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Acute Kidney Injury

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ACUTE KIDNEY INJURY

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Introduction

Acute Kidney injury (AKI) is sudden decrease in glomerular filtration rate which is manifested by an increase in serum creatinine concentration or oliguria and classified by stage and cause. AKI is said to occur likely in 20% of patients that visit the hospital. Patients that present with volume overload, electrolyte disorders, uremic complications, and drug toxicity tends to cause this injury (Levey, A. S., & James, M. T., 2017).

It is very important for an advance nurse practitioner to understand the pathophysiology of this disease process and the disease mechanism considering the number of cases that are presented to the hospital. Understanding the disease process will help provide better care to the people that are being affected.

Pathophysiological Processes.

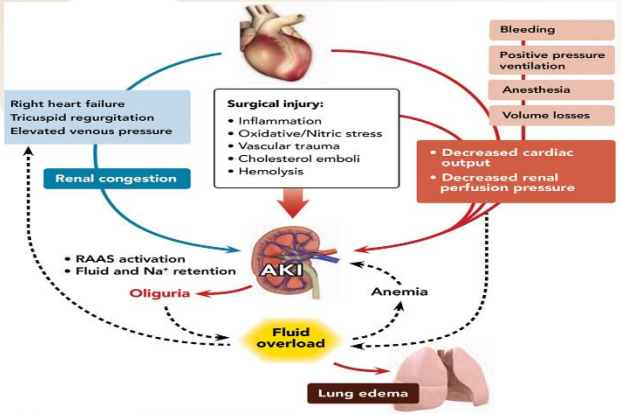
- Acute Kidney Injury is an extensive clinical disorder that is including tubular necrosis, pre-renal azotemia, acute interstitial nephritis, acute glomerular and vasculitic renal diseases, and acute post-renal obstructive nephropathy. Based on the article, the conditions listed previously may cohabit in a single patient (Koza, Y, 2016).

- When the renal blood flow is Impaired, it can lead to hypoxic injury to the renal tubular cells by reducing intracellular ATP, disturbing the intracellular calcium homeostasis, infiltration of leukocytes, damaging the endothelium, releasing cytokines and adhesion molecules and causing apoptosis (Koza, Y, 2016).

- The ischemic model according to the article has another important characteristic of its little relevance to periods of decreased perfusion. This could be seen when a major surgery is performed because 80 percent renal-artery occlusion for 2 hours doesn't result into sustained renal dysfunction (Koza, Y, 2016).

- In Acute Kidney Injury, the renin angiotensin-aldosterone system, the renal sympathetic system, and the tubuloglomerular feedback system are activated. The changes in circulatory induces renal vasoconstriction and can lead to increase release of arginine vasopressin, which contributes to water retention (Koza, Y, 2016).

Reference: keywordsuggest.org/gallery/327766.html

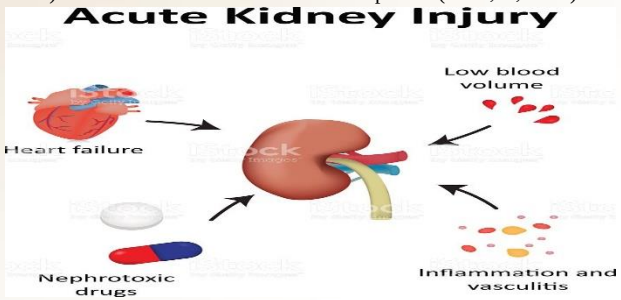


Signs & Symptoms

Acute kidney injury does not have symptoms not until the kidneys begin to fail. Acute kidney injury could be detected through other test that was order by the physician managing the case (Levey, A. S., & James, M. T., 2017).

Some of the signs and symptoms a patient might experience include but not limited to too little urine leaving the body, swelling in legs (Levey, A. S., & James, M. T., 2017). ankles, and around the eyes, fatigue or tiredness, shortness of breath, confusion, Nausea, seizure or coma in severe cases, Chest pain or pressure (Levey, A. S., & James, M. T., 2017).

Since acute kidney injury doesn't have any particular signs and symptoms to watch for until it is detected through the other test from the doctor, it will be very imoortant for the patient to monitor their health closely incase of any unforeseen cases that could occur patient (Koza, Y, 2016).



Reference: https://media.istockphoto.com/vectors/acute-kidney-injury

Underlying Pathophysiology

The incidence of acute kidney injury varies in different patient population and different parameter are used for criteria. Based on the Kidney Disease Improving Global Outcome (KDIGO) clinical practice guidelines, acute kidney injury is increase in serum creatinine by 0.3 mg/dl (≥26.5 µmol/l) within 48 hours; or an increase in serum creatinine to greater than 1.5 times baseline, which is known or assumed to have happened within the preceding 7 days; or a urine volume that is less than 0.5 ml/kg/h for 6 hours (Basile, D. P., Anderson, M. D., & Sutton, T. A., 2012)

Necrosis and apoptosis of tubular cells lead to tubular obstruction, which contributes to the reduction of glomeruli filtration rate (GFR). Elevated intracellular calcium levels from tubular damage cause a series of cellular-level alterations that culminate in increased tubuloglomerular feedback, and thus, diminished GFR (Matthews, E., 2018).

Vascular compromise causes elevated endothelial injury markers, and production of inflammatory mediators that result in reduced GFR; due to tenacious imbalance between the mediators of vasoconstriction and dilatation that result in intrarenal vasoconstriction and, finally, ischemia. High levels of vasoconstrictors and low levels of vasodilators cause continued hypoxia and cell damage or cell death during the process of acute kidney injury. Ongoing research into the pathophysiology of AKI may yield potential targets in the clinical management of this syndrome (Levey, A. S., & James, M. T., 2017).

Significance of Pathophisiology

Understanding the patho-physiology of this disease process is very beneficial to both the clinician and the patient.

An indepth understanding of the disease process by the clinicians will help clinicians to easily explain the etiology of the disease process to the patients and manage the disease to the best of their ability. Once the patients have a good understanding of the subject matter, they can easily strive to manage the health condition in order to remain healthy. The patient could be more compliant when they have the knowledge of disease (Tignanelli, Wiktor, Vatsaas, Sachdev, Heung, Park, & Napolitano, L. M, 2018).

Implications for Nursing Care

The successful treatment of Acute Kidney Injury requires early recognition and intervention of the disease process (Koza, Y, 2016).

The clinicians must communicate effectively with the patients to make sure necessary protocols are being followed when caring for the patient with AKI. Some of the rules the clinicians have to strictly enforce when a case of AKI is being suspected will be to keep strict records of volume intake and output, the labs values such as creatinine also needs to be monitor. Clinicians and patients working together to correct the actual cause of AKI is the ultimate goal in achieving success (Koza, Y, 2016).

Conclusion

Acute Kidney Injury remains a difficult problem within clinical setting. The recent developments in trying to define and understand pathophysiology of Acute Kidney Injury was mentioned to have helped clinicians with their diagnosis and treatment of the disease process (Basile, D. P., Anderson, M. D., & Sutton, T. A., 2012).

It is very encouraging that there are ongoing efforts in tackling the disease process. Based on all the research articles, clinicians good understanding of the disease process will ensure that patients also receives the best level of care (Koza, Y, 2016).

References

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