CRASH 2015 – Critical Care Symposium

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Perioperative acute kidney injury (AKI) - An update

Learning Objectives:

1) AKI and risk of death – does it matter?
2) Overview of current terminology: Acute Renal Failure, pre-renal azotemia, Acute Tubular Necrosis, Post-renal Failure, Acute Kidney Injury
3) New biomarkers for AKI, limitations and future indications
4) Fluid therapy for AKI
5) "Do no harm" How to protect kidney function from iatrogenic injury
6) Management of renal replacement therapy patients in the operating room

Perioperative Organ Injury is common after surgery and has been likened to the #3 cause of death in the United States.[1] AKI is the most common form of perioperative organ injury and occues in up to 1% of general surgery patients[2] and 11% of cardiac surgery patients.[3] AKI is associated with an increased risk of death.

Contemporary definitions for AKI by AKIN and KDIGO include urine output as well as rise in serum creatinine as relevant measures for AKI staging. An increase of as little as 0.3 mg/dl in serum creatinine defines stage I AKI.[4] [5]

Acute tubular necrosis (ATN) is a histologic diagnosis. ATN is thought to occur after significant cellular ischemia, for example in the setting of hypovolemia. However, other mechanisms such as occur in sepsis make the diagnosis pre-renal azotemia equally useless. The development of cellular damage occurs on a biologic continuum, making it impossible to define a point were pre-renal azothemia ends and ATN begins. Volume therapy should be guided by volume status and not by a questionable determination towards ATN vs. prerenal azothemia. Postrenal obstruction can be evaluated using bedside ultrasound. [6]

Many animal models use a temporary occlusion of the renal blood vessels to induce AKI. This is not a sufficient model to emulate sepsis, the most common cause of AKI. This may be a reason for the lack of translation of renoprotective substances into successful clinical trials. Decreased perfusion pressure, increased renal vascular resistance, hypoxia, ischemia / reperfusion, RAAS activation, sepsis, bacterial toxins, contrast agents, and nephrotoxic medications are clinically relevant mediators of AKI.

Fractional excretion of sodium is not reliable to aid in AKI therapy. [7]

New biomarkers such as urine TIMP2 and IGFBP7 permit early diagnosis of impending AKI. Wheather interventions will translate to improved outcomes is not yet proven. [8]

Balanced crystalloid solutions such as Plasmalyte of Lactated Ringers are preferred over Normal Saline in AKI patients.[9,10]

Hydroxyethylstarches (HES) should be avoided.[11,12]

Pharmacotherapy for AKI (e.g. dopamine, fenoldopam) has failed.[13-15]

Renal replacement therapy is instituted for:

- Acidosis
- Electrolyte abnormality (hyperkalemia)
- Hypervolemia
- Uremia
- Certain intoxications

Patients requiring surgery while on RRT should be assessed with a focus on:

- Control of the initial indication for RRT (e.g. correction of hyperkalemia)
- Review volume and electrolyte status
- Decide if patient's RRT may be discontinued for duration of procedure
- Avoid large volume and electrolyte shifts
- Use meticulous sterile technique
- Check electrolytes frequently
- Be prepared for medical management of hyperkalemia

Summary

- AKI is common after surgery and associated with increased morbidity and mortality
- HES should no longer be used
- Balanced cristalloid solutions appear more desirable than NS
- Pharmacologic therapy of AKI has failed
- For patients on RRT presenting for surgery knowledge of the indication for RRT is the basis for risk stratification

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