hypertension
4. Preexisting chronic kidney Disease (CKD)
5. Heart or liver failure
6. Sepsis
7. Use of intravascular radiocontrastAgents
8. Cardiac surgery after use of a Radiocontrast agent
9. Polypharmacy: Many medications are associated with AKI. Some of the most are nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen; antimicrobials such as aminoglycosides, amphotericin B, vancomycin, and acyclovir; cardio vascular drugs such as ACE inhibitors and angiotensinreceptor blockers; diure tics;

antidepressants; chemotherapy agents such as cisplatin and methotrexate; and intravascular contrast media.

AKI staging

It is based on the severity of the kidney disease and the level of serum creatinine, GFR and urine output.

There are three main stages;

1. Stage 1:
   a. Serum Creatinine 1.5 to 1.9 times baseline or greater than/equal to 0.3 mg/dL
   b. Increase with urine output of less than 0.5 mL/kg/h for 6 to 12 hours.
2. Stage 2:
   a. Serum Creatinine 2.0 to 2.9 times baseline and
   b. Urine output of less than 0.5 mL/kg/h for greater than/ equal to 12 hours.
3. Stage 3:
   a. Serum Creatinine 3.0 times baseline or increase in Serum Creatinine to greater than/equal to 4.0 mg/dL; or initiation of RRT; or in patients younger than 18 years,
   b. Decrease in estimated GFR to less than 35 mL/min/1.73 m2 and
   c. Urine output less than 0.3 mL/kg/h for 24 hours or more; or anuria for 12 hours or more.

Etiology

The etiology is categorized based on the affected part of nephron in the kidney.

Category	Part of nephron affected	Clinical condition	Causes
1. Prerenal	Afferent arteriole is affected as a results of vasoconstriction	Decrease renal perfusion Hypovolemia Decreased cardiac output, and Acute hemorrhage	NSAIDs
2. Intrarenal or intrinsic	blood vessels, glomeruli, tubules, or interstitium as a result of ischemia	decrease in renal perfusion	Nephron toxins
3. Postrenal or obstructive	Obstruction of urinary tract	Increased pressure, decreased GFR, and kidney injury	Tumors Calculi Neurogenic bladder, or prostate gland enlargement

Clinical presentation

Clinical course of AKI is very predictable however, the degree of injury and the complexity of the affected part' function and duration of the illness varies considerably. Some of the most common signs and symptoms of AKI are;

- Oliguria, urine output is initially normal or even increased
- Impairment of fluid, electrolyte, and acid-base balance
- Listlessness
- Confusion
- Fatigue
- Anorexia,
- Nausea and Vomiting

- Peripheral edema
- Weight gain

Four phases of the general clinical course are as follows;

1. Initial (onset) phase. The time between the injury and the reduction in kidney function. Appropriate nursing action such as identifying hypotensive episodes, nephrotoxic agents, and other risks can help prevent or minimize subsequent injury.
2. Oliguric phase. In the first seven days after onset the urine output decreases considerably to 400ml per day and could last for 10-14 days and in some case even up to weeks or months.
3. Diuretic phase. The phase after oliguria in which the nephrons recover from the injury and results in increased urine production is called as diuretic phase lasting for 1 -2 weeks. But the ability of glomerular membrane to excrete urea is diminished which lead to the inability to concentrate the urine. The increase in urine output may be usually from 1-3 liters per day and can be as high as 5 liters per day which often results in hypovolemia, hypotension, hyonatremia and hypokalemia. This gradually improves the acid base balance and normalization of blood urea nitrogen and serum creatinine levels in blood.
4. Recovery phase. This phase begins as soon as the kidneys recover completely and begin to excrete metabolic waste. The blood urea nitrogen and serum creatinine levels in blood will be back to normal. This usually takes weeks to years and in some it never recovers leading to chronic kidney disease which would necessitate lifelong management or renal replacement therapy. A detailed history is very important in differentiating acute kidney injury from chronic kidney disease. Some of the keys features of chronic kidney disease are normocytic anemia, hyperphosphatemia, and hypocalcemia,

Assessment guidelines

1. History of infections like Acute post streptococcal glomerulonephritis and gastrointestinal infection
2. History of trauma like injured skeletal muscle resulting from trauma, muscle overexertion or drug overdose. Any situations which could result in release of myoglobin such as Muscle compression, crush injuries, and prolonged immobility.
3. History of cardiovascular disease which could result in impaired renal perfusion.
4. History of hypotensive episode because of failed airway or breathing.
5. Medication history would give clues related to nephrotoxic drug use.
6. Signs and symptoms of fluid overload or fluid loss.

Monitor lab values closely

1. Blood Urea Nitrogen
2. Serum Creatinine
3. Serum Electrolytes particularly sodium, potassium, magnesium, calcium, and phosphate.

Nursing and collaborative care

The primary goal of care in acute kidney injury is to prevent further injury and facilitate recovery. Nursing care of patient consists of early identification and appropriate intervention of the cause behind AKI. Some other priorities include fluid and electrolyte imbalance correction, maintenance of acid-base balance, adequate nutrition and very close monitoring of signs and symptoms of any complication.

The most crucial part of monitoring is the intake and output of fluids. Daily weight and accurate documentation are imperative in the correction of fluid and electrolyte imbalance. Correction of fluid imbalance will prevent further damage to the kidneys.

Fluid correction can be initiated by administering isotonic crystalloids in the case of non-hemorrhagic shock rather than colloids like albumin or starches which would increase the intravascular volume for the patient who is in AKI or at risk for AKI.

Fluid replacement therapy begins usually with 0.9% sodium chloride except in the management of volume overload. Use of low dose inotropic drugs such as dopamine is not recommended because of its increase risk in cardiac complications rather than increasing renal perfusion. Inotropic drugs are recommended for patients with inadequate cardiac output In the case of hypervolemia fluid restriction is initiated by reducing the fluid intake of the patient to 500-600ml plus any fluid loss per day.

Signs and symptoms of cardiac failure and pulmonary edema such as respiratory deterioration, increased work of breathing and decreasing oxygenation must be monitored closely and precautions for respiratory failure and oxygen supplement should be made available.

The risk of hyperkalemia is well documented with AKI so initiating insulin and dextrose in the case of serum potassium values greater than 6.5 mEq/L. along with this Sodium bicarbonate I.V will push the potassium into cells. However, this approach is temporary as the potassium eventually is released by the cells which necessitate the importance of closer monitoring. Calcium gluconate or calcium Chloride may act as an antagonist in counteracting the toxic hypokalemic effects which left untreated may lead to cardiac dysrhythmias. Nebulization with albuterol and cation exchange resin can be very effective as an emergency treatment.

Some of the indication which is to be monitored for initiating renal replacement therapy are volume overload, compromised oxygenation, metabolic acidosis, pericarditis, pericardial effusion, cardiac dysrhythmias, and impaired neurologic status.Tissue perfusion is greatly affected by decreased mobility and edema (as a result of fluid overload), so assessing the patient skin condition is vital in preventing pressure ulcers. Nursing interventions like skin care, ambulation depending on the physical condition of the patient and adequate maintenance of nutritional status are to be carried out.

Psychosocial considerations

Psychosocial health is one of the most affected because of the fear of prognosis of the disease to a chronic condition which would result in major lifestyle modification. As the patients are overloaded with complex information, unfamiliar environment, and caregivers along with altered regular routines results in increased stress, anxiety and fear.

Nurse’s role is to act an educator by proving all information related to the disease process and the therapy options. Adequate knowledge level would minimize anxiety and fear. In addition to this interdisciplinary rounds and care plan will provide a holistic care to the patient.

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