Structure and Function of the Kidney

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The Normal Glomerulus

• It consists of a tuft of anastomosing capillaries.
• Mesangium: mesangial cells.
Glomerulonephritis

- Most important group of generalised parenchymal diseases
- Classification is difficult - a mix of clinical and pathological descriptions
  - clinical features
  - morphology (e.g., various histology patterns)
  - pathogenetic mechanisms (e.g., anti-GBM disease)
  - aetiology
- Can be primary or secondary
Four Major Pathogenetic Forms Of Glomerular Injury

In non-proliferative glomerulopathy:

- Damage by antibodies
- Damage mediate by complement

In proliferative glomerulopathy:

- Damage by circulating proinflammatory cells (especially neutrophils and macrophages)
- Damage by locally activating resident cells (for example mesangial cells)
Terminologies To Understand Glomerular Diseases

- Glomerulonephritis
- Diffuse
- Focal
- Segmental
- Membranous
- Proliferative
- Sclerosis
Diffuse

• When all glomeruli of the kidney is involved in disease process.
Focal

• When some glomeruli of the kidney is involved in disease process.
Segmental

• When part of a glomerulous is involved in disease process.
Proliferative

- Where there are increased number of cells in glomeruli...may die to infiltration of PMNs.

- Will result in loss of bowman space and less GFR/urine output- commonly result in acute renal failure.
MEMBRANOUS GLOMERULONEPHRITIS
(Thickened Basement Mem.)
Sclerosis (Trichrome stain)

- Increased collagen, blue colored in this stain.
Crescent is formed by proliferation of epithelial cells and monocytes and fibrin.
Duration

• Acute
  • eg: Acute Diffuse Proliferative glomerulonephritis.

• Chronic
  • eg. Chronic Glomerulonephritis
### Characteristics of common Glomerular Diseases At Presentation

<table>
<thead>
<tr>
<th>Heavy proteinuria</th>
<th>Proteinuria &amp; haematuria</th>
<th>Predominant haematuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal Change</td>
<td>Lupus nephritis</td>
<td>Acute post strep</td>
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<tr>
<td>Focal sclerosis</td>
<td>Membrano-proliferative</td>
<td>Crescentic (RPGN)</td>
</tr>
<tr>
<td>Membranous</td>
<td>Endocarditis</td>
<td>Haemolytic uraemic syndrome</td>
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<td>Diabetes Mellitus</td>
<td>Henoch-Schonlein purpura</td>
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<td>Amyloidosis</td>
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Outcome of Glomerulonephritis

### Graph

- **Percentage still with renal function**
- **Years after apparent onset**

#### Legends:
- **MPGN / FSGS / MGN / SLE**
- **MCD / Mesang PGN / Post Infectious**
- **RPGN**
Acute Nephritic Syndrome

- Syndrome characterised in typical cases by:
  - Haematuria
  - Oliguria
  - Edema
  - Hypertension
  - Reduced GFR
  - Proteinuria
  - Fluid overload
Clinical Features of the Acute Nephritic Syndrome

- Haematuria is usually macroscopic with pink or brown urine (like coca cola)
- Oliguria may be overlooked or absent in milder cases
- Edema is usually mild and is often just peri-orbital- weight gain may be detected
- Hypertension common and associated with raised urea and creatinine
- Proteinuria is variable but usually less than in the nephrotic syndrome
Aetiology of the Nephritic Syndrome

• Most common cause is acute post infectious glomerulonephritis
• Group A beta haemolytic streptococci of certain serotypes important in NZ
• IgA disease and henoch-schonlein purpura, crescentic glomerulonephritis and SLE can also present in this way
Management Issues In The Nephritic Syndrome

- Appropriate investigations: skin and throat swabs, strep serology, complement, urea, creatinine electrolytes, urinalysis and CXR

- BP, urine output and daily weight
- fluid and diet management
- treat hypertension and fluid overload
- treat infection
The Nephrotic Syndrome

- Is not a disease but a group of signs and symptoms seen in patients with heavy proteinuria
- Presents with oedema
- Proteinuria usually > 3.5g / 24hrs (>0.05g / kg / 24hrs in children)
- Serum albumin < 30g/l
- Other features: hyperlipidaemia, and hypercoaguable state
Acute Glomerulonephritis

