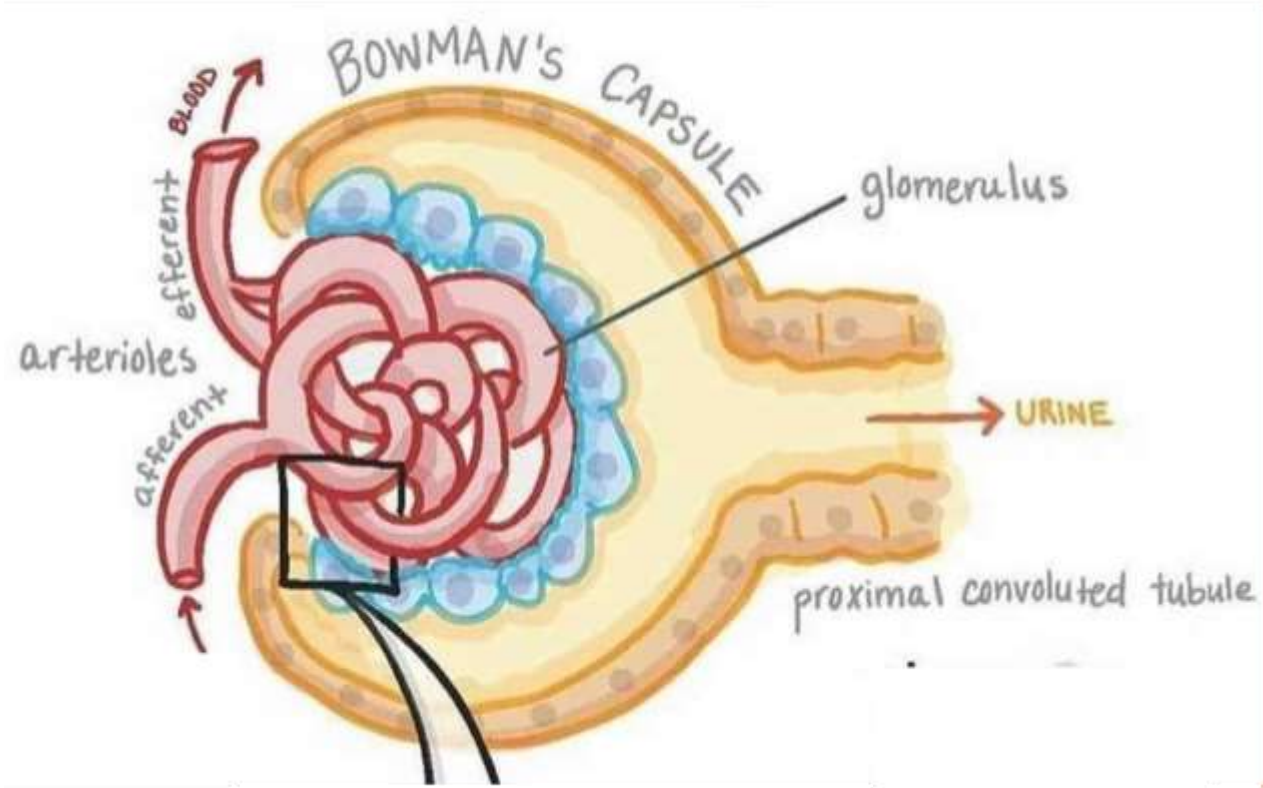
GLOMERULAR DISEASES

ILOS

Define glomerulonephritis
Describe pathogenesis of glomerular injury
Define nephritic and nephrotic syndromes
Enumerate causes of each

GLOMERULAR DISEASES

- **Definition**
  Diseases primarily involving renal glomeruli
  Classified into two broad groups:
  Primary glomerulonephritis: glomeruli main site
  Secondary glomerular diseases: systemic/hereditary


BOWMAN's CAPSULE
glomerulus
BLOOD
efferent
arterioles
afferent
URINE
proximal convoluted tubule

CLINICAL MANIFESTATIONS

Four common features: proteinuria, hematuria
Hypertension, disturbed excretory function
Major glomerular syndromes: nephritic/nephrotic
Acute/chronic renal failure
Asymptomatic proteinuria and hematuria

ACUTE NEPHRITIC SYNDROME

Acute onset: microscopic hematuria, mild proteinuria
Hypertension, edema, oliguria
Classic in post-streptococcal GN
Hematuria gives smoky urine, red cell casts
Proteinuria mild (<3 gm/24 hrs)

- EDEMA & HYPERTENSION IN NEPHRITIC SYNDROME
  Hypertension variable, generally mild
  Edema mild from sodium/water retention
  Oliguria variable
- CONDITIONS WITH NEPHRITIC SYNDROME
  PRIMARY GN: Acute GN (post-/non-streptococcal)
  Rapidly progressive GN
  Membrano-proliferative GN
  Focal/diffuse proliferative GN, IgA nephropathy
- SYSTEMIC DISEASES WITH NEPHRITIC SYNDROME
  SLE, Polyarteritis nodosa
  Wegener’s granulomatosis
  Henoch-Schonlein purpura

NEPHROTIC SYNDROME FEATURES

- Massive proteinuria (>3 gm/24 hrs)
  Hypoalbuminaemia, edema, hyperlipidaemia
  Lipiduria, hypercoagulability
- Mechanisms in nephrotic syndrome
  Hypoalbuminemia from urinary albumin loss
  Edema due to low colloid osmotic pressure
  Hyperlipidemia: increased cholesterol, triglycerides
  Lipiduria from leaky glomerular barrier
  Hypercoagulability: risk of thrombosis

CAUSES OF NEPHROTIC SYNDROME

In children: mostly primary glomerulonephritis
In adults: systemic diseases (diabetes, amyloidosis, SLE)
Most common primary GN in adults: membranous GN (40%)

NEPHRITIC VS. NEPHROTIC SYNDROME

- Proteinuria: mild (<3 gm) vs. heavy (>3 gm)
  Hypoalbuminemia: uncommon vs. present
  Edema: mild vs. marked, generalized
  Hematuria: present vs. absent
  Hypertension: present vs. in advanced disease
- Hyperlipidemia: absent vs. present
  Lipiduria: absent vs. present
  Oliguria: present vs. in advanced disease
  Hypercoagulability: absent vs. present

PATHOGENESIS OF GLOMERULAR INJURY

- Most primary GN has immunologic pathogenesis
  Antibody-mediated injury: In situ immune complex
  Circulating immune complex deposition
  Cell-mediated immune injury: sensitized T cells

- ANTIBODY-MEDIATED INJURY MECHANISMS
  In-situ: antibodies react directly within glomerulus
  Form immune complexes, cause inflammation
  Circulating: Ag/Ab complexes trapped in glomeruli
  Initiate injury and inflammation
- CELL-MEDIATED IMMUNE INJURY
  Sensitized T cells cause glomerular injury
  Involved in progression of some GN types

HISTOLOGICAL ALTERATIONS IN GN

Hypercellularity: mesangial/endothelial/epithelial proliferation
Leucocytic infiltration (neutrophils, monocytes, lymphocytes)
Thickening of glomerular basement membrane
Crescent formation from parietal cell proliferation

OTHER GLOMERULAR ALTERATIONS

Intra-capillary thrombosis, fibrin deposition
Lipid accumulation
Changes may be diffuse (>50% glomeruli)
Global (entire glomerulus)
Focal (<50% glomeruli)
Segmental (part of glomerulus) alinization and sclerosis