From the Desk of Editor in Chief

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When and what to give or avoid in AKI management

Timed and targeted therapy for Acute Kidney Injury (AKI) involves identifying high-risk patients early through biomarkers[1] and implementing precise interventions before functional decline, such as managing fluid balance, avoiding nephrotoxins, optimizing mean arterial pressure (>65 mm of Hg), and using renal replacement therapy (RRT) when indicated.[2] Differentiating between pre-renal, renal or post renal (which can easily be done with a good clinical history and some basic tests), can guide towards more targeted guidelines. While no specific drug directly reverses AKI, early intervention focusing on underlying causes improves outcomes.

For early identification; in cases where risk of injury is known some urinary and serum biomarkers can be utilized to identify damage before Serum Creatinine rises (e.g., NGAL, KIM-1, Cystatin C, NAG, TIMP-2, IGFBP7).

Whereas, targeted interventions are aimed to support hemodynamics like maintaining mean arterial pressure (MAP) >65 mm Hg, using intra venous fluids or vasopressors (e.g., norepinephrine) to ensure renal perfusion. Avoiding both hypovolemia (which may add pre-renal element to existing injury) and fluid overload which increases morbidity and mortality is key to fluid management.
Nephrotoxins stewardship also very important pillar in AKI management; discontinuation of NSAIDs, ACE inhibitors, ARBs, PPI and aminoglycosides.

While immediate renal replacement therapy (RRT) is necessary for life-threatening complications (e.g., refractory hyperkalemia, acidosis, pulmonary edema), evidence on "very early" initiation (before metabolic crisis) is mixed, with some studies showing no mortality benefit compared to a more cautious, patient-individualized approach.[3]

There is no specific drug which has shown consistent, widespread efficacy to prevent AKI, research continues into agents targeting specific mechanisms, such as Alkaline Phosphatase for sepsis-associated AKI, that works by detoxifying endotoxins and pro-inflammatory compounds (e.g., ATP) into protective adenosine, thereby reducing renal inflammation and improving kidney function.[4]

Thus the management goals should be preservation with optimizing renal function and prevent progression. To secure homeostasis by maintaining fluid, electrolyte, and acid-base balance and to prevent secondary organ damage.

Effective communication with patient and their families by providing clear explanation of AKI, its cause in their patient, proposed treatment plan and prognosis is also job of nephrologist.

References:

1. Naqvi R, Hossain N, Butt S, Bhellar Z, Fatima E, Imtiaz S, et al. Efficacy of multiple Biomarkers: NGAL, KIM1, Cystatin C and IL18 in predicting pregnancy related acute kidney injury. Pak J Med Sci. 2023;39(1):34-40. doi.org/10.12669/pjms.39.1.6930

2. Mehta RL. Timed and targeted therapy for acute kidney injury: a glimpse of the future. Kidney Int. 2010 Jun; 77(11):947 – 9.
3. Smith OM, Wald R, Adhikari NKJ, Pope K , Weir MA, Bagshaw SM on behalf of the Canadian Critical Care Trials Group. Standard versus accelerated initiation of renal replacement therapy in acute kidney injury (STARRT-AKI): study protocol for a randomized controlled trial. BioMed central. 2013, 14:320. http://www.trialsjournal.com/content/14/1/320
4. Steenvoorden TS, Rood JAJ, Bemelman FJ, Armstrong Jr. R, Leuvenink HGD, van der Heijden JW, Vog L. Alkaline phosphatase treatment of acute kidney injury—an update. Nephrol Dial Trans.2024, 39:1239 – 47