Acute Oliguria

Chavasak kanokkantapong
Oliguria and Anuria

• Oliguria
  - Decreasing in urine volume less than that required for excretion of normal daily metabolic function.
  - In adult < 400 ml/day.

• Anuria
  - Absence of urine output or < 50 ml/day
  - Indicate obstruction of urinary tract.
  - Rarely from renal infarction or renal cortical necrosis.
Acute Oliguria

• Definition: < 400 ml of urine / day
• The most earliest sign of ARF.
• Identify reversible causes leading to decreasing in high morbidity and mortality of ARF.
• Complication of acute oliguria
Complication of ARF
<table>
<thead>
<tr>
<th>Approximate Serum [K⁺] (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
</tr>
<tr>
<td>B</td>
</tr>
<tr>
<td>C</td>
</tr>
<tr>
<td>D</td>
</tr>
<tr>
<td>E</td>
</tr>
</tbody>
</table>

#### Note

- A: Normal range
- B: Mild hyperkalemia
- C: Moderate hyperkalemia
- D: Severe hyperkalemia
- E: Very severe hyperkalemia
Inpatient Mortality - ARF

Dialysis
No Dialysis

JASN 17:1143, 2006

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Acute VS Chronic renal failure

• Anemia (Anemia of chronic disease)
• History of chronic symptom
• Small size of both kidneys by ultrasonography
• “Board” cast from urine analysis
• Renal osteodystrophy(rare)
RIFLE Criteria

- Risk
- Injury
- Failure
- Loss
- End stage kidney disease
# RIFLE

<table>
<thead>
<tr>
<th>Criteria</th>
<th>sCr</th>
<th>UOP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk of renal injury</td>
<td>0.3 mg/dl increase</td>
<td>&lt; 0.5 ml/kg/hr for &gt; 6 h</td>
</tr>
<tr>
<td>Injury to the kidney</td>
<td>2 X baseline</td>
<td>&lt; 0.5 ml/kg/hr for &gt; 12h</td>
</tr>
<tr>
<td>Failure of kidney function</td>
<td>3 X baseline OR &lt;br&gt; &gt; 0.5 mg/dl increase if &lt;br&gt; $S_{Cr} &gt;= 4$ mg/dl</td>
<td>Anuria for &gt; 12 h</td>
</tr>
<tr>
<td>Loss of kidney function</td>
<td>Persistent renal failure for &gt; 4 weeks</td>
<td></td>
</tr>
<tr>
<td>End-stage disease</td>
<td>Persistent renal failure for &gt; 3 months</td>
<td></td>
</tr>
</tbody>
</table>

# Acute Kidney Injury Network Criteria

<table>
<thead>
<tr>
<th>Stage</th>
<th>Creatinine Criteria</th>
<th>UOP Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>↑SCr ≥ 0.3 mg/dL or ↑SCr ≥ 150-200%</td>
<td>&lt; 0.5 mL/kg/hr for &gt; 6 hr</td>
</tr>
<tr>
<td>2</td>
<td>↑SCr &gt; 200-300%</td>
<td>&lt; 0.5 mL/kg/hr for &gt;12 hr</td>
</tr>
<tr>
<td>3</td>
<td>SCr ≥ 354 µmol/L + acute ↑≥44 µmol/L in ≤24hr or RRT initiated</td>
<td>&lt; 0.3 mL/kg/hr for 24 hr or anuria for 12 hr</td>
</tr>
</tbody>
</table>
Acute renal failure

- Abrupt reduction in renal function
- Resulting in azotemia (Increasing of BUN)
- Usually classified according to
  - Pre-renal (Decrease renal blood flow)
  - Renal parenchyma (Intrinsic renal)
  - Post-renal (Obstruction)
Pre-renal
Decrease renal blood flow

Intrinsic renal

Post-renal
Obstruction

Decrease renal blood flow

Front View of Urinary Tract

Kidney

Ureter

Bladder

Sphincter

Intrinsic renal

Obstruction

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Pre-renal Failure

• Hypovolemia
  - hemorrhage, GI fluid loss, 3rd spacing, renal loses, trauma, surgery, burns

• Relative hypovolemia (effective volume)
  - sepsis, hepatic failure, anaphylaxis, vasodilator drugs, nephrotic syndrome, anesthetic agents
Pathophysiology of Pre-renal Azotemia

- Decrease in renal blood flow
- Compensatory renal afferent arteriolar vasoconstriction.
- Resulting in decrease of GFR.
- Increase reabsorption of sodium, water, and urea.