Acute Post-streptococcal Glomerulonephritis

Non-streptococcal causes
Poststreptococcal Glomerulonephritis
Case 1

• A 18 year-old man presents with hematuria. He also had cold and sore throat recently (two weeks ago). Physical exam is unremarkable. Labs: Cr 1.9 mg/dl, u/a with large blood, - prot, 20-30. What is the most likely diagnosis?
Poststreptococcal GN

- Usually occurs 10 days after pharyngitis and 14 days after skin infection (not synpharyngitic)

- Fallen incidence in US, but common in some rural areas, poor hygiene places, and tropical countries

- Occurs more often in males and children
Clinical Presentation

• Most patients have milder disease
• Classically, presents with overt nephritic syndrome and oliguric ARF
• Symptoms can include gross hematuria (100% microscopic), HA, HTN (60-80%), hypervolemia, and edema (80-90%)
Course

- Irreversible Renal Failure rare – less than 1 % in children, slightly higher in adults
- Resolution usually quick, plasma Cr usually returns to previous levels by 3-4 weeks
- Hematuria resolves usually within 3-6 months, proteinuria falls at a slower rate
- Some patients experience htn, recurrent proteinuria, and renal insufficiency 10-40 yrs after
- > 20% of adults may have some degree of persistent proteinuria and or compromise of GFR 1 year out
Treatment

• Eliminate strep infxn with abx
• Supportive therapy
• Diuretics and antihypertensives to control bp and extracellular fluid volume
Other Acute Glomerulonephritis

Non-streptococcal causes

- Pneumococcal pneumonia
- Hepatitis B, C
- Malaria

• Morphological features of these disease are similar to that of acute post streptococcal GN, only prognosis would be different.
Case 1

• A 25 year-old man presents with gross hematuria. He also has symptoms of URI that started 2 days ago. Physical exam is unremarkable. Labs: Cr 1.5 mg/dl, large blood, ++ prot, 20-30 dysmorphic RBCs/HPF. What is the most likely diagnosis?
IgA Nephropathy (Berger’s Disease)

- The most common cause of GN, esp in Asians
- Exaggerated mucosal IgA response → trapped in glomeruli → mesangial cell proliferation
- Gross hematuria with URI
- Microscopic hematuria & proteinuria common
- Prognosis: generally good, bad if proteinuria > 2 g/d
- Treatment:
  - ACE inhibitors, steroids, cyclophosphamide, fish oil
Minimal Change Disease

- **Syndrome**: Nephrotic syndrome
  - Type of proteinuria: selective (only albumin comes out).
  - Due to loss of the normal charge barrier of GBM
  - **Pathogenesis**: Lymphokine production by T cells
- Most common cause of nephrotic syndrome in children (2-6 years).
- **Light Microscopy**:
  - Normal glomeruli.
  - Lipid droplet in proximal tubular epithelium
Effacement of foot processes due to loss of foot process (giving the appearance of fusion of the epithelial cell)
Membranous nephropathy (GN)

- Syndrome: Nephrotic syndrome
- Most common nephrotic syndrome in ADULT.
- Etiology:
  - Idiopathic or genetic
  - Drug (penicillamine)
  - SLE, Diabetes mellitus
  - Adenocarcinoma of lung and colon.
Membranous GN

H&E stain: diffuse thickening of the capillary wall.  

Sub epithelial deposit
Membranous GN
Clinical Features and Prognosis

• *Some patient develop hypertension and hematuria.*
• It has a variable and indolent course.
• 40% patient progress to renal failure or end stage renal disease after 2-20 years.
• 10-30% with partial or complete remission of proteinuria.
• No or infrequent effect with steroid.
Wegener’s Granulomatosis
Upper Respiratory Tract
Nose

• Nasal crusting
• Frequent nosebleeds
• Erosion and perforation of the nasal septum. The bridge of the nose can collapse resulting in a “saddle-nose deformity”.
Kidney

- Glomerulonephritis w/ associated hematuria and proteinuria
- Can lead to renal failure if not treated aggressively
- Renal masses (rare)
- Active urine sediment: red blood cell casts
Systemic Lupus Erythematosus
Diagnostic criteria

American College of Rheumatology
4/11 criteria (sens 85%, specif 95%) “SOAP BRAIN MD”

• Serositis – heart, lung, peritoneum
• Oral ulcers – painless esp palate
• Arthritis – non-erosive
• Photosensitivity
Diagnostic criteria continued

