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Summer 2020

### Acute Kidney Injury

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Boadu, Mary, "Acute Kidney Injury" (2020). *Nursing Student Class Projects (Formerly MSN)*. 418.  
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# Acute Kidney Injury

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## Introduction

A myriad of medical advances has taken effect in our lifetime, but a phenomenon continues with the prevalence of Acute Kidney Injury (AKI). AKI is most prevalent in hospitalized patients in the intensive care units. The possibility of AKI increases by 50% with underlying conditions like diabetes, hypertension and genetic factors (CDC, 2020).

Furthermore, AKI is precipitated by sepsis prognosis and may potentially advance to chronic kidney disease (CKD), hence the importance of identifying, managing and treating AKI. Advanced nurse practitioners should be privy to the most recent information regarding pathophysiology, treatment and continual care for these patients.

## Signs and Symptoms

Patients in the early stage of the disease process may not manifest any signs and symptoms. AKI usually does not have distinct signs and symptoms although decreased urine output occurs in about 70% of the patients (Patschan & Müller, 2015).

Further signs and symptoms include:

- Bleeding complication
- Cardiac arrhythmias
- Diarrhea
- Electrolyte imbalances
- Edema
- Fatigue and tiredness
- Hypertension with heart failure and pulmonary edema
- Nausea and vomiting

(Patschan & Müller, 2015).

## Underlying Pathophysiology

AKI is "defined as deterioration of kidney function, as reflected by a significant increase in serum creatinine in most patients, (70%) urine output is decreased as well" (Patschan & Müller, 2015). AKI may present with or without kidney damage over a few hours or a few days. Most patient susceptible to AKI include diabetic, hypertensive and geriatric patients. (Pavkov, 2018). Following the recommendations of the Kidney Disease Improving Global Outcome (KDIGO). Later stages of AKI can also be diagnosed if the following criteria are fulfilled:

- serum creatinine increase of greater than on .3 mg/dl within 48 hours,
- A 1.5-fold serum creatinine increase in seven days as compared to a known or suspected baseline (Patschan & Müller, 2015).

The most affected population is African Americans, American Indians, and Hispanics who would eventually develop chronic kidney disease (CKD) due to inappropriate or inadequate treatment of underlining conditions. Prevalent in hospitalized patients with complications from sepsis, surgery and heart conditions (Pavkov, 2018).

More evidence suggests that underlying risk factors such as metabolic syndrome, obesity, hypertension, diabetes and cardiovascular disease progressively leads AKI to CKD (Rangaswamy & Sud, 2018). There are three major causes of AKI; pre-renal, intra-renal and post-renal.

AKI is as a result of renal hypoperfusion. Rangaswamy & Sud (2018), classify AKI into three phases, namely the developmental stage, extension stage and resolution stage. The developmental stage can be described as the initial insult which then compromises GFR. This phase may not necessarily show in patients with CKD and geriatrics. The extension phase is the evidence phases where signs and symptoms mentioned above me prevalent. These patients may end up with CKD or need dialysis due to disease progression. The final stage, known as the resolution stage conglomerate the result of injury and repair mechanism (Rangaswamy & Sud, 2018).

Patients with CKD are at increased risk for AKI due to a reduction in glomerular filtration rate (GFR).

Reference: SceinceDirect.com



## Significance of Pathophysiology

AKI can have deteriorating effects on the patient, therefore is pertinent for the advanced clinician to have an in-depth understanding of this disease process. Understanding baseline risk factors, renal stressor and key diagnostics for detecting AKI can prevent further complications. Significance of AKI is particularly important to understand in patients in the intensive care unit (ICU) who are particularly susceptible to the pathophysiology (Acute Kidney Injury (AKI), 2015). Studies suggest that decreased creatinine levels and adequate urine output leads to underestimation of severity of kidney injury. These may not be true measures of kidney function therefore; clinicians should be apt in monitoring susceptible patients.

## Nursing Implication

Several studies strongly reiterate the fact that early detection and management of risks of acuity in kidney injury can minimize the reoccurrence of chronic kidney injury disease (CKD). The emphasis of acute renal injury management and early detection to prevent deterioration. Ostermann et al., concur the use of medication therapy to treat AKI as unsuccessful. They also suggest implementing continual care for patients with acute kidney injury in the prevention of the development of chronic kidney disease. Increasing early detection for acute kidney injury disease will enhance better treatment options and prevent further deterioration to chronic kidney disease in the long term.

## Conclusion

As a student CRNA, AKI prognosis will determine the kind of medication patients receive during surgery. Most medications are eliminated through the kidneys therefore, it is pertinent know the effects of medications administered to these patients. Drug dosing in critically ill patients is challenging due to altered pharmacokinetics and pharmacodynamics, multiple drug interactions and limited evidence to guide accurate prescribing puts patients with acute kidney injury at a high risk for under dosing or overdosing. (Ostermann et al., 2018). To conclude, AKI is becoming more prevalent due to various comorbidities like hypertension and diabetes, this article sheds more light on the importance of early identification and management.

## Additional Sources

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Table adopted from: Patschan & Müller, 2015

**Table 2: Etiology of AKI. Pre-renal AKI accounts for 55%, while intra-renal AKI is being diagnosed in 45% of all patients. Post-renal AKI occurs rarely with 5%.**

pre-renal	arterial hypotension	heart failure, fluid-loss, intensified anti-hypertensive treatment
	acute glomerulonephritis	<b>infections:</b> postinfectious (various bacteria, less frequent: viruses, fungi) <b>autoimmune-mediated diseases:</b> e.g. systemic lupus erythematosus, purpura schenlein-henoch, essential mixed cryoglobulinemia, anti-GBM syndrome, granulomatosis with polyangitis, microscopic polyangitis <b>idiopathic:</b> IgA-Nephropathy, idiopathic rapid progressive GN
intra-renal	acute tubulointerstitial nephritis	<b>drugs:</b> beta-lactams, proton pump inhibitors, allopurinol, NSAID <b>infections:</b> e.g. leptospirosis, streptococcus, EBV <b>electrolyte / metabolic disorders:</b> hypokalemia, hyperuricemia, hypercalcemia <b>autoimmune-mediated diseases:</b> systemic lupus erythematosus, sjögren's syndrome
	acute vasculopathy	renal artery embolism, renal vein thrombosis, thrombotic microangiopathy, renal crisis in systemic sclerosis
	acute tubular necrosis	<b>induced by tubulotoxic drugs:</b> aminoglycosides, cidofovir, foscarnet, vancomycin <b>induced by renal ischemia:</b> blood- / fluid-loss, shock of various origin
post-renal	urinary tract obstruction	hematoma of renal pelvis / ureter, malignancies (bladder, ureter, intestine, uterus), neurological disorders



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