Urine Na < 20 mEq/L and FE Na < 1%
BUN/Cr ratio > 20:1
Urine osmolality > 500 mOsm/L
Normal urinalysis.
Pre-renal
Decrease renal blood flow

Intrinsic renal

Post-renal
Obstruction

Front View of Urinary Tract

Kidney

Ureter

Bladder

Sphincter

Urethra

Decrease renal blood flow

Intrinsic renal

Obstruction

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Intrinsic renal

- **Vasular**
  - TTH/HUS, Chol emboli, Malignant HT

- **Glomerular**
  - PSGN

- **Tubulo-interstitium**
  - ATN, AIN, Obstruction
Intrinsic-renal ARF

Acute tubular necrosis

• Ischemia
• Nephrotoxic ie

• Tubular necrosis leads to sloughing renal tubular cell into lumen.
• Obstruction urinary flow, contributing to reduction in GFR.
• Dysfunction of tubular cell leads to diluted urine, high urinary sodium, and isostenuria.
Intrinsic -renal ARF

• Acute glomerulonephritis
  - Proteinuria, hematuria, Rbc cast.

• Acute interstitial nephritis
  - Sterile pyuria, eosiophilia, eosiophiluria.

• Ateriolar injury
  - Accelerated hypertension, Vasculitis,
    Microangiopathic (thrombotic thrombocytopenic
    purpura, hemolytic-uremic syndrome)

• Cholesterol emboli
Pre-renal
Decrease renal blood flow

Intrinsic renal

Post-renal
Obstruction

Front View of Urinary Tract

Decrease renal blood flow

Intrinsic renal

Obstruction
Post-renal ARF

- Generally must involve in collecting system of both kidneys or in solitary kidneys.
- Obstruction should be considered in acute anuric patient, history of polyuria alternating with oliguria.

Prostate enlargement, Tumor, Stone, Urethral stricture, Massive crystal deposition (uric acid, acyclovia)
Acute Renal Failure

- Prerenal ARF
  - Acute tubular necrosis

- Intrinsic ARF
  - Acute interstitial nephritis
  - Acute GN

- Postrenal ARF
  - Acute vascular syndromes
  - Intratubular obstruction
Initial diagnostic tools in AKI

- History and Physical exam
- Detailed review of the chart, drugs administered, procedures done, hemodynamics during the procedures.
- Urinalysis
  - SG, PH, protein, blood, crystals, infection
- Urine microscopy
  - casts, cells (eosinophils), urine lytes
- Renal imaging US
- Markers of CKD
  - iPTH, size<9cm, anemia, high phosphate, low bicarb
- Renal biopsy
Diagnostic test

- Serum creatinine
  - typical increase 1-2 mg/dl/day
  - Increase >5mg/dl/day suggestive rhabdomyolysis
- BUN/Scr ratio (normal 10-20:1)
  - Increase > 20:1 suggestive dehydration(prerenal), upper gastrointestinal bleeding, obstructive uropathy(uncommon)
Increase in Creatinine without AKI

- Inhibition of tubular creatinine secretion
  Trimethoprim, Cimetidine, Probenecid

- Interference with creatinine assays in the lab (false elevation)
  cefoxitin, flucytosine
Increase in BUN without AKI

• Increased production
  GI Bleeding
  Catabolic states (Prolonged ICU stay)
  Corticosteroids
  Protein loads (TPN-Albumin infusion)
Urine analysis

• Pre-renal
  Normal, or hyaline casts

• Acute tubular necrosis
  Dirty brown, opaque, presence of tubular casts

• Acute glomerulonephritis
  Reddish brown urine, cola-colored urine, Rbc cast

• Interstitial nephritis
  Sterile pyuria, eosinophiluria
RBC cast (glomerulonephritis)

Muddy brown granular cast (ATN)

Waxy cast

WBC cast

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“Muddy” (Pigmented) Granular Casts
## Urine indices

<table>
<thead>
<tr>
<th></th>
<th>Pre-renal</th>
<th>ATN</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Urine sodium</strong> (U_{Na}) mEq/liter</td>
<td>&lt;20</td>
<td>&gt;40</td>
</tr>
<tr>
<td><strong>Urine osmolality</strong>, mosm/kg</td>
<td>&gt;500</td>
<td>&lt;350</td>
</tr>
<tr>
<td><strong>Fractional excretion of filtered sodium</strong>  (FE_{Na})</td>
<td>&lt;1 %</td>
<td>&gt;2%</td>
</tr>
</tbody>
</table>
\[
FE_{Na} = \frac{(U_{Na} \times P_{Cr} \times 100)}{(P_{na} \times U_{cr})}
\]

*Kidney Int 2002;62:2223*
Cautions of Urinary Indexes

• Samples must be collected before the use of fluid replacement, dopamine, mannitol.

