Glomerulonephritis
And other Glomerulopathies
Glomerulonephritis vs. Non-inflammatory Glomerulopathy

- Inflammation
- Proliferation
- Immune complexes dense
- Hematuria
- Acanthocytes
- RBC casts

- No inflammation
- Sclerosis
- Thickened BM – fine immune complexes
- Heavy proteinuria
- Fatty casts
- Oval fat bodies

Glomerulonephritides (Nephritic sediment)  "Non-it is" Glomerulopathies (Nephrotic sediment)
Glomerulonephritides

- Anti-GBM disease
- Immune complex disease
- Pauci immune disease ANCA positive
- Idiopathic

Diabetic nephropathy
- Nephroangiosclerosis (Nephrosclerosis-HTN)
- Focal segmental glomerulosclerosis (FSGS)
- Membranous
- Minimal change disease
- Infiltrative

Causes of Glomerulopathies

Dysmorphic red cells: Phase contrast microscopy showing dysmorphic red cells in a patient with glomerular bleeding. Acanthocytes can be recognized as ring forms with vesicle-shaped protrusions (arrows). Courtesy of Hans Kohler, MD.

Non-"itis" Glomerulopathies
Anti-Glomerular Basement Membrane Disease

- Goodpasture’s syndrome—glomerulonephritis and pulmonary hemorrhage
- Mediated by antiglomerular basement membrane antibody which also reacts with BM of pulmonary capillaries
- 1/3 of patients don’t have pulmonary involvement
- Rapidly progressive glomerulonephritis (RPGN) virtually 100%

RPGN – Definition

- Rapid progression to ESRD (days or weeks)
- More than 50–70% of glomeruli involved with crescents
- Not all crescentic GN is RPGN
Crescents

Nonspecific response to severe injury (rents) in glomerular capillary wall
Movement of plasma products into Bowman's Space
• Macrophages
• T cells
• Pro-inflammatory cytokines
  • Interlukin–1
  • Tumor necrosis factor–alpha
Proliferation of cells of Bowman’s Capsule (parietal epithelium) and fibrin formation
Postinfectious GN
Lupus nephritis
IgA nephropathy (Berger’s disease)
Membranoproliferative GN
- Immune complex formation
- Complement driven
Postinfectious Glomerulonephritis

- Infectious agents are the most common inciting antigens associated with immune complex mediated GN
- Post-streptococcal most common
- Other bacteria, viral, parasitic, rickettsial and fungal infections
- RPGN 10% of cases
75% of patients with SLE have renal manifestations
Proteinuria most common renal finding
RPGN 10% of cases
Can have necrotizing process also

Classification of Lupus Nephritis
- Class I – minimal mesangial
- Class II – mesangial proliferative
- Class III – focal proliferative
- Class IV – diffuse proliferative
- Class V – membranous
- Class VI – advanced sclerosing
IgA Nephropathy
Berger’s Disease

- Initial presentation – 40–50% with gross hematuria within 2–5 days of URI
- 30% incidentally found on routine urinalysis
- 10% present as nephrotic syndrome or RPGN
- IgA on immunofluorescent microscopy
- Focal proliferation of mesangium
- Indistinguishable from Henoch–Schonlein purpura
Can be classified by electron microscopy
- Type I - subendothelial deposits
- Type II - Dense deposit disease (DDD)
- Type III - subendothelial and subepithelial deposits

Better classified by immunofluorescence
- Due to immunoglobulins
- Due to activation of alternate complement pathway - C3 nephritis and (DDD)
- Types I and III due to chronic infection or other inflammation such as monoclonal gammopathy causing immunoglobulins
- Type II due to abnormality of alternate complement pathway and activation of C3
- RPGN 10–20%
Due to chronic infection or inflammation.
ANCA positive
Associated with necrotizing GN
Frequently renal limited (no vasculitis)
Granulomas and polyangiitis (GPA or Wegener’s) – associated with vasculitis and pulmonary involvement (PR3–ANCA)
Systemic microscopic polyangiitis (MPA) – no granulomas and without pulmonary involvement (MPO–ANCA)
RPGN virtually 100%
Idiopathic

- ANCA negative
- Immune complex negative
- Anti–GBM negative
- Proliferation and possible crescents indicating inflammatory process nonetheless