Acute Kidney Injury (AKI)
And
Deep Venous Thrombosis (DVT)

DR. Akbar
Definition of Acute Kidney Injury (AKI) based on “Acute Kidney Injury Network”

<table>
<thead>
<tr>
<th>Stage</th>
<th>Increase in Serum Creatinine</th>
<th>Urine Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.5-2 times baseline OR 0.3 mg/dl increase from baseline</td>
<td>&lt;0.5 ml/kg/h for &gt;6 h</td>
</tr>
<tr>
<td>2</td>
<td>2-3 times baseline</td>
<td>&lt;0.5 ml/kg/h for &gt;12 h</td>
</tr>
<tr>
<td>3</td>
<td>3 times baseline OR 0.5 mg/dl increase if baseline&gt;4mg/dl OR Any RRT given</td>
<td>&lt;0.3 ml/kg/h for &gt;24 h OR Anuria for &gt;12 h</td>
</tr>
</tbody>
</table>
Epidemiology

AKI occurs in

- ≈ 7% of hospitalized patients.
- 36 – 67% of critically ill patients (depending on the definition).
- 5-6% of ICU patients with AKI require RRT.
Mortality in AKI

- Mortality increases proportionately with increasing severity of AKI

- AKI requiring RRT is an independent risk factor for in-hospital mortality.

- Mortality in patients with AKI requiring RRT 50-70%.

- Even small changes in serum creatinine are associated with increased mortality.
Acute Kidney Injury

AKI

PRERENAL

INTRINSIC

POSTRENAL
Acute Kidney Injury

- **PRERENAL: 40-80%**
  - Volume loss/Sequestration
  - Impaired Cardiac Output
  - Hypotension (and potentially hypo-oncotic states)

- **Net result:** glomerular hypoperfusion
Acute Kidney Injury

• RENAL/INTRINSIC: 10-30%
  – Vascular disorders:
    – small vessel
    – large vessel
  – Glomerulonephritis
  – Interstitial disorders:
    – Inflammation
    – intercalative processes
  – Tubular necrosis:
    – Ischemia
    – Toxin
    – Pigmenturia
Acute Kidney Injury

• POSTRENAL: 5-15%
  – Intrarenal
    – Crystals
    – Proteins
  – Extrarenal
    – Pelvis/Ureter
    – Bladder/Urethra
Acute Kidney Injury

- Prerenal and ATN encountered most often in the hospital setting: 70-75% in many studies
- Most common diagnostic consideration is therefore between these two conditions
- Prerenal:
  1. Intravascular volume depletion
  2. Hypotension
  3. Edematous states
  4. Localized renal ischemia
- ATN:
  1. All causes for prerenal, leading to post-ischemic ATN
  2. Toxins
Increase in Creatinine without AKI (false positive)

• **Inhibition of tubular creatinine secretion**
  Trimethoprim, Cimetidine, Probenecid

• **Interference with creatinine assays in the lab (false elevation)**
  acetoacetate, ascorbic acid, cefoxitin
  flucytosine
Increase in BUN without AKI

- **Increased production**
  - GI Bleeding
  - Catabolic states (Prolonged ICU stay)
  - Corticosteroids
  - Protein loads (TPN-Albumin infusion)
AKI: Diagnostic studies-urine

- Urinalysis for sediment, casts
- Response to volume repletion with return to baseline SCr 24-72 hr c/w prerenal event
- Urine Na; FENa

\[
\text{FENa} (\%) = \frac{\text{UNa} \times \text{SCr}}{\text{SNa} \times \text{UCr}} \times 100
\]

- FENa < 1%: Prerenal
- FENa 1-2%: Mixed
- FENa > 2%: ATN
Urinalysis in Acute Kidney Injury

Normal/bland

Abnormal sediment

Hematuria
- RBC casts
- Proteinuria

WBC casts

Eosinophils

RTE cells
- Pigmented casts

Crystalluria

Non-albumin proteinuria

Prerenal
- Postrenal
- Oncotic AKI

Glomerulopathy
- Vasculitis
- Thrombotic MA

Pyelonephritis
- Interstitial nephritis

AIN
- Athero-embolic AKI

ATN
- Myoglobin
- Hemoglobin

Uric acid
- Toxins
- Drugs

Plasma cell dyscrasia

Abnormal sediment

Normal/bland

Hematuria
- RBC casts
- Proteinuria

WBC casts

Eosinophils

RTE cells
- Pigmented casts

Crystalluria

Non-albumin proteinuria

Prerenal
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Plasma cell dyscrasia
Acute Kidney Injury