- **Blood disorders** - ↓RBC (Coombs +), PLT, WCC, Lymphocytes
- **Renal involvement** – proteinuria /± casts
- **ANA** – titer > 1:160
- **Immunologic phenomena** – LE cells, anti-dsDNA Ab, anti-Sm Ab, antiphospholipid Ab, false WR +
- **Neurological disorders** – seizures/ psychosis
- **Malar rash** – cheeks + nasal bridge
- **Discoid rash** – rimmed with scaling, follicular plugging
Butterfly discoid lupus  Discoid LE with "butterfly" distribution in a 24 year-old woman with no systemic involvement. Courtesy of Samuel Moschella, MD.
Chilblain lupus  Chilblain lupus characterized by reddish-blue nodules (in this case, on the fingers) occurring in cold weather. The lesions improved after the administration of nifedipine. Courtesy of Samuel Moschella, MD.
Membranous lupus nephritis  Electron micrograph of membranous lupus nephritis. The subepithelial immune deposits (D) are characteristic of any form of membranous nephropathy, but the intraendothelial tubuloreticular structures (arrow) strongly suggest underlying lupus. GBM = glomerular basement membrane; Ep = epithelial cell. Courtesy of Helmut Rennke, MD.
Tubuloreticular structures in lupus nephritis

Electron micrograph in diffuse proliferative lupus nephritis shows massive subendothelial deposits (D) and characteristic tubuloreticular structures (arrow) in the endothelial cells (En). The subendothelial deposits cause marked thickening of the glomerular capillary wall, leading to a wire loop appearance on light microscopy. Ep = epithelial cell; GBM = glomerular basement membrane. Courtesy of Helmut Rennke, MD.
Systemic Lupus Erythematosus

• **Diagnosis:**
  – clinical presentation - rash, arthralgia, fever, tiredness, anaemia etc
  – hypocomplementaemia - (low C3 and C4)
  – antinuclear antibodies and anti DNA antibodies

• **Treatment:**
  – depends on histological severity (WHO class I - V)
  – nearly all get corticosteroids
  – WHO Class IV usually get corticosteroids and cyclophosphamide
Focal segmental
Glomerulosclerosis (FSGS).
Focal, segmental Glomerulosclerosis

Trichrome stain demonstrates blue, collagen deposition.
Amyloidosis of Kidney

- **Gross:** waxy pale surface
- **LM:**
  - Pink hyaline like deposit
    - in mesangium
- **Cogored**
  - LM: brick red
  - Polarized light: apple green birefringes

- **Type of amyloid:**
  - Primary: Amyloid light chain (Multiple myeloma)
  - Secondary (reactive): AA
Systemic Vasculitis

- Refers to classical polyarteritis nodosa, microscopic polyarteritis, Wegener’s granulomatosis, Henoch Schonlein purpura, Churg Strauss vasculitis and some cases of rheumatoid arthritis
- Pauci immune rapidly progressive glomerulonephritis is common presentation - sometimes with lung haemorrhage (not classical Goodpasture’s syndrome)
- Diagnosis has been made easier by anti neutrophil cytoplasmic antibody (ANCA) tests
Lupus vasculitis  Palpable purpuric lesions on the shins in a patient with lupus and necrotizing vasculitis of the skin, kidney, and brain. Courtesy of Samuel Moschella, MD.
Alport Syndrome

- Hereditary form of GN accompanied by nerve deafness, and various eye disorders
- Males tend to be affected more frequently and severely than females
- Most patients have an x-linked mode of inheritance, but autosomal dominant & recessive patterns also exist

**Morphology**
- focal segmental glomerulosclerosis
- GBM exhibits irregular areas of thickening or thinning with lamination and splitting of the lamina densa
Glomerular Diseases: Adjunctive Treatment to Slow Progression of Renal Disease

- BP control to < 130/80
- ACE inhibitors (or ARBs) to max dosage or combination if tolerated
- Non DH Ca blocker to reduce proteinuria
- Avoid dihydropyridine Ca channel blockers unless combined with ACEIs (increase proteinuria)
- Diuretics (\(\downarrow\)BP, \(\downarrow\)edema)
- Diet: Na restriction, protein restriction (0.8 g/kg/d)
- Smoking cessation
- ? Treatment of hyperlipidemia
Risk of Renal Disease Progression by Systolic BP: A Meta-Analysis of 11 RCTs