• Urine must not contain glucose or contrast material

• FENA < 1% in myoglobinuria or CIN
New Biomarkers in AKI
Alternatives to Serum Creatinine

• Urinary Neutrophil Gelatinase-Associated Lipocalin (NGAL)
• Urinary Interleukin 18
• Urinary Kidney Injury Molecule 1 (KIM-1)
Radiographic studies

• Renal ultrasonography
• Plain film of abdomen
• Excretory urography (IVP)
• Renal angiography, Renal scan
• Urologic studies
• Renal biopsy
Ultrasound in Obstructive uropathy
Complications of acute oliguria

- **Cardiovascular**
  - pulmonary edema, HT, arrhythmia, pericarditis

- **Infections**
  - 30-70% of patients
  - leading of mortality
  - respiratory and UTI

- **Neurologic**
  - confusion, asterixis, seizures

- **Gastrointestinal**
  - N/V
  - GI bleed (10-30%)
  - Anemia (GI bleed, frequent blood)
Treatment of AKI

- Treatment is largely supportive in nature.
- Pharmacologic treatments under study:
  - Dopamine: no benefit
  - Atrial Natriuretic Peptide (ANP) or ANP-analogue (Anaritide): promising
  - Human Insulin like growth factor 1: no benefit
- Renal Replacement therapy remains the cornerstone of management of minority of patients with severe AKI

*Nephron Clin Pract 2009;112:c222-c229*
Supportive treatment

- Fluid management
- Dietary modification
- Blood pressure control
- Metabolic control
- Drug dosages
- Infection
- GI bleeding
- Anemia
Fluid management of Pre renal

• Challenge

In oliguric patients who are not volume overloaded. Infusion of 1 to 2 L of normal saline intravenous over 2 to 4-hour with close monitoring of vital signs, physical examination, and urine output.
Fluid Management for ATN

Fluid replacement should be equal to insensible loss (about 500 ml/day in afebrile patients) plus urinary and other drainage losses.
Dietary modification

- protein catabolism can be substantial (200-250 g per day) in patients with ARF
- a negative nitrogen balance may lead to malnutrition, impaired immune function, and increased risk of morbidity / mortality
- Salt < 2-4 g/day NaCl
- Potassium < 40 mEq/day
- Magnesium-containing compounds should be avoided.

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III. Blood pressure control
IV. Metabolic control
   Phosphate restriction, Phosphate binder
   Metabolic acidosis
   Hyperkalemia
V. Drug dosages
VI. Infection
VII. GI bleeding
VIII. Anemia
Dialysis

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Indication for acute dialysis

A = Severe metabolic acidosis
E = Severe electrolyte imbalance (Hyperkalemia)
I = Intoxicatio
O = Volume overload
U = Severe uremia (uremic encephalopathy, Platelet dysfunction, uremic pericaditis)

Prophylaxis in hypercatabolic stage patient.
Renal Dialysis
New and Improved Techniques

• Continuous renal-replacement therapy
  - slow and controlled ultrafiltration
  - marked decrease in frequency and duration of hypotension episodes

• Bio-compatible membrane
  - improved survival (57% vs 46%)
  - recovery of renal function (64% vs 43%)
Clinical course

• Renal failure phase usually lasts between 7 and 21 days if the primary insult (ischemia, nephrotoxin) can be corrected.

• Recovery is usually heralded by an increase in urine output and a gradual reduction of BUN and Cr.
Future Directions

• Targeting inappropriate vasoreactivity
  - Atrial natriuretic factor
  - adenosine-receptor antagonists
  - phosphodiesterase inhibitors

• Decrease free radicals
  - lazaroids
  - antioxidants
Future Directions

• Ameliorate tubular obstructions
  - arginine-glycine-aspartic acid peptides

• Regenerate tubular cells
  - insulin-like growth factor I
  - epidermal growth factor
  - hepatocyte growth factor