How to Prevent Acute Kidney Injury in the Perioperative Period

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Introduction

Acute Kidney Injury (AKI) is a frequently encountered complication in surgical patients leading to significant morbidity and even mortality. It is estimated that approximately 40% of AKI in hospitalized patients occur in peri-operative period and is multi-factorial in origin. Anesthesiologists have an important role to play in peri-operative renal protection.

Definition

AKI is the disease in kidney function resulting in either an absolute increase in serum Creatinine of 0.3 mg/dl or more within 48 hours, an increase in serum Creatinine of 1.5 times from baseline within a period of 7 days or a urine output of 0.5 ml/kg/hr or less for > 6 hours.

Patients at risk for peri-operative AKI

Independent risk factors for AKI reported in studies are advancing age, body mass index (BMI) > 32, preexisting renal dysfunction, DM, decreased serum albumin, decreased cardiac performance, cardio-pulmonary bypass procedure, malignancy and high-risk surgery, hospital stay >48 hours, emergency surgery, peripheral vascular disease, liver disease and COPD. The most common causes for AKI in perioperative period are sepsis, cardiogenic and hypovolemic shock and drug induced AKI [1].

Surgery associated with high risk of developing AKI

Cardiac, vascular, major abdominal, trauma & orthopedic and transplant surgery patients have much higher risk of developing AKI due to high vulnerability associated with comorbidities and surgical risk. In addition, prolonged abdominal laparoscopic surgery can lead to AKI due to decreased renal blood flow and reduced renal function owing to pneumo-peritoneum.

Pathogenesis of peri-operative AKI

Most peri-operative AKI are caused by either pre-renal azotemia (secondary to renal hypo-perfusion) or Acute Tubular Necrosis (ATN) due to ischemic and/or nephrotoxic insults.

Strategies to prevent peri-operative AKI

One of the main objectives for peri-operative renal protection is prevention.

Volume status

 Maintenance of adequate intra-vascular volume is crucial and invasive monitoring can be helpful. Hemodynamic parameters like achieving mean arterial pressure (MAP) > 65-70 mm Hg, CVP 10-15 mm Hg, PAWP of 10-15 mm Hg, PAWP of 10-15 mm Hg with urine output > 0.5 cc/kg/hr guide adequate fluid therapy. Lactated ringer solution result in less acidosis and hyperkalemia than normal saline[2].

Renal perfusion

 Decreased renal perfusion can be due to decreased cardiac output, decreased MAP or increased renal vaso-constriction. Hypotensive anesthesia should be avoided in patient with risk of AKI.

Peri-operative nephrotoxins

Avoidance/ minimal peri-operative exposure to nephrotoxins such as antibiotics like aminoglycosides, cephalosporins, contrast agents, NSAIDS and chemotherapy agents.

Mannitol

An osmotic diuretic used to increase the intravascular volume with flushing of debris from renal tubules. Current literature does not support its routine use however its use in transplant surgery and crush injury patients is well established for preservation of renal function.

Furosemide

Being a loop diuretic, it increases renal blood flow, urine flow through tubules thus preventing tubular obstruction and decreased tubular O2 consumption[3].

Other drugs

A meta-analysis has shown that patient who received ACE inhibitors or ARB had lower incidence of AKI. Similarly, Statins
by virtue of their anti-inflammatory, anti-oxidant and endothelial protection effect leads to decreased AKI. Maintenance of adequate serum glucose levels (110-150mg/dl) is also required for prevention of AKI.

**Conclusion**

Approximately one out of 3 cases of AKI in hospital occurs in surgical patients with associated risk of morbidity and mortality. Identifying patients at risk of AKI based on pre-existing renal conditions and type of surgery is important. Peri-operative renal protection is also a responsibility of anesthesiologists and includes optimization of hemodynamics, intravascular volume and cautious use of nephrotoxins.

**References**