GN: Approach to Diagnosis

• History and physical (fluid-volume status, BP, signs of systemic disease)
• What is the syndrome?
• Directed labs: hepatitis and lupus serologies (ANA), C3, C4
• Renal biopsy in most patients with proteinuria > 1 gram/day or with nephritic features
Summary

- GN present with hematuria and/or proteinuria
- Can be renal limited or systemic
- History, physical exam, microscopic urinalysis and directed serologies are essential in diagnosis
- Renal biopsy needed in most cases, esp in nephrotic syndrome and RPGN
- Adjunctive treatment of proteinuria includes BP lowering (<130/80), ACE inhibitors/ARBs or combination, and often diuretics for edema
Autosomal Dominant Polycystic Kidney Disease (ADPKD)
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Autosomal Dominant Polycystic Kidney Disease (ADPKD)

- Accounts for 7-10% of people on dialysis
- 50% of sufferers have renal failure by age 60
- Cysts may occur in liver and pancreas but do not usually give problems
- 5-10% have saccular cerebral aneurysms
- A family history of subarachnoid haemorrhage justifies MRA or CT angio
- Common complications include hypertension, pyelonephritis, abdominal pain, haematuria and renal stones
Pathophiology and Genetics

- Dominant inheritance often obvious from family tree but new mutations do occur
- Mutations of at least 2 genes: PKD-1 on Chr 16 and PKD-2 on Chr 4
- Abnormal gene products are polycystin 1 and 2 which seem to be involved in cellular signaling
- Cyst cells immature and proliferate and secrete fluid.
- About 1-2% of nephrons are involved
- Most cases diagnosed by ultrasound
Treatment of ADPKD

• No specific treatment
• Diligent blood pressure control slows decline of renal function
• ACEi and ARB have no advantages over other agents
• Extended treatment of UTI necessary
• Cyst aspiration does not improve kidney function
Acute and Chronic Urate Nephropathy

- Acute nephropathy with overproduction of uric acid and kidney obstruction with uric acid crystals

- Can occur with treatment of malignant disease with cytotoxics, heat stroke and status epilepticus

- Treat with fluids and prophylaxis with allopurinol

- Role of uric acid in chronic renal failure disputed but does occur with some familial disorders

- Association between hyperuricaemia, hypertension vascular disease, hyperlipidaemia and diabetes
Gout, Uric Acid and Renal Disease

- Uric acid calculi, parenchymal deposits of uric acid and tubular obstruction with urate can cause renal damage
- An elevated plasma uric acid does not in itself seem to cause renal damage
- 1/4 of patients with gout get uric acid stones
- 1/4 of patients with uric acid stones will have gout
Acute Interstitial Nephritis
# Acute Renal Failure

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prerenal</td>
<td>Hypovolemia, Decreased cardiac output, Renal vasoconstriction</td>
</tr>
<tr>
<td>Intrinsic</td>
<td>Acute Tubular Necrosis, Glomerulonephritis, Vascular disorders</td>
</tr>
<tr>
<td>Postrenal</td>
<td>Bladder Neck, Ureteral, Tubular</td>
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</tbody>
</table>
### Most Common Causes

- **Drugs**: 71%  (Antibiotics = 1/3)
- **Infection**: 15%
- **Idiopathic**: 8%
- **TINU Syndrome**: 5%
- **Sarcoidosis**: 1%
Drug Causes of AIN

Antibiotics Cephalosporins, Ciprofloxacin, Ethambutol, Isoniazid, Macrolides, Penicillins, Rifampin, Sulfonamides, Tetracycline, Vancomycin

NSAIIDs Almost all agents, including selective COX-2 inhibitors

Diuretics Furosemide, Thiazides, Triamterene

Miscellaneous Acyclovir, Allopurinol, Amlodipine, Azathioprine, Captopril, Carbamazepine, Clofibrate, Cocaine, Diltiazem, Famotidine, Indinavir, Mesalazine, Omeprazole, Phenteramine, Phenytoin, Pranlukast, Propylthioruacil, Quinine, Ranitidine
## Infectious Causes of AIN

