RENAL PATHOLOGY

• NORMAL
• CONGENITAL
• “CYSTS”
• GLOMERULAR
• TUBULAR/INTERSTITIAL
• BLOOD VESSELS
• OBSTRUCTION
• TUMORS
1. Renal Vein
2. Renal Artery
3. Renal Calyx
4. Medullary Pyramid
5. Renal Cortex
6. Segmental Artery
7. InterlobAR Artery
8. Arcuate Artery → interlobULAR
9. Arcuate Vein
10. Interlobar Vein
11. Segmental Vein
12. Renal Column
13. Renal Papillae
14. Renal Pelvis
15. Ureter
S.E.M. T.E.M.

- Epithelial foot process
- Endothelial cell pore
- Pedicels
- Endothelial cell
- Glomerular capillary lumen
- Basal lamina
- Fenestrations
CHRONIC RENAL FAILURE

Fluid and Electrolytes: Dehydration, Edema, Hyperkalemia, Metabolic acidosis

Calcium Phosphate and Bone: Hyperphosphatemia, Hypocalcemia, Secondary hyperparathyroidism, Renal osteodystrophy

Hematologic: Anemia, Bleeding diathesis

Cardiopulmonary: Hypertension, Congestive heart failure, Pulmonary edema, Uremic pericarditis

Gastrointestinal: Nausea and vomiting, Bleeding, Esophagitis, gastritis, colitis

Neuromuscular: Myopathy, Peripheral neuropathy, Encephalopathy

Dermatologic: Sallow (greenish-yellow) color, Pruritus, Dermatitis
CONGENITAL

- AGENESIS
- HYPOPLASIA
- ECTOPIC
- HORSESHOE
HYPOPLASIA
ECTOPIC (usually PELVIC)
HORSESHOE
CYSTIC DISEASES

- CYSTIC RENAL “DYSPLASIA”
- Autosomal DOMINANT (AD-ULTS)
- Autosomal RECESSIVE (CHILDREN)
- MEDULLARY
  - Medullary Sponge Kidney (MSK)
  - Nephronophththisis-Medullary
- ACQUIRED
- SIMPLE
CYSTIC RENAL “DYSPLASIA”
• ENLARGED
• UNILATERAL or BILATERAL
• CYSTIC
• Have “MESENCHYME”
• NEWBORNS
• VIRAL, GENETIC (rare)
AUTOSOMAL DOMINANT
• HEREDITARY, PKD1, PKD2
• FOLLOWS AUTOSOMAL DOMINANT PEDIGREE
• COMPLEX GENETICS
• RENAL FAILURE in 50’s
Autosomal Recessive

- Childhood
- Kidneys look exactly like the adult type
- PKHD1
- Patients who survive childhood often develop hepatic fibrosis
MEDULLARY CYSTS

- MEDULLARY SPONGE KIDNEY (MSK), usually an incidental finding on CT or US

- NEPHRONOPHTHISIS, cysts @ CMJ, hereditary (AR), progressive
ACQUIRED (DIALYSIS)
“SIMPLE” CYSTS

• Cortical
• Also called “retention” cysts
• Also “acquired”
• Incidental, asymptomatic
• VERY very very common
GLOMERULAR DISEASES
CLINICAL MANIFESTATIONS

• ACUTE NEPHROTIC SYNDROME
• RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS
• NEPHROTIC SYNDROME
• CHRONIC RENAL FAILURE
• ASYMPTOMATIC HEMATURIA or PROTEINURIA
PATHOLOGIC MANIFESTATIONS

- CELLULAR PROLIFERATION
  - Mesangial
  - Endothelial
- LEUKOCYTE INFILTRATION
- CRESCENTS (RAPIDLY progressive)
- BASEMENT MEMBRANE THICKENING
- HYALINIZATION
- SCLEROSIS
PATHOGENESIS

• Antibodies against inherent GBM
• Antibodies against “planted” antigens
• Trapping of Ag-Ab complexes
• Antibodies against glomerular cells, e.g., mesangial cells, podocytes, etc.
• Cell mediated immunity, i.e., sensitized T-cells as in TB
MEDIATORS

• NEUTROPHILS, MONOCYTES
• MACROPHAGES, T-CELLS, NK CELLS
• PLATELETS
• MESANGIAL CELLS

• SOLUBLE: CYTOKINES, CHEMOKINES, COAGULATION FACTORS
ACUTE GLOMERULONEPHRITIS

• Hematuria, Azotemia, Oliguria, in children following a strep infection
• POSTSTREPTOCOCCAL (old term)
• HYPERCELLULAR GLOMERULI
• INCREASED ENDOTHELIELUM AND MESANGIUM
• IgG, IgM, C3 along GMB FOCALLY
• 95% full recovery
RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS

- Clinical definition, NOT a specific pathologic one

- “CRESCENTIC”

