Global Action. Local Change.

KDIGO Controversies Conference on Acute Kidney Injury
- Breakout Group Questions -

Breakout Group 1: Nomenclature and Diagnostic Criteria

1. Is sufficient evidence now available to warrant a change in the definitions/classification/staging system for AKI?
    a. AKI/AKD/CKD continuum
    b. Definition of subclinical AKI / kidney stress
    c. Persistence (vs transient, vs relapsing?)
    d. How to define patients with falling creatinine
    e. Role of etiology/setting
    f. Urine output vs Creatinine criteria
    g. Role of biomarkers, biopsy, and imaging
    h. Fluid imbalance as indicated of decreased function
    i. Community vs. hospital-acquired (manifest?)
    j. Children vs adults

2. How should existing (or new) definitions of AKI be implemented at the bedside, in research?
    a. How should baseline kidney function be determined? (baseline creatinine, eGFR, renal reserve, imaging or combinations)
    b. How should urine output be evaluated (body composition? fluid balance? setting/context?)
    c. What is the role for real time or kinetic GFR?
    d. How and how often should kidney function / injury / stress be monitored?
    e. Role of expert interpretation/judgment

3. What is the best way to define renal recovery?
    a. AKI vs AKD?
    b. Complete vs partial
    c. How to define recovery of subclinical AKI: Renal reserve? Biomarkers? Fluid balance?

Breakout Group 2: AKI Risk Stratification and Assessment

1. What are the roles for risk-stratification of patients for AKI?
    a. Which patients? When?
    b. Clinical models
    c. Biomarkers
    d. Renal angina index (RAI)
    e. Renal functional reserve
    f. Applications to mitigate risk

2. Once AKI is diagnosed, what is the approach to determining the cause and prognosis?
    a. Evaluation and monitoring
    b. Endpoints/expected clinical course
    c. Clinical models
    d. Role of biomarkers, biopsy, and imaging
    e. Urine creatinine excretion, urine chemistries, sediment
    f. Furosemide stress test
    g. Renal functional reserve
    h. Framework for AKI classification (to replace pre-renal/intra-renal)?

3. How should patients be followed after AKI?
    a. Which patients should be followed up?
    b. Which parameters should be monitored?
    c. Short-term vs long-term
    d. Role for biomarkers, renal functional reserve

Breakout Group 3: Fluid Management and Hemodynamic Support

1. What are the indications for fluid administration in patients with or at risk for AKI?
    a. How should patients be selected for fluid therapy?
    b. What are the targets/endpoints/contraindications to fluid administration?
    c. Do differing clinical contexts & etiologies of AKI affect fluid management?
    d. Does the timing (early vs established) of AKI alter management?
    e. How to balance fluid management and vasopressor treatment?

2. How should fluids be given in patients at risk for/with AKI?
    a. How much fluid constitutes a fluid challenge/ initial fluid bolus in the setting of various stages of kidney dysfunction?
    b. Bolus therapy vs continuous infusions
    c. How should response to fluid be evaluated and monitored?

3. How does the composition of various intravenous fluid preparations affect kidney function or influence outcomes in patients with AKI?
    a. Electrolytes
    b. Tonicity, colloids/crystalloids
    c. Non-hemodynamic (drug-like) effects of fluids (on the kidney)

4. What are the indications/contraindications for fluid removal in patients with or at risk for AKI and how should fluid removal be affected in such patients?
    a. To what extent is fluid overload causative of organ injury including persistent kidney dysfunction compared to being merely a marker of illness severity?
    b. What are the risks of fluid removal?
    c. In what circumstances are extracorporeal therapies preferred over diuretics?
    d. How do we determine rate of fluid removal (tolerability) and eventual goal for fluid removal (quantification of fluid overload)?

Breakout Group 4: Nephrotoxic Agents and Drugs that Affect Kidney Function

1. In light of current evidence what can be recommended for the prevention and management of contrast-associated AKI?
    a. Does contrast-associated AKI exist? Relative impact?
    b. Prevention strategies (i.e., PRESERVE trial)
    c. Management

2. Is there sufficient evidence to classify drugs that affect kidney function and/or are nephrotoxic in a clinically useful way?
    a. Criteria for classification? (just association? change in function vs injury? Role of imaging? Role of biomarkers?)

   b. Variation of toxicity depending on susceptibility (age, clinical context, other medications?)
   c. Drug combinations: Impact of nephrotoxic burden; which combinations contribute to greater risk of AKI?
   d. Differentiation between essential and non-essential nephrotoxic drugs
   e. Role of ACE-I / ARBs in AKI
   f. Consequences of drug-associated AKI vs other causes? (degree of harm, reversibility)
   g. Contribution of nephrotoxins to other causes?

3. What prevention strategies should be applied for drug-associated AKI?
   a. Hypervigilance, early detection
   b. Clinical management (including fluids)
   c. Role of biomarkers
   d. Drug stewardship
   e. Computer decision tools

4. How should potentially nephrotoxic drugs be managed during AKI (including during RRT) and following an episode of AKI?
   a. Recommended patient-care plan for patients with drug-induced AKI (including prevention of progression of AKI)
   b. (Re-)initiation of medication that is potentially nephrotoxic following an episode of AKI? (chronic medications vs new medications)

Breakout Group 5: Renal Replacement Therapy

1. What criteria should be used to initiate RRT in patients with AKI?
   a. Severity/duration
   b. Biomarkers
   c. Demand/capacity balance
   d. Risk for complications
   e. Potential for recovery (note some overlap with Group 1)
   f. ICU vs outside the ICU
   g. Resource-limited environments
   h. Non-renal factors (e.g., sepsis, heart disease)

2. What is the optimal strategy (including modality) for providing acute RRT?
    a. Patient factors (hemodynamics, fluid balance, brain injury/edema)
    b. Etiology of AKI
    c. Clinical setting (ICU vs outside the ICU; resource-limited environment)
    d. Dose (including over time)
    e. Anticoagulation
    f. Nutrition management
    g. Monitoring
    h. Discontinuation and modality transition

3. How should RRT be combined with extracorporeal respiratory and cardiac support (e.g., ECMO, $ECCO_2R$)?
    a. Indications
    b. Technique and monitoring

4. How do RRT decisions affect long-term outcomes and how should patients be followed?
    a. Effects on renal recovery
    b. Approach to follow-up and timing

5. Is the terminology 'RRT' sufficiently accurate, descriptive, and patient-centered?