LABORATORY DATA

- Creatinine; also BUN/Cr ratio
- CBC: anemia, thrombocytopenia
- HCO$_3^-$: anion gap, lactic acid, ketones
- K
- CPK/LDH/Uric acid/liver panel
- Serologies:
  - Complement
  - ESR, RF, ANA, ANCA, AntiGBM
  - Electrophoresis
- Toxicology studies
Acute Kidney Injury

IMAGING STUDIES

• Ultrasound: evaluates renal size, able to detect masses, obstruction, stones
• CT: detects masses, stones; caveat exists when IVCD is considered
• MRI/MRA: can detect RAS; use of Gadolinium carries uncertain R/B ratio in AKI 2° potential hemodynamic changes similar to IVCD, and NFD

In the AKI setting, U/S provides most...
Prevention of AKI in ICU

• Recognition of underlying risk factors
  – Diabetes
  – CKD
  – Age
  – HTN
  – Cardiac/liver dysfunction
• Maintenance of renal perfusion
• Avoidance of hyperglycemia
• Avoidance of nephrotoxins

Management of AKI in ICU

- Treatment is largely supportive in nature
  Maintain renal perfusion
- Correct metabolic derangements
- Provide adequate nutrition
- Role of diuretics
- Renal Replacement therapy remains the cornerstone of management of minority of patients with severe AKI
Maintaining renal perfusion

- Human kidney has a compromised ability to autoregulate in AKI.
- Maintaining haemodynamic stability and avoiding volume depletion are a priority in AKI.

- The individual BP target depends on age, co-morbidities (HTN) and the current acute illness.
- A generally accepted target remains MAP ≥
Volume resuscitation – which fluid?

• no statistical difference between volume resuscitation with saline or albumin in survival rates or need for RRT.
Renal vasodilators?

renal dose dopamine (<5 μg/kg of body weight/min) increases RBF and, to a lesser extent, GFR. Dopamine is unable to prevent or alter the course of ischaemic or nephrotoxic AKI]. Furthermore, dopamine, even at low doses, can induce tachy-arrhythmia’s, myocardial ischaemia, and extravasation out of the vein can cause severe necrosis .Thus, the routine administration of dopamine to patients for the prevention of AKI or incipient AKI is no longer justified.
Is there a role for Fenoldopam in prevention or treatment of AKI in ICU setting?

- Dopamine-1 receptor agonist, lack of Dopamine-2, and alpha-1 receptor effect, make it a potentially safer drug than Dopamine!

- Reduces in hospital mortality and the need for RRT in AKI

- Reverses renal hypoperfusion more effectively than renal dose Dopamine

- So far so good specially in cardiothoracic ICU patients, awaiting more powered trials in other groups!
Is there a role for diuretics in the treatment of AKI in ICU setting?

• Loop diuretics may convert an oliguric into a non-oliguric form of AKI that may allow easier fluid and/or nutritional support of the patient. Volume overload in AKI patients is common and diuretics may provide symptomatic benefit in that situation. However, loop diuretics are neither associated with improved survival, nor with better recovery of renal function in AKI.
EPO

• Erythropoietin (EPO) has tissue-protective effects and prevents tissue damage during ischaemia and inflammation, and currently trials are performed with EPO in the prevention of AKI post-cardiac surgery, CIN and post-kidney transplantation.
Deep vein thrombosis
Deep Vein Thrombosis (DVT)

- Normal
- DVT

- Blood flow
- Detached blood clot
- Valve
- Blood clots
Deep Vein Thrombosis

- 80% proximal thrombosis
- 20% calf vein thrombosis
- 50% progress to PE
- Cancer
- Estrogen use
- Family history
- IBD
- Nephrotic syndrome
- Blood transfusions
- Thrombophilia

- Surgery
- Prior DVT
- Central venous access
- Cancer chemotherapy or radiotherapy