- Anti-GBM Ab
- IMMUN CPLX
- Anti-Neut. Ab
NEPHROTIC SYNDROME
• MASSIVE PROTEINURIA
• HYPOALBUMINEMIA
• EDEMA
• LIPIDEMIA/LIPIDURIA
• NUMEROUS CAUSES:
  – MEMBRANOUS, MINIMAL CHANGE, FOCAL SEGMENTAL.
  – DIABETES, AMYLOID, SLE, DRUGS
MEMBRANOUS GLOMERULONEPHRITIS

- Drugs, Tumors, SLE, Infections
- Deposition of Ag-Ab complexes
- Indolent, but >60% persistent proteinuria
- 15% go on to nephrotic syndrome
MINIMAL CHANGE GLOM. (LIPOID NEPHROSIS)

- MOST COMMON CAUSE of NEPHROTIC SYNDROME in CHILDREN
- EFFACEMENT of FOOT PROCESSES

epithelial foot process
endothelial cell pore
FOCAL SEGMENTAL GLOMERULO-SCLEROSIS

• Just like its name
  – Focal
  – Segmental
  – Glomerulo-SCLEROSIS (NOT –itis)

• HIV, Heroine, Sickle Cell, Obesity

• Most common cause of ADULT nephrotic syndrome
MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS

- MPGN can be idiopathic or 2º to chronic immune diseases Hep-C, alpha-1-antitrypsin, HIV, Malignancies
- GBM alterations, subendo.
- Leukocyte infiltrations
- Predominant MESANGIAL involvement
IgA NEPHROPATHY (BERGER DISEASE)

- Mild hematuria
- Mild proteinuria
- IgA deposits in mesangium
HEREDITARY HEMATURIA SYNDROMES

• ALPORT SYNDROME
  – Progressive Renal Failure
  – Nerve Deafness
  – VARIOUS eye disorder
  – DEFECTIVE COLLAGEN TYPE IV

• THIN GBM (Glomerular Basement Membrane) Disease, i.e., about HALF as uniformly thin as it should be
CHRONIC GLOMERULONEPHRITIS

• Can result from just about ANY of the previously described acute ones
  – THIN CORTEX
  – HYALINIZED (fibrotic) GLOMERULI
  – OFTEN SEEN IN DIALYSIS PATIENTS
SECONDARY (2º) GLUMERULONEPHROPATHIES

- SLE
- Henoch-Schonlein Purpura (IgA-NEPH)
- BACTERIAL ENDOCARDITIS
- DIABETES (Nodular Glomerulosclerosis, or K-W Kidney)
- AMYLOIDOSIS
- GOODPASTURE
- WEGENER
- MYELOMA
TUBULES
INTERSTITIUM
BLOOD VESSELS
OBSTRUCTION
TUMORS
TUBULAR DISEASES

- ACUTE TUBULAR NECROSIS
- TUBULOINTERSTITIAL NEPHRITIS
  - PYELONEPHRITIS
    - ACUTE
    - CHRONIC
  - DRUGS
  - TOXINS
- URATE NEPHROPATHY
- HYPERCALCEMIA/NEPHROCALCINOSIS
- MULTIPLE MYELOMA
ACUTE TUBULAR NECROSIS

• Destruction of renal TUBULAR epithelium
• Loss of renal function
• 50% of ACUTE renal failure
• Two types:

ISCHEMIC
NEPHROTOXIC

- AMINOGLYCOSIDES
- AMPHOTERICIN B
- CONTRAST AGENTS
ATN PATHOGENESIS

• BLOOD FLOW DISTURBANCES (ISCHEMIC)
• TUBULAR INJURY (NEPHROTOXIC)
CLINICAL COURSE

• INITIATION (36 hours)
  – Mild OLIGURIA
  – Mild AZOTEMIA

• MAINTENANCE
  – More OLIGURIA
  – More AZOTEMIA
  – DIALYSIS NEEDED

• RECOVERY
  – HYPOKALEMIA main problem
  – BUN, CREATININE return to normal
TUBULO/INTERSTITIAL NEPHRITIS

• INFECTIONS, i.e., pyelonephritis
• TOXINS, heavy metals, chemo, NSAIDS
• METABOLIC, urates, Ca++, Oxalates
• PHYSICAL, obstruction, radiation
• IMMUNOLOGIC, esp. transplant rejection
PYELONEPHRITIS

• GI Gram NEGATIVES: E. COLI, Proteus, Klebsiella, Enterobacter, Strep. faecalis, usually “NORMAL” flora
• ASCENDING, by FAR, the most common, i.e., reflux, obstruction
• HEMATOGENOUS too
• ACUTE PYELONEPHRITIS, neutrophils
• CHRONIC PYELONEPHRITIS, lymphocytes, scars
ACUTE or CHRONIC PYELONEPHRITIS?
ACUTE or CHRONIC PYELONEPHRITIS?
ACUTE or CHRONIC PYELONEPHRITIS?
FACTORS

• OBSTRUCTION: Congenital or Acquired
• INSTRUMENTATION
• VESICOURETTERAL REFLUX
• PREGNANCY
• AGE, SEX, why sex? F>>>M
• PREVIOUS LESIONS
• IMMUNOSUPPRESSION or IMMUNODEFICIENCY
DRUGS/TOXINS causing INTERSTITIAL NEPHRITIS