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial</td>
<td><em>Corynebacterium diphtheriae</em>, legionella, staphylococci, streptococci, yersinia</td>
</tr>
<tr>
<td>Viral</td>
<td>CMV, EBV, HIV, HCV, HSV, hantaviruses, mumps, polyoma virus</td>
</tr>
<tr>
<td>Other</td>
<td>Leptospira, mycobacterium, mycoplasma, rickettsia, syphilis, toxoplasmosis</td>
</tr>
</tbody>
</table>
## Clinical Presentation

### AIN of any cause
- Nausea
- Vomiting
- Malaise

### Drug-Induced AIN
- Rash 15%
- Fever 27%
- Eosinophilia 23%
Laboratory Manifestations

• Acute rise in plasma creatinine concentration
• Eosinophilia and eosinophiluria
• Urine sediment: wbcs, rbcs, white cell casts
• Proteinuria (< 1 g/day)
• Signs of tubulointerstitial damage
Treatment

• Discontinuation of offending agent

• Corticosteroids
  - Prednisone 1 mg/kg to a max of 40-60 mg x 1-2 weeks
  - IV Methylprednisolone 0.5 – 1 g/day x 3 days
  - None (Ali’s recommendation)
Prognosis

• Worse if methicillin-induced AIN
• 40% patients have persistent elevation of serum Cr
• Recovery less likely if prolonged period of renal failure, NSAID-induced AIN, interstitial granulomas or fibrosis on biopsy
Heme pigment-induced acute tubular necrosis

• Myoglobinuria: rhabdomyolysis.

• Hemoglobinuria: intravascular hemolysis.
Rhabdomyolysis

- The release of muscle cell contents as the result of traumatic or nontraumatic injury of skeletal muscle
- Physical findings may consist of
  - Tender, “doughy” muscles
  - Edema
  - weakness
  - Compartmental compression symptoms with signs and symptoms of neurovascular compromise may develop, necessitating the need for emergent fasciotomy.
The majority of cases of rhabdomyolysis are nontraumatic

Alcohol abuse

Massive muscle compression from immobilization in drug induced coma

Drug-induced

Seizures

Occlusive peripheral vascular disease.

Combination therapy with itraconazole, simvastatin, and cyclosporine

Conversion from one fibric acid to another, or from one statin-fibrate combination to another

Detergent ingestion
Acute Kidney Injury
Classical approach

• Three classic causes
  – Pre-renal failure
    • Hypovolaemia, hypotension
  – Intrinsic renal failure
    • Acute Tubular Necrosis,
    • Toxic injury
  – Post-renal failure
    • Renal outflow tract obstruction
Acute Kidney Injury

- Spectrum of disorders from reduced function to established failure
- Multi-factorial causes
- Associated with reduced Glomerular filtration and Oliguria, which may initially be appropriate.
- Involves sub lethal damage causing depolarization and loss of normal function
- Cell death through necrosis and apoptosis
# RIFLE classification

<table>
<thead>
<tr>
<th>Risk</th>
<th>GFR/Creatinine criteria</th>
<th>Urine Output criteria</th>
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<tbody>
<tr>
<td>Increase in creatinine x1.5</td>
<td>UO &lt; .5ml/kg/hr for 6hrs</td>
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<tr>
<td>Or GFR decrease &gt;25%</td>
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<table>
<thead>
<tr>
<th>Injury</th>
<th>Increase in creatinine x 2</th>
<th>UO &lt; .5ml/kg/hr for 12hrs</th>
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<tbody>
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<td>Increase in creatinine x 2</td>
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<td></td>
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<tr>
<td>Or GFR decrease &gt;50%</td>
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<tr>
<th>Failure</th>
<th>Increase in creatinine x 3</th>
<th>UO &lt; .3ml/kg/hr for 24 hrs or Anuria for 12hrs</th>
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<tbody>
<tr>
<td>Increase in creatinine x 3</td>
<td></td>
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<tr>
<td>Or GFR decrease &gt;75%</td>
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<tr>
<th>Loss</th>
<th>Persistent ARF = complete loss of renal function &gt; 4 weeks</th>
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<tr>
<th>ESRD</th>
<th>End Stage Renal Disease &gt; 3 months</th>
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Incidence In ICU

- 67% of Critical care patients have AKI
- 12% class R
- 27% class I
- 28% class F

- However more than half of patients with class R will progress to either class I or F
Incidence in ICU

• Approximately 5% of patients in general intensive cares will require RRT
• Equates to 200-300 per million population
• Similar numbers to severe sepsis or ARDS

• 10-20% of surviving patients require ongoing RRT beyond hospital discharge
Sepsis and AKI

• Sepsis accounts for nearly 50% of all causes of AKI
• Combination of Factors
  – Immunological
  – Toxic
  – Inflammatory
• Effect renal microvasculature and Tubular cells
AKI and Mortality

• As already mentioned rising RIFLE class associated with increasing mortality

• Despite advances in critical care medicine and technology, patients who are treated with RRT still have a mortality of 50-60%
Prerenal Acute Renal Failure
Prerenal Acute Renal Failure

- GFR is reduced as a result of hemodynamic disturbances that decrease glomerular perfusion.