- Age >40 years
- Immobilization
- MI
- CHF
- Paralysis
<table>
<thead>
<tr>
<th>Patient Group</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical patients</td>
<td>10–20</td>
</tr>
<tr>
<td>General surgery</td>
<td>15–40</td>
</tr>
<tr>
<td>Major gynecologic surgery</td>
<td>15–40</td>
</tr>
<tr>
<td>Major urologic surgery</td>
<td>15–40</td>
</tr>
<tr>
<td>Neurosurgery</td>
<td>15–40</td>
</tr>
<tr>
<td>Stroke</td>
<td>20–50</td>
</tr>
<tr>
<td>Hip or knee arthroplasty, hip fracture surgery</td>
<td>40–60</td>
</tr>
<tr>
<td>Major trauma</td>
<td>40–80</td>
</tr>
<tr>
<td>Spinal cord injury</td>
<td>60–80</td>
</tr>
<tr>
<td>Critical care patients</td>
<td>10–80</td>
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Geerts et al. Chest, 2004; 126:338S
Signs and symptoms

- Discoloration of the legs
- Calf or leg pain or tenderness
- Swelling of the leg or lower limb
- Warm skin
- Surface veins become more visible
- Leg fatigue
Importance of DVT diagnosis

- If left untreated, can suffer fatal PE
- Treatment of proximal DVT reduces the risk of fatal PE to <1% and the risk of developing recurrent DVTs to 5%
PE SXS/ Signs (PIOPED II)

- Dyspnea (79%)
- Tachypnea (57%)
- Pleuritic pain (47%)
- Leg edema, erythema, tenderness, palpable cord (47%)
- Cough/ hemoptysis (43%)
Homan's Sign

Forcible ankle dorsiflexion with the knee flexed causing deep calf pain.
- Malignancy
  +1
- Lower limb paralysis/recent plaster cast
  +1
- Bedridden for >3 days/surgery (<4 weeks)
  +1
- Localized tenderness along deep veins
  +1
- Entire leg swollen
  +1
- Calf swelling >3 cm asymptotically
Wells criteria

• > 75%: High
• 17%: Moderate
• < 3%: Low
D-dimer test

Increased in:

- Arterial and venous thrombosis
- Major surgery
- Hemorrhage
- Trauma
- Pregnancy
- Cancer
• Venography: Gold standard

• Ultrasonography:
venography
Management

• Medical management

• Surgical management
Medical management

• Compressional stockings
• Heparin
• Warfarin
• Vitamin K antagonist
Mechanisms of Elastic Compression Stockings

- ↓ Venous dilatation
- ↑ Flow velocity
- Improve valve function
- ↑ Tissue Factor Pathway Inhibitor
Duration of Anticoagulation for DVT Event

<table>
<thead>
<tr>
<th>Event</th>
<th>Duration</th>
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<tbody>
<tr>
<td>First Time event of Reversible cause (surgery/trauma)</td>
<td>At least 3 mos</td>
</tr>
<tr>
<td>First episode of idiopathic VTE</td>
<td>At least 6 mos</td>
</tr>
<tr>
<td>Recurrent idiopathic VTE or continuing risk factor (e.g., thrombophilia, cancer)</td>
<td>At least 12 mos</td>
</tr>
<tr>
<td>Symptomatic isolated calf-vein thrombosis</td>
<td>6 to 12 weeks</td>
</tr>
</tbody>
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*From American College of Chest Physicians*
<table>
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<th>Risk group</th>
<th></th>
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<tbody>
<tr>
<td>Hip replacement</td>
<td>Fondaparinux</td>
</tr>
<tr>
<td>Knee replacement</td>
<td></td>
</tr>
<tr>
<td>Hip Fracture</td>
<td>Fondaparinux</td>
</tr>
<tr>
<td>Major trauma</td>
<td></td>
</tr>
<tr>
<td>Abdominal surgery</td>
<td>UFH, LMWH, IPC, warfarin, ES</td>
</tr>
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</table>
Effective prophylaxis

- Subcutaneous heparin – 5000 IU Q8H for 5 days
- Aspirin 150 mg started preoperatively and continued till 45 days
- Graduated elastic compression stockings
- Intermittent pneumatic compression
- Mechanical foot pumps
Surgical management

• IVC filter
• Embolectomy
• Thrombolysis
Indication

• Pulmonary embolism with contraindication to anticoagulation
• Recurrent pulmonary embolism despite adequate anticoagulation.
Embolectomy

• Indication:
  – In those where thrombolysis is contraindicated or fails
Thrombolysis

• Considered once P.E. diagnosed

• If chosen, hold anticoagulation during thrombolysis infusion, then resumed

• Associated with higher incidence of major hemorrhage
Thank you