Editorial **ISSN: 2456-7388**

Preventing Perioperative Acute Kidney Injury

Pallavi Ahluwalia[1]

[1]Professor, Department of Anaesthesia, Teerthankar Mahaveer Medical College, TMU, Moradabad.

Peri-operative acute kidney injury (AKI) is a feared consequence of surgery and is associated with considerable morbidity and mortality. It has both short and long term deleterious effects. The term AKI is used to describe a rapid deterioration (hours to days) of renal function. The patho-physiology of peri-operative AKI is complex and involves both ischemia and inflammation as causative factors. Recently, several definition systems for AKI were proposed, incorporating both small changes of serum creatinine and urinary output reduction as diagnostic criteria. Urine output does not predict postoperative AKI.[1,2] Despite the continued effort to standardize the definition of AKI using S.Cr, these methods have several flaws. The increase In S.Cr is late in the course of AKI such that by the time the diagnosis is made using standard laboratory methods the disease is well established. In addition, S.Cr can be influenced by volume overload, nutrition, steroids, and muscle trauma.

Novel biomarkers are under investigation as fast and accurate predictors of AKI. For example, N-acetyl-β-d glucosamininidase in the urine is a direct marker of lysosomal injury in the proximal tubule, whereas cystatin C in the urine is a marker of reduced uptake by damaged proximal tubules.[2] Some other promising biomarkers are kidney injury molecule-1, microalbumin, neutrophyl gelatinase-associated lipocalin, interleukin 18, and liver fatty acid binding protein.[3] A promising study conducted in intensive care units (ICUs) showed that the combined use of the two novel biomarkers insulin-like growth factor binding protein 7 and tissue inhibitor of metalloproteinases- 2 was a sensitive and fast way to detect AKI. A similar study showed that insulin-like growth factor binding protein 7 and tissue inhibitor of metalloproteinases-2 can also be used for the detection of post-cardiac surgery AKI.[4]

Thus, these biomarkers can prove beneficial in early intervention to prevent further deterioration in renal function.

Several special considerations regarding the risk of AKI are of note in the surgical patient. Co-morbidities are important risk factors for AKI. The surgery in itself, especially emergency and major surgery in the critically ill, is associated with a high incidence of AKI. Certain types of surgeries, such as cardiac and transplantation surgeries, require special attention because they carry higher risk of AKI.

Nephrotoxic drugs, contrast dye, and diuretics are commonly used in the perioperative period and are responsible for a significant amount of in-hospital AKI. Before surgery, the anaesthetist is required to identify patients at risk of AKI, optimize anaemia, and treat hypovolaemia. During surgery, normovolaemia is of foremost importance. Also, the surgical and anaesthesia team is advised to use measures to reduce blood loss and avoid unnecessary blood transfusion. Hypotension should be avoided because even short periods of mean arterial pressure <55–60mmHg carry a risk of postoperative AKI. Higher blood pressures are probably required for hypertensive patients. Evidence so far do not recommend the use of one vasopressor over the other.[5] Low dose dopamine is no longer considered "renoprotective" and is not recommended.[5]

Avoid the use of aminoglycosides unless no suitable less nephrotoxic alternative exists.

Urine output can be reduced significantly during surgery and is unrelated to perioperative renal function. Thus, fluids should not be given in excess for the sole purpose of avoiding or treating oliguria. Use of hydroxyethyl starch needs to be reconsidered. Several recent studies report an increase in incidence of AKI and renal replacement therapy in critically ill patients infused with HES rather than crystalloids. Recent evidence indicates a beneficial effect of administering low-chloride solutions. Balanced crystalloid solutions may prove superior to chloride rich solutions in preventing AKI.

Key points to be kept in mind during fluid management are- The use of intraoperative urinary output as a guide to fluid administration may not be beneficial. Avoid the use of diuretics unless a need to treat volume overload arises. Use measures during surgery to avoid blood loss and unnecessary PRBC transfusion.

Address for correspondence:
Dr. Pallavi Ahluwalia
Professor, Department of Anaesthesia,
Teerthankar Mahaveer Medical College, TMU,
Moradabad.
Email Id: drpallaviahluwalia@yahoo.com

References

1. Hahn RG. Volume kinetics for infusion fluids. Anesthesiology 2010; 113: 470–81.
2. Norberg A, Hahn RG, Li H, et al. Population volume kinetics predicts retention of 0.9% saline infused in awake and isoflurane- anesthetized volunteers. Anesthesiology 2007; 107: 24–32.
3. Charlton JR, Portilla D, Okusa MD. A basic science view of acute kidney injury biomarkers. Nephrol Dial Transplant 2014; 29: 1301–11.
4. Meersch M, Schmidt C, Van AkenH, et al. Urinary TIMP-2 and IGFBP7 as early biomarkers of acute

kidney injury and renal recovery following cardiac surgery. PLoS ONE 2014; 9: e93460.
5. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney InjuryWork Group: KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012; 2: 1–138.

How to cite this article: Ahluwalia P. Preventing Perioperative Acute Kidney Injury. Acad. Anesthesiol. Int. 2017;2(1):1-2.

Source of Support: Nil, **Conflict of Interest:** None declared.