- The defining feature of prerenal ARF is the absence of cellular injury and the normalization of renal function with reversal of the altered hemodynamic factors.
Intravascular volume depletion  Altered intrarenal hemodynamics

Etiologies of Prerenal ARF

Decreased effective arterial blood volume  Abdominal compartment syndrome
• Hx
• P/E
• Urine sediment (usually normal, without cellular elements or abnormal casts, unless chronic kidney disease is present)
• Una < 15 meq/L (>20 in ATN)
• U/Pcreat > 20 (<15 in ATN)
• FeNa < 1% (>1% in ATN)
• UNa/K < 1/4
• BUN/creat > 20
Prerenal → Prolonged hypoperfusion → ATN
Postrenal Acute Renal Failure
# Etiologies of Postrenal ARF

<table>
<thead>
<tr>
<th>Intrinsic</th>
<th>Extrinsic</th>
<th>Lower tract obstruction</th>
</tr>
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<tbody>
<tr>
<td><strong>Stone</strong></td>
<td><strong>Retroperitoneal fibrosis</strong></td>
<td>Urethral stricture</td>
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<tr>
<td>Papillary necrosis</td>
<td><strong>Aortic aneurysm</strong></td>
<td>BPH</td>
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<tr>
<td>Blood clot</td>
<td><strong>Retroperitoneal or pelvic malignancy</strong></td>
<td>Prostate CA</td>
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<tr>
<td>TCC</td>
<td></td>
<td>TCC of bladder</td>
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<tr>
<td></td>
<td></td>
<td>Bladder stones</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Blood clot</td>
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<tr>
<td></td>
<td></td>
<td>Fungus ball</td>
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<tr>
<td></td>
<td></td>
<td>Neurogenic bladder</td>
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<td></td>
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<td>Malpositioned catheter</td>
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Urine output?

• The obstruction:
• Complete  →  Anuria

InComplete

Normal
Polyuria
Fluctuating
Complete obstruction

Recovery after relief of obstruction depends on:

- Severity
- Duration
  - Less than 1 wk duration, recovery complete.
  - Little or no recovery after 12 wk.
• Relief of obstruction may be accompanied by a post-obstructive diuresis;
• Excretion of salt and water retained during the obstruction.
• Persistent salt-wasting and impaired urinary concentrating ability.
Response to an Early Biomarker

**Be Warned, Be Watchful**

- Monitor intensively
- Monitor fluid balance, urine output
- Monitor blood pressure, cardiac function
- Monitor electrolytes, kidney function
Response to an Early Biomarker

**Do No Harm**

- Avoid and treat hypotension
- Avoid and treat hypovolemic
- Avoid and treat oliguria
- Avoid contrast agents
- Avoid nephrotoxic medications
Response to an Early Biomarker

_Early Intervention with CRRT_

- Early fluid overload
- Cytokine removal in sepsis
- Toxin removal after contrast administration
Myths

• Frusemide
  – Theoretically may reduce tubular injury
  – Due to shutting down Na/K/Cl ATPase
  – Reduces oxygen demand
  – May help with fluid balance

• But
  – No clinical evidence
  – Accumulates in Oliguria
  – Nephrotoxic and Ototoxic
  – May actually increase mortality and or need for RRT
Myths

• Dopamine
  – Low dose Dopamine (2-3mcg/kg/min), known as “renal dose”
  – No effect on mortality or need for Renal replacement therapy
Myths

• Vasopressors and AKI
  – Although Noradrenaline causes vasoconstriction with renal vasculature
  – No evidence of worsening AKI
  – But should be used after adequate volume resuscitation
Myths

• Mannitol
  – Currently no evidence of protective effect
  – Causes an osmotic diuresis with may benefit fluid balance