- Synthetic Penicillins
- Rifampin
- Thiazides

- 2 weeks later: Fever, eosinophilia, rash, and an acute renal failure type of picture
ANALGESIC NEPHROPATHY

- ASPIRIN, TYLENOL, NSAIDS
  - TUBULointerstitial Nephritis
  - Papillary Necrosis (also Dm & HbS)
URATE NEPHROPATHY

- Precipitation of Uric Acid Crystals in the TUBULES, especially in a LOWER than usual PH situation (mini-TOPHUS)

H & E alcohol fixed

POLARIZED LIGHT MICROSCOPY
PRINCIPLE: In extreme or uncontrolled or chronic HYPERCALCEMIA, calcium stones form in the tubulo-interstitium of the kidney, which can eventually lead to tubular obstruction and loss of function.
MULTIPLE MYELOMA

- Bence Jones proteinuria (immunoglobulin light chains)
- AMYLOIDOSIS
VASCULAR DISEASES

• BENIGN NEPHROSCLEROSIS
• MALIGNANT NEPHROSCLEROSIS (i.e., malignant hypertension)
• RENAL ARTERY STENOSIS
• THROMBOTIC MICROANGIOPATHIES
  – Hemolytic-Uremic Syndromes, Child, Adult, TTP
• THROMBI, EMBOLI, INFARCTS
  – SICKLE CELL
  – DIFFUSE CORTICAL NECROSIS
BENIGN NEPHROSCLEROSIS

• Sclerosis, i.e., “hyalinization” of arterioles and small arteries, i.e., arterio-, arteriolo-
• Is this part of “routine” atherosclerosis????
• VERY VERY VERY common
MALIGNANT NEPHROSCLEROSIS (i.e., malignant hypertension)

- NOT a part of “routine” atherosclerosis
- By definition, associated with rapidly progressive hypertension (1-2% of HTN)
- VASCULAR DAMAGE
- FIBRINOID NECROSIS
- “ONION SKINNING”
- SIGNIFICANT LUMENAL NARROWING
What is “onion-skinning”?

What is an onion?

What is “fibrinoid” necrosis?
Renal Artery Stenosis

• Rare cause of HTN
• SMALL Kidney
• 1) Plaque type is usual cause, yes regular old atherosclerosis
• 2) Fibromuscular “dysplasia” type:
  – INTIMAL HYPERPLASIA
  – MEDIAL HYPERPLASIA
  – ADVENTITIAL HYPERPLASIA
  – In younger women
PLAQUE, i.e., Atherosclerosis  

Fibromuscular Dysplasia
MICROANGIOPATHIES (thrombotic)

• Hemolytic-Uremic Syndrome
  – Familial
  – Childhood
  – Adult

• TTP (Thrombotic Thrombocytopenic Purpura), IDIOPATHIC
MICROANGIOPATHIES

COMMON PROCESSES

– Hemolysis
– Thromboses in renal capillaries
– Thrombocytopenia (a “consumption” coagulopathy)
– FIBRIN PLUGS
OTHER VASCULAR

- Atherosclerosis
- Atheroemboli
- Sickle Cell
- Diffuse Cortical Necrosis
RENAL INFARCTS

• WEDGE SHAPED
• WELL DELINEATED
• “WHITE” (anemic) INFARCT
• Perhaps a little “YELLOW”
• HEAL WITH A SCAR
OBSTRUCTIONS

• UROLITHIASIS
• CONGENITAL
• PROSTATE ENLARGEMENT
• TUMORS
• INFLAMMATION
• SLOUGHED CLOTS, PAPILLAE
• PREGNANCY
• NEUROGENIC
UROLITHIASIS

• CALCIUM (OXALATE or PHOSPHATE) 70%

• MAGNESIUM AMMONIUM PHOSPHATE 20%

• URIC ACID 10%
TUMORS

• BENIGN
  – Papillary Adenoma
  – Fibroma/Hamartoma
  – Angiomyolipoma
  – Oncocytoma

• MALIGNANT
  – Renal Cell Carcinoma (Clear Cell Carcinoma, Adenocarcinoma, Hypernephroma)
  – Urothelial (Transitional)
RENAL CELL CARCINOMA

- TOBACCO RELATED, STRONGLY
- SOME HEREDITARY/FAMILIAL
- MOST are “CLEAR CELL”, a few PAPILLARY
- YELLOW grossly, “CLEAR” cells microscopically
- STRONGLY tend to invade the renal VEIN early, in preference to lymphatics.

Does the kidney have lymphatics?
UROTHELIAL (TRANSITIONAL) RENAL CARCINOMAS

• In renal pelvis. Why?
• 1/10 as common as renal cell carcinomas
• EXACTLY the same appearance as lower urinary tract carcinomas. Why?
• MUCH more likely to obstruct the kidney than renal cell carcinomas. Why?
• Associated with ureter and bladder carcinomas. Why?