Acute Renal Failure

Mark D. Baldwin D.O.
F.A.C.O.I.
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Definition of Acute Renal Failure

• An abrupt increase in the BUN and Creatinine with corresponding problems in handling of fluids, Potassium, Phosphorus, and acid-base balance. This is usually a greater than 50% decline in the GFR.
Problems with the Definition

- Serum Creatinine does NOT reflect the degree of renal dysfunction or improvement
- Urine output or lack of may also not reflect the degree of dysfunction
- A better definition may be Acute Kidney Injury (AKI)
Types of Acute Kidney Injury

• Acute Renal Failure can be:
  Oliguric <400 ml/da
  or
  Non-Oliguric >400 ml/da

  Non-Oliguric has a much better prognosis
Acute Renal Failure

--In the Pre-Dialysis Era, ARF had a 50-70% Mortality Rate.

--Today with Dialysis, ARF still has a 50-70% Mortality Rate.

--Thus Patients die With ARF rather than Of ARF.
Types of Acute Renal Failure

- ARF
  - Pre-Renal
  - Intrinsic Renal
    - Vascular
    - Glomerular
    - Interstitial
    - Tubular
  - Post-Renal
Phases of Acute Renal Failure

- **Initiation Phase** - drop in BP, nephrotoxins, early sepsis—rise in BUN/Cr, decreasing urine output
- **Oliguric Phase** - usually less than 400 ml/da, may require dialysis
- **Recovery/Diuretic Phase** - increasing urine output, decreasing BUN/Cr, Potassium, Phosphorus, and Magnesium
Differentiation of AKI

Acute Tubular Necrosis 45%
Pre-Renal 21%
Acute on Chronic R.F. 13%
Obstruction 10%
Glomerulonephritis/vasculitis 4%
Acute Interstitial Nephritis 4%
Athroemboli 1%

Based on 748 cases from 13 tertiary care centers

Kidney Int 1996; 50(3):811
RIFLE Criteria

- **Risk**: 1.5 fold increase in Creatinine or 25% decline in GFR or decrease urine output of <0.5 ml/kg/hr for 6 hours
- **Injury**: Two fold increase in Creatinine or 50% decline in GFR or decrease urine output of < 0.5 ml/kg/min for 12 hours
- **Failure**: Three fold increase in Creatinine or 75% decline in GFR or decrease urine output of 0.5 ml/kg/min for 24 hours or Anuria for 12 hours
- **Loss**: Complete loss of renal function, requiring dialysis for > 4 weeks
- **ESRD**: Complete loss of renal function, requiring dialysis for >3 months

Bellomo, et al
Crit Care. 2004 Aug;8(4):R204-12
Acute Dialysis Qualitative Initiative (ADQI)
RIFLE and Risk of Death

- Risk (1.5 fold increase) 2.4 relative mortality risk
- Injury (2 fold increase) 4.14 relative risk
- Failure (3 fold increase) 6.37 relative risk

- From a review of 13 studies of Critical care patient with AKI vs. without AKI
AKI-KDIGO Guidelines 2012

1. Increase serum creatinine > 0.3 mg/dl w/in 48 hours OR
2. Increase serum creatinine > 1.5x baseline w/in 7 days OR
3. Decreased urine volume < 0.5 ml/kg/hr over a 6 hour period or greater

Approach to a Patient with ARF

- History and Physical
- Review Intake/Output, Blood Pressures
- History of recent Cardiac Cath, Angiogram, Cardiac Surgery, Hypotensive episodes
- Urinalysis including microscopic exam
- Renal ultrasound-rule out obstruction
- Renogram-can show diminished flow to the kidneys
- ANA, ANCA, Anti-GBM, C3, C4, ASO, Hepatitis Serology
- Renal Biopsy
Post Renal Obstructive

- May be acute, chronic or acute on chronic
- Functional renal recovery depends on duration of the obstruction
- Post obstructive diuresis will lead to ARF unless fluid and electrolyte balances are closely monitored maintained
- The nephrologist’s role is in contacting the interventional radiologist or urologist to remove the obstruction
Post Renal Obstructive
<table>
<thead>
<tr>
<th></th>
<th>Pre-Renal</th>
<th>Acute Tubular Necrosis</th>
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<tr>
<td>BUN/Cr</td>
<td>&gt;15-20/1</td>
<td>&lt;10-15/1</td>
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<tr>
<td>Spec Grav</td>
<td>&gt;1.020</td>
<td>&lt;1.010</td>
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<tr>
<td>U osm</td>
<td>&gt;500</td>
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<tr>
<td>U Na</td>
<td>&lt;10</td>
<td>&gt;30-40</td>
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<tr>
<td>FeNa</td>
<td>&lt;1%</td>
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<td>Sediment</td>
<td>Nothing or a few</td>
<td>Numerous Dirty brown</td>
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<td>hyaline casts</td>
<td>Casts</td>
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Pre-Renal Failure

• A decrease in either total circulatory volume or effective circulatory volume (I.e. CHF or Sepsis). This leads to activation of the Renin-Angiotensin-Aldosterone System and ADH. Thus enhanced Na and H2O reabsorbtion.
Causes of Pre-Renal Failure

- Dehydration
- Vomiting, Diarrhea, NG losses, fistulas
- Excessive sweating
- Sepsis
- Diuretic phase of ARF or Post-Obstructive Diuresis
- CHF
- ACE-I or ARBs
- “3rd Space” Losses
Intrinsic Renal Failure-Acute Tubular Necrosis

- Direct insult to the kidney
- May be a result of vascular, glomerular, interstitial, or tubular causes
- Final common pathway of untreated pre-renal or post renal failure
Pathophysiology of ATN

- Hypoxia of the tubular microvasculature leads to tubular necrosis and loss of reabsorbtion and secretory abilities of the tubules. Thus, Acute Tubular Necrosis.
Pathophysiology of ATN-2

- Afferent and Efferent Arteriolar Vasoconstriction
- Mesangial Contraction
- Release of Reactive Oxygen species, NO, ATII, PG’s, Catecholamines
- Tubular Necrosis due to tubular obstruction and back-leak
Pathophysiology of ATN-3

- Cellular Edema
- Increased free Ca++
- Release of compartmentalized enzymes
- Destruction in Cytoskeleton
- Reperfusion injury from reactive Oxygen species, WBC’s, Complements, and cellular debris
ATN
Common Causes of Intrinsic Renal Failure

- Sepsis
- Drugs
- Rhabdomyolysis
- SLE, Wegners, Goodpatures
- Polyarteritis Nodosa
- IgA Berger’s
- Sustained Hypotension
- Post CABG, Angiogram
- Post Streptococcal GN
- Allergic Interstitial Nephritis
- Hemolytic Uremic Syndromes
IgA-Berger’s Disease
Wegner’s Granulomatosis
Management of Acute Renal Failure

• TREAT UNDERLYING CAUSE!!!!!!!!! i.e. Sepsis
• Volume replacement and BP support
• High Dose of Loop Diuretic may convert oliguric to non-oliguric renal failure
• “Renal dose Dopamine” DOES NOT WORK
• Atrial Naturetic Peptide Does NOT work
• Use of Biocompatible dialysis membranes decrease mortality due to less immune stimulation
Drug Induced ARF

- Can be due to Direct toxicity to the tubules i.e. Aminoglycosides
- Can be due to Acute Interstitial Nephritis i.e. TMP/STX, Methicillin
- Can be due to Multiple causes i.e. Contrast
- Can be due to vasoconstriction of the renal arterioles i.e. NSAIDs or Cyclosporine
Common Drugs in ARF

- Contrast Media
- NSAIDs-The MOST Common Drug
- Aminoglycosides
- Penicillins
- Sulfas
- Cephalosporins
- Cyclosporine
- Foscarnet
Common Drugs in ARF (cont)

- Vancomycin
- COX-2 inhibitors
- ACE-I or ARBs in patients w/ RAS
- Intravenous immunoglobulin
- Mannitol
- Hetastarch
- SPICE K-2
Rhabdomyolysis

• Although well recognized in trauma, it is often over looked in non traumatic causes.
• Myoglobin is not directly toxic.
Causes of Non-Traumatic Rhabdomyolysis

- Impaired level of consciousness
- Seizures
- Stroke
- Drug Overdose
- Decreased PO4
- Decreased K
Causes of Non-Traumatic Rhabdomyolysis

- Hyperthermia/Hypothermia
- ETOH
- HMG-Co Reductase inhibitors
- McArdle’s Syndrome
- Tetnaus
- Gas Gangrene
- Decreased Mg
- Decreased Na
Diagnosis of Rhabdomyolysis

- *KEY*: Large Blood on U.A. and few RBCs
- Elevated CPK
- Creatinine >> BUN
- Elevated-Lactate, LDH, PO4, Uric Acid, K
- Decreased Ca
Treatment of Rhabdomyolysis

- Alkaline diuresis D5W or D5 ½ NS with 1 amp NaHCO3 and 20 gm mannitol 6-12 l/da infusion. But must treat early and vigorously. Although the role of alkaline diuresis is not firmly established, it is still cautiously recommended.

- May require dialysis
Athroembolic-Cholesterol Embolic Renal Failure

- Can be Spontaneous in patients with severe athrosclerosis. Commonly seen following angiography, CABG, or Aortic Surgery.
- Due to showering of microemboli and probable local allergic reaction in the glomerulus.
Clinical Feature of Athroembolic Renal Failure

- Blue Toes
- Rash to anterior lower legs
- Livido reticularis
- Peripheral Eosinophilia
- Increased Sed Rate
- Urine Eosinophils
Hemolytic –Uremic Syndrome (HUS)

• Acute Renal Failure associated with microangiopathic hemolytic anemia and thrombocytopenia
• Etiology:
  - E. Coli-Shiga-like toxin, verotoxin
  - Shigella
  - Strep pneumonia
  - Inherited HUS
HUS

• Etiology (cont)
  Drugs-Mitomycin
    -Cyclosporin
    -Oral contraceptives
  Pregnancy related
  Transplant related
  Cancer related
Clinical Features of HUS

- Diarrhea (especially in infectious HUS)
- Increased BUN/Creat
- Decreased Hb/Hct, Decreased Platelet
- Decreased Haptoglobin
- Increased Reticulocyte count
- Fragmented RBCs-Schistocytres, Helmet cells
- CNS Involvement—poor prognosis
Treatment of HUS

- Plasma Exchange
- Dialysis
- Steroids
Indications for Dialysis

1. Volume overload, refractory to diuretics
2. Symptomatic Uremia
3. Electrolyte Abnormalities—i.e. Hyperkalemia
4. Severe Acid-Based Abnormalities
5. Toxin Removal
Toxins Removed by Dialysis

- Aspirin
- Lithium
- Theophyllin-to some degree
- Digoxin-to some degree
- Ethylene Glycol
- Methanol
- Isopropyl